Description of the Chronic Pelvic Pain Syndrome (CPPS) Survey from page

https://www.surveyhero.com/c/3ixzzkns

This survey is intended only for men with non- bacterial prostatitis, pelvic pain, Chronic Pelvic Pain Syndrome (CPPS) or prostate-related pain symptoms lasting longer than 6 months who have already undergone urological diagnostic evaluation. It is assumed that:

1) you know your PSA (prostate-specific antigen) level
2) you have undergone a urinalysis and urine culture
3) you have undergone semen analysis and semen culture
4) no persistent bacterial infection has been detected
5) you have undergone digital rectal examination (DRE) multiple times and prostate cancer has been excluded
6) you have consulted several urologists
7) despite treatment, your symptoms remain chronic or recurrent

This survey concerns non- bacterial prostatitis, pelvic pain, Chronic Pelvic Pain Syndrome in men (CPPS). Research indicates that non-bacterial prostatitis, pelvic pain, Chronic Pelvic Pain Syndrome may involve multiple mechanisms: inflammatory, neurological, muscular / myofascial, immunological, prostate-related, seminal vesicle–related, other causes.

Chronic pelvic pain can be a complex and often frustrating condition, especially when initial tests and treatments do not provide clear answers. However, clinical experience and numerous patient-reported outcomes consistently show that a step-by-step, phenotype-based approach—identifying and addressing underlying mechanisms one by one—can lead to meaningful and lasting improvement. Although this process may take time, many patients have achieved significant symptom relief by gradually understanding their individual triggers and responses. Real-world patient experiences, including those shared on platforms such as http://www.ucpps.men and similar communities, highlight that recovery is often not immediate, but progressive and achievable with a structured, personalized strategy. This reinforces an important message: even in long-standing cases, improvement is possible when the condition is approached comprehensively and systematically.

Bacterial prostatitis accounts for only a small minority of prostatitis-like syndromes, with chronic bacterial prostatitis estimated at approximately 5–10% of cases; therefore, persistent pelvic pain with negative urine and semen cultures should prompt evaluation for non-bacterial CP/CPPS mechanisms. Each case must be diagnosed by a urologist and possibly referred to other healthcare specialists.

http://www.ucpps.men

Neurobiological Sensitivity of the Pelvic Structures

The prostate, seminal vesicles, and surrounding pelvic tissues represent one of the most densely innervated and functionally complex regions of the male body. This area integrates **autonomic (sympathetic and parasympathetic), sensory, and somatic neural pathways**, which together regulate urinary function, ejaculation, sexual arousal, and orgasm. Because of this high degree of neural integration, even minor physiological or biochemical changes may be perceived as significant sensory events, including pain or discomfort.

From a neuroanatomical perspective, the pelvic organs are innervated primarily through the **pelvic plexus**, which contains a mixture of sympathetic fibers (originating from the thoracolumbar spinal cord) and parasympathetic fibers (originating from sacral segments S2–S4). These fibers control smooth muscle contraction, glandular secretion, vascular tone, and ejaculatory function. In parallel, somatic sensory input—particularly via the **pudendal nerve**—provides high-resolution perception from the perineum, urethra, and external genitalia. This dual innervation creates a system that is both **highly responsive and highly vulnerable to dysregulation**.

Functionally, these structures are central to sexual physiology. The prostate and seminal vesicles contribute to seminal fluid production and are actively involved in the **emission phase of ejaculation**, while coordinated activity of pelvic floor muscles contributes to expulsion. At the same time, sensory input from these structures participates in the generation of **sexual arousal and orgasmic signaling** within the central nervous system. This explains why the same anatomical region is capable of producing both **intense pleasure and significant pain**, depending on the underlying physiological state.

In the context of chronic pelvic pain, several mechanisms may amplify this sensitivity. One of the most important is **neurogenic inflammation**, in which peripheral nerve endings release mediators such as substance P and calcitonin gene-related peptide (CGRP). These substances increase local blood flow, vascular permeability, and immune activation, while simultaneously lowering the threshold for pain perception. Mast cells located in pelvic tissues may be activated in parallel, releasing histamine and other inflammatory mediators that further sensitize nearby nerve endings. This creates a **positive feedback loop between the nervous system and the immune system**, in which even minor stimuli can produce disproportionate pain.

Another key factor is **central sensitization**, in which repeated or prolonged nociceptive input leads to increased excitability of neurons in the spinal cord and brain. In this state, signals originating from the prostate, seminal vesicles, or pelvic floor are amplified, and normal physiological processes—such as ejaculation, bladder filling, or mild mechanical pressure—may be interpreted as painful. Importantly, this mechanism does not require ongoing tissue damage; rather, it reflects a change in how sensory information is processed.

The **pelvic floor muscles** also play a significant role in modulating sensitivity. Chronic tension or impaired relaxation of these muscles can lead to mechanical compression of nerves and blood vessels, reduced tissue perfusion, and the formation of myofascial trigger points. This can increase both baseline discomfort and the intensity of responses to otherwise minor stimuli. In such cases, pain may fluctuate depending on posture, physical activity, stress level, or sexual activity.

Hormonal and autonomic factors further influence this system. The same neural pathways that regulate erection, ejaculation, and libido also modulate vascular tone and glandular activity. As a result, interventions that alter hormonal balance, sympathetic tone, or neurotransmitter activity—such as certain medications or supplements—may produce noticeable changes in pelvic sensation. In a sensitized system, even small shifts in these regulatory pathways may be perceived as pain, pressure, or altered sexual function.

Finally, the close anatomical and functional relationships between the urinary tract, reproductive organs, gastrointestinal system, and central nervous system mean that cross-organ interactions are common. For example, bowel distension, bladder irritation, or stress-related autonomic activation may all influence pelvic organ sensitivity. This interconnectedness contributes to the variability and unpredictability of symptoms in CPPS.

In summary, the high sensitivity of the prostate, seminal vesicles, and surrounding pelvic structures reflects their role as a **neurofunctional hub integrating sexual, urinary, immune, and sensory processes**. Because these systems are tightly interconnected, even minor perturbations—whether mechanical, biochemical, or pharmacological—may produce amplified sensory responses. This explains why patients may experience significant changes in symptoms in response to relatively small triggers or therapeutic interventions, particularly in the presence of underlying sensitization.

Sequential Therapeutic Introduction and Dose Titration in CPPS

A fundamental principle is that **only one new intervention should be introduced at a time**, regardless of whether the intervention is pharmacological, nutraceutical, or physiotherapeutic. Following the initiation of a given therapy, an **observation period** should be maintained, the duration of which depends on the pharmacodynamics of the agent and the expected latency of effect. In most cases, early tolerability can be assessed within several days, whereas meaningful therapeutic response may require a longer interval, typically extending up to several weeks. This staged approach allows for the isolation of effects attributable to a specific intervention and prevents confounding interactions between multiple simultaneously introduced therapies.

If the initial intervention produces a **clinically meaningful improvement and is well tolerated**, it should be maintained at the current dose. Only after stabilization of the response should a subsequent intervention be considered, introduced with the same degree of temporal separation. In contrast, if the intervention is **well tolerated but ineffective**, a gradual dose escalation may be undertaken. Dose adjustments should be performed cautiously, at defined intervals, allowing sufficient time for reassessment after each increment. If, despite appropriate titration, no benefit is observed, the intervention should be discontinued and an alternative mechanism-targeted therapy should be considered.

In situations where the introduction of a new therapy leads to **symptom exacerbation or adverse effects**, the most recently introduced intervention should be the first to be reduced or withdrawn. This principle is essential for maintaining clarity in therapeutic interpretation. A return of symptoms to baseline following discontinuation strongly suggests that the withdrawn intervention was either poorly tolerated, mechanistically inappropriate for the dominant phenotype, or capable of inducing a paradoxical response. Such paradoxical worsening is not uncommon in CPPS, particularly in individuals with heightened neurogenic or immune sensitivity.

Dose management should consistently follow a **“start low, go slow” strategy**. Initiation at the lowest clinically accepted dose reduces the risk of overstimulation of sensitive biological systems, including the nervous, autonomic, and immune networks, which are frequently dysregulated in CPPS. Gradual titration allows the organism to adapt to pharmacological modulation and reduces the likelihood of misinterpreting adverse reactions as primary disease progression. This is particularly relevant in patients with neuropathic pain features, central sensitization, or histamine-related reactivity, in whom abrupt pharmacological shifts may provoke symptom flares.

Equally important is the maintenance of **temporal separation between therapeutic trials**. Introducing multiple new agents within a short time frame significantly reduces the ability to attribute clinical changes to a specific intervention and increases the risk of cumulative side effects. In early phases of management, clarity of response should be prioritized over treatment intensity. Only after individual responses have been characterized should **combination therapy** be considered. At that stage, combining interventions that target distinct mechanisms—such as pelvic floor dysfunction, neurogenic sensitization, and inflammatory pathways—may yield additive or synergistic benefits.

Continuous clinical monitoring is essential throughout this process. Patients should be encouraged to document symptom patterns in a structured manner, including pain intensity and quality, urinary and sexual function, gastrointestinal symptoms, and identifiable triggers. Particular attention should be paid to delayed responses, such as post-ejaculatory flares or activity-related exacerbations, which may not be immediately apparent following intervention initiation. This longitudinal observation facilitates more accurate phenotype classification and supports rational therapeutic sequencing.

In summary, the sequential introduction and titration of therapies in CPPS serve not only as a treatment strategy but also as a diagnostic tool. By carefully observing the organism's response to targeted interventions, it becomes possible to infer the relative contribution of different pathophysiological mechanisms. This method aligns with contemporary phenotype-based models of CPPS and supports a personalized, mechanism-oriented approach to management.

Description of the Chronic Pelvic Pain Syndrome (CPPS) Survey

This questionnaire is based on the latest available medical knowledge and current multidisciplinary understanding of chronic pelvic pain syndrome (CPPS). However, it is intended solely as an educational and supportive tool and does not replace consultation with a qualified healthcare professional. The results obtained from this questionnaire should not be considered a medical diagnosis, and no clinical decisions should be made without appropriate medical evaluation. The creators of this questionnaire do not assume responsibility for any interpretations or actions taken based on its results.

This questionnaire is designed to provide a comprehensive, phenotype-based assessment of chronic pelvic pain syndrome (CPPS) by integrating symptoms, triggers, biological responses, and treatment outcomes into a single structured framework. Rather than focusing on one organ or a single cause, it captures the multidimensional nature of the condition, including pelvic floor dysfunction, neurological sensitivity, immune and histamine-related mechanisms, vascular factors, gastrointestinal influences, and psychophysiological components. By analyzing not only what symptoms are present but also what aggravates or relieves them, the questionnaire allows for identification of dominant mechanisms and overlapping patterns, supporting a more precise, individualized diagnostic and therapeutic approach in line with current multidisciplinary clinical practice.

Point 1. How old are you?

(select one answer)

Purpose of this question

This item is not included merely for demographic description. Its main purpose is to provide an **interpretive clinical frame** for everything that follows in the questionnaire. Age does not diagnose CPPS, and it should never be used in isolation. However, it changes how subsequent answers are weighted when the clinician or researcher interprets the overall phenotype. In other words, age acts as an **epidemiological and biological context variable**. It influences how likely one is to prioritize functional pelvic floor overactivity, neuropathic pain, post-inflammatory tissue change, lower urinary tract dysfunction, endocrine factors, vascular factors, and age-related prostate pathology in the differential picture.

In modern chronic pelvic pain medicine, the goal is not to say "young men have one disease and older men have another." Rather, the goal is to understand that the **same complaint** — for example perineal pain, post-ejaculatory pain, urinary urgency, or pelvic pressure — may carry a different **probability structure** depending on age. That is why this question belongs at the beginning of the survey. It helps define the biological background against which the rest of the symptom complex will later be interpreted.

Age <20

When symptoms consistent with chronic pelvic pain appear in this youngest age category, the survey is usually capturing a pattern in which **primary structural prostate disease is less likely to be the dominant explanation**. In this age range, interpretation more often shifts toward a **functional, neuromuscular, autonomic, sexual-pattern, or neurodevelopmental phenotype**. If a respondent in this category later reports perineal pain, pain worsened by sitting, pain after ejaculation, urinary urgency with normal urine culture, pelvic floor tension, cold sensitivity, racing thoughts, attention-related dysregulation, or heightened bodily vigilance, the overall profile becomes more consistent with **pain amplification within a sensitized pelvic system** rather than with age-related gland disease.

This does **not** mean that symptoms in a man under 20 should be dismissed as "just tension." On the contrary, this age group requires careful attention because when chronic pelvic pain begins early, it may reveal a **highly reactive regulatory phenotype**: increased pelvic floor recruitment under stress, maladaptive sexual behaviors such as prolonged arousal without release, post-infectious sensitization, or an interaction between gut symptoms, autonomic arousal, and pelvic pain. In some cases, pain beginning at a very young age may persist long enough to become a chronic multi-system disorder if not understood correctly.

Age 20–29

This is one of the most clinically important age groups in CPPS work. In many men, this is the decade in which the syndrome first becomes fully established. It is also the decade in which several major perpetuating factors frequently converge: prolonged sitting, high occupational or academic stress, intense or dysregulated sexual activity, compulsive sexual stimulation, prolonged computer use, autonomic overactivation, irregular sleep, and emerging gut-related triggers. If later sections show pain with sitting, pain with ejaculation, perineal burning, post-void discomfort, penile hypersensitivity, or strong stress reactivity, the interpretation in this age group very often supports a **neuro-muscular-autonomic phenotype**.

At the same time, this age range should not be oversimplified. A patient in his twenties may still have meaningful post-infectious change, inflammatory semen findings, pelvic floor spasm, or early structural consequences of repeated irritation. Thus, age 20–29 often represents the point where the condition is best understood not as "psychological" and not as "pure prostatitis," but as a **multi-trigger chronic pelvic pain pattern** that commonly begins with a specific trigger and then becomes self-reinforcing.

Age 30–39

In this age group, the questionnaire often begins to capture a more **entrenched chronic pain phenotype**. Functional mechanisms remain very important, but there is usually a longer history of exposure to perpetuating factors: years of sitting, years of stress, repeated flares after ejaculation, chronic sleep dysregulation, long-standing bowel reactivity, or prolonged pelvic floor overactivity. Therefore, answers in men aged 30–39 often need to be interpreted within a model of **accumulated chronicity** rather than a newly emerging syndrome.

If someone in this age bracket later reports broad pain distribution, allodynia, tingling, urinary hypersensitivity, rectal fullness, migraine, fibromyalgia-like features, or multiple failed treatments, age 30–39 may suggest that the original trigger is no longer the whole story. Instead, the clinician may be looking at a **chronic pelvic pain network disorder** in which nerves, muscles, autonomic regulation, immune reactivity, and cognition all contribute. This is also an age at which imaging or

TRUS may start to show chronic sequelae in some patients, such as post-inflammatory irregularities or fibrotic change, which may coexist with sensitization rather than replace it.

Age 40–49

This decade is often a **transitional interpretive zone**. A pure functional CPPS phenotype remains entirely possible, but the probability of meaningful contribution from the prostate, bladder outlet, hormonal balance, endothelial/vascular factors, and chronic tissue change is higher than in younger groups. Accordingly, the same symptom constellation — for example post-ejaculatory pain, urinary hesitancy, perineal fullness, weak stream, pelvic pressure — may carry more structural relevance in this age group than it would in a teenager or a man in his early twenties.

This does not mean that age 40–49 should automatically push the evaluator toward a prostate-only interpretation. Rather, it should increase vigilance for **mixed phenotypes**. A man in this range may have pelvic floor overactivity and neuropathic burning **together with** mild outlet dysfunction, prostate tenderness, venous congestion, hormonal imbalance, or MRI/TRUS evidence of chronic post-inflammatory change. In such cases, age does not replace symptom analysis; it tells the clinician to interpret symptoms within a wider biological frame.

Age 50–59

By this stage, age begins to matter more strongly because lower urinary tract symptoms, prostate-related changes, vascular factors, endocrine shifts, and chronic post-inflammatory structural consequences become more plausible co-contributors. If a respondent in this category later endorses weak stream, incomplete emptying, nocturia, pelvic pressure, deep prostate-region pain, improvement with alpha-blockers, or benefit from tadalafil, age 50–59 increases the probability that the phenotype contains a **smooth-muscle, outlet, vascular, or prostate-associated layer** in addition to any functional pain mechanisms.

At the same time, it is essential not to over-attribute symptoms to age. Many men in this decade still have pain patterns that are disproportionately neuropathic, myofascial, stress-amplified, or gut-linked. What changes is not that CPPS disappears and prostate disease “takes over,” but that the evaluator must more readily consider **coexistence**: chronic tissue change plus nerve hypersensitivity, urinary dysfunction plus pelvic floor overactivity, or vascular heaviness plus post-ejaculatory pain.

Age 60–69

This age group requires broader differential thinking. Prostate enlargement, bladder outlet obstruction, medication-related urinary effects, vascular insufficiency, androgen-related changes, and age-associated comorbidity are more relevant here than in younger groups. If the questionnaire later reveals significant nocturia, weak stream, retention-like episodes, chronic pelvic heaviness, erectile dysfunction, or symptom fluctuation with medications affecting smooth muscle or circulation, age 60–69 makes a **mixed urological-pain phenotype** more likely.

However, even here, chronic pelvic pain should not be reduced to gland size or age alone. A patient in his sixties may still have a clearly neuropathic or pelvic floor–dominated syndrome, especially if he reports burning pain, electric sensations, allodynia, symptom worsening with sitting, or strong response to neuromodulating treatments. Therefore, this age category should be interpreted as increasing the need for **careful multi-domain correlation**, not as replacing the broader CPPS framework.

Age >69

In men over 69, age itself becomes a strong clinical context variable because structural, vascular, endocrine, medication-related, and urinary tract contributors are all more likely to coexist. In this category, subsequent answers on pain quality, urinary symptoms, sexual symptoms, imaging, and treatment response should be read with a particularly strong emphasis on **multimorbidity and overlap syndromes**.

Still, the presence of advanced age does not invalidate a diagnosis of chronic pelvic pain syndrome. If the later questionnaire profile is dominated by burning pain, allodynia, sitting intolerance, stress sensitivity, pelvic floor spasm, or disproportionate symptom severity relative to findings, then a chronic pain / sensitization model remains entirely relevant. In other words, the oldest age band should never be interpreted as "all symptoms are explained by age," but rather as "age broadens the number of plausible coexisting mechanisms that must be integrated."

Professional summary of Point 1

The purpose of Point 1 is to define the **biological context in which all later symptoms will be interpreted**. Age does not diagnose CPPS, but it changes the relative weight of functional, neurological, muscular, endocrine, vascular, bladder-outlet, and prostate-related explanations. In professional use, this question should be treated as an **interpretive framework variable**, not as a standalone predictor.

Point 2. How long have you had persistent or frequent recurring pelvic pain?

(select one answer)

Purpose of this question

This item is one of the most important structural questions in the entire survey. Its purpose is not merely to record duration for descriptive statistics. It is designed to estimate the **degree of chronicity**, the likelihood that secondary pain-maintaining mechanisms have already developed, and the probability that the patient's current symptom pattern is no longer explained by the original trigger alone. In guideline-based language, chronic prostatitis / CPPS is defined by **persistent or recurrent pain lasting at least several months**, and chronicity itself changes the biology of pain.

The longer pain persists, the more likely it is that the patient has moved from an initial trigger-driven state into a broader **chronic pain phenotype** involving pelvic floor guarding, central sensitization, maladaptive sexual patterns, activity avoidance, sleep disruption, altered interoception, anxiety about symptoms, and, in some patients, structural post-inflammatory or fibrotic tissue consequences. Thus, Point 2 is best understood as a **disease-stage question**. It helps distinguish early chronic pelvic pain from entrenched multi-axis chronic pelvic pain.

Duration of the disease > 6 months

This response defines entry into the survey's target population: men with pain that is already chronic rather than acute or transient. At this duration, the original trigger may still remain relatively visible. For example, a patient may still clearly associate symptom onset with an infection, a sexual overload event, prolonged sitting, a stressful life period, or a gastrointestinal insult. Therefore, when pain has persisted for just over six months, interpretation should still give relatively strong weight to the **initial trigger phenotype**.

At the same time, pain at this stage is already chronic enough that early secondary mechanisms may have begun to form. The patient may already show urinary hypersensitivity, pelvic floor clenching, fear of sitting, increased attention to symptoms, or avoidance of ejaculation. However, compared with longer-duration categories, this group often offers a greater chance of identifying a relatively dominant mechanism before the syndrome becomes deeply layered. Clinically, this category often corresponds to a **still partially reversible chronic phase**, provided the dominant perpetuating mechanism is recognized early.

Duration of the disease > 6–12 months

This response indicates that the condition has remained active long enough to move beyond a short-lived chronic transition. At this stage, many men still remember the initiating event clearly, but the syndrome often begins to shift from "pain caused by one thing" toward "pain maintained by several interacting systems." Pelvic floor overactivity, poor sleep, autonomic hyperarousal, recurrent symptom checking, and fear of physical or sexual triggers may already be reinforcing the original biological insult.

Clinically, this is a particularly important duration band because it frequently marks the period in which the patient is **no longer simply recovering from an insult** but is beginning to live inside a chronic feedback loop. If later questionnaire sections show stress sensitivity, post-ejaculatory pain, urinary urgency without infection, breathing dysfunction, or increasing symptom spread, then this duration range supports the interpretation that sensitization and pelvic floor dysfunction are already becoming clinically relevant.

Duration of the disease > 1–2 years

This category strongly suggests that the syndrome is no longer being sustained by the original trigger alone. By one to two years, chronic pelvic pain often behaves less like a local post-event condition and more like a **self-maintaining pain system**. In many patients, this is the stage at which symptom patterns become broader, more variable, and more layered. Patients may begin to report changes in pain location, more severe reactions to sitting, wider symptom reactivity to stress or food, sleep disturbance, reduced sexual confidence, and stronger urinary or bowel cross-talk.

This duration also increases the likelihood that even if the original inflammatory or infectious event has resolved, the patient may now have **persistent nociceptive sensitization, habitual pelvic floor contraction, or durable behavioral reinforcement patterns**. If imaging, semen markers, or prostate-region examinations later show low-grade chronic abnormalities, one to two years is also long enough for early post-inflammatory tissue consequences to begin to matter clinically.

Duration of the disease > 2–5 years

This duration usually indicates a well-established chronic disorder. The patient is unlikely to have only one active mechanism at this stage. Instead, the syndrome frequently includes some combination of: pelvic floor overactivity, neuropathic pain features, chronic urinary hypersensitivity, stress amplification, altered sexual patterning, sleep impairment, gastrointestinal contribution, and immune or histamine-related triggers. In clinical reasoning, a duration of two to five years strongly increases the probability that pain has become **systemically reinforced**.

This category is also important because it is long enough for post-inflammatory, fibrotic, or tissue-remodeling changes to appear in some patients, even if standard testing remains partly normal. In other words, symptom persistence in this duration band should prompt the evaluator to think in

terms of a **mixed model**: tissue history, nerve amplification, muscular guarding, and behavioral adaptation coexisting within one chronic syndrome.

Duration of the disease > 5–10 years

Pain in this duration range almost always suggests a deeply entrenched chronic phenotype. By this stage, many patients have undergone numerous consultations, repeated testing, fluctuating diagnoses, partial treatment responses, and progressive lifestyle adaptation around the pain. It becomes increasingly likely that the current symptom picture is being maintained not just by the original biological problem but by **long-established neurophysiological patterning**.

This does not mean the syndrome has become "purely central" or "purely functional." On the contrary, in a subset of men, five to ten years is sufficient time for chronic post-inflammatory change, tissue fibrosis, persistent deep tenderness, vascular congestion, or secondary biomechanical effects to become clinically relevant. What changes at this duration is that such tissue-level abnormalities, if present, are very likely to exist **within a sensitized pain system**, so the patient may experience them with greater intensity and broader symptom spread than the structural findings alone would predict.

Duration of the disease > 10 years

Once symptoms have persisted beyond ten years, chronic pelvic pain should be interpreted as a **long-standing multi-system disorder** unless there is strong evidence to the contrary. At this stage, it is rarely clinically useful to assume a single active cause. Instead, the evaluator should expect overlap between pain-memory processes, pelvic floor dysfunction, activity adaptation, sexual adaptation, urinary and bowel cross-sensitization, autonomic dysregulation, and, in some patients, durable tissue-level change.

In practical terms, a duration beyond ten years suggests that treatment interpretation becomes especially valuable. Responses to pelvic floor therapy, breathing retraining, antihistamines, neuromodulators, steroids, dietary interventions, or vascular-targeted approaches can help identify which mechanisms are **still actively modulating symptoms** despite the very long disease history. This is one reason duration is such an important variable: it changes how much weight should be given to the original trigger versus the current pain-maintaining systems.

Duration of the disease > 20 years

This longest duration band suggests a profoundly chronic syndrome with a high probability of **layered and self-reinforcing pathophysiology**. Patients in this group often no longer fit a narrow organ-based model. Their current phenotype may include nerve hypersensitivity, muscle guarding, conditioned pain responses, severe symptom vigilance, chronic urinary or bowel cross-talk, and possibly persistent structural sequelae from earlier inflammatory or mechanical processes.

A duration beyond twenty years should not be interpreted pessimistically as proof that the condition is untreatable. Rather, it indicates that symptom interpretation must be particularly sophisticated. In this group, "what still helps" and "what still worsens" become especially important, because they may reveal which components remain biologically active within a very old pain network. This duration category therefore marks not only maximal chronicity, but also maximal need for **phenotype-specific interpretation rather than simple diagnosis labeling**.

Professional summary of Point 2

The purpose of Point 2 is to determine the **stage of chronicity** and to estimate how far the patient may have progressed from an original trigger-driven state into a multi-layer chronic pain phenotype. Shorter chronic durations still allow stronger emphasis on the initiating mechanism. Longer durations increasingly support the presence of sensitization, pelvic floor overactivity, autonomic dysregulation, symptom conditioning, and, in some patients, structural post-inflammatory or fibrotic sequelae. In professional interpretation, this question is not just about time. It is about **how much the disease has had time to reorganize the pelvic pain system**.

Point 3. Which of the following apply to you?

(select all that apply)

In the current questionnaire, Point 3 includes the following options: **My job causes significant stress; My job requires prolonged sitting; After work I also spend long periods sitting at home; My job requires lifting heavy objects; I currently have or previously had a high sexual drive; I currently have or previously had a low sexual drive; Other (please specify).**

Purpose of Point 3

Point 3 is designed to establish the **functional background** in which the pain syndrome developed and is being maintained. It does not ask directly about pain, infection, urinary symptoms, or treatment response. Instead, it identifies the **daily physiological environment** in which the pelvic pain system operates. This is critically important in CPPS, because chronic pelvic pain rarely develops in a vacuum. It usually emerges within a pattern of repeated physical loading, autonomic overactivation, sexual overuse or sexual dysregulation, insufficient recovery, or long-term behavioural exposure such as prolonged sitting.

From a clinical perspective, Point 3 serves as a **background phenotype builder**. It helps answer questions such as: Is the patient living in a high-sympathetic state? Is the pelvic floor under repeated compressive load? Is the pelvis repeatedly exposed to increased intra-abdominal pressure? Is there a pattern of high sexual stimulation with insufficient recovery time between ejaculatory events? Or is the opposite true — namely low sexual drive, sexual avoidance, and possible endocrine or pain-related suppression of sexual function? This question therefore helps define the **terrain** on which later symptoms arise.

It is important to emphasize that the options in Point 3 are **not diagnoses**. Each one is a contextual factor. Their value lies in how they interact with later sections on urinary symptoms, ejaculation-related symptoms, pudendal features, pelvic floor findings, gastrointestinal symptoms, histamine-related triggers, and treatment response. In modern pelvic pain medicine, these background exposures are not “minor lifestyle details”; they are part of the pain phenotype itself.

My job causes significant stress

When a respondent endorses significant occupational stress, the survey is identifying a potentially major **psychophysiological driver** of chronic pelvic pain. This should not be interpreted in a simplistic way as meaning that the pain is “psychological” or “caused by stress alone.” Rather, it indicates that the body may be functioning in a state of **persistent sympathetic activation**, reduced parasympathetic recovery, increased muscle guarding, and impaired pain regulation. In many men with CPPS, the pelvic floor behaves as part of this broader stress-response system: under chronic stress, the person may unconsciously tighten the lower abdomen, gluteals, anal sphincters, and pelvic floor muscles, particularly during concentration, anxiety, or emotional suppression.

Clinically, this answer becomes especially meaningful if later sections show **chest-dominant breathing, breath-holding under stress, difficulty relaxing the pelvic floor, worsening during periods of psychological stress, increased symptom intensity when focusing on the body, sleep disturbance, or autonomic symptoms such as palpitations and cold extremities**. In that context, occupational stress should be viewed as a real biological load that can increase pelvic floor tone, amplify nociception, worsen urinary urgency, and reduce the threshold for post-ejaculatory or sitting-related flares. EAU guidance explicitly recognizes the relevance of psychological, behavioural, sexual, and functional consequences in chronic pelvic pain phenotyping.

This answer can also carry prognostic meaning. If the patient's job stress is high and later ranking questions place stress near the top of immediate triggers, then the clinician should strongly consider a **neuro-autonomic / psychophysiological phenotype**. In such a case, pelvic pain is often not generated solely by peripheral tissues, but by an interaction between a sensitized pelvic system and a chronically activated stress-regulation network.

My job requires prolonged sitting

This is one of the most important mechanical background variables in the entire survey. Prolonged sitting creates repeated and often cumulative **compressive loading of the perineum, pelvic floor, pudendal region, posterior scrotal area, and deep soft tissues of the pelvis**. It may also reduce positional variability, impair venous outflow, maintain shortened hip flexion patterns, and increase guarding in the pelvic floor and lower abdominal wall. In men who already have a vulnerable pelvic system, prolonged sitting can operate as a chronic load-maintenance mechanism rather than merely a temporary aggravator.

This answer becomes particularly significant if later sections reveal **perineal pain, pain in the anus or prostate region, pain worsened by sitting, driving, cycling, leaning forward, tingling or numbness in the thighs or buttocks while sitting, or a symptom pattern compatible with pudendal territory pain**. In such combinations, prolonged occupational sitting supports a **mechanical–neuropathic** or **mechanical–myofascial** interpretation. The survey is therefore using this item not simply to record sedentary behaviour, but to test whether the patient's pelvis is repeatedly exposed to the exact kind of loading known to aggravate many chronic pelvic pain phenotypes.

It is also important to distinguish this answer from later "sitting worsens pain" items. Here, Point 3 is not yet asking whether sitting directly causes flares. It is establishing that the patient is chronically exposed to a high-risk mechanical environment. A man whose job requires long sitting may later report no major sitting-related flare, in which case sitting may be background load but not the dominant symptom trigger. By contrast, if both are present, the case for a sitting-dependent pelvic pain phenotype becomes much stronger.

After work I also spend long periods sitting at home

This option is especially valuable because it distinguishes between **occupational exposure** and **near-continuous daily exposure**. A patient may already have a sitting-heavy job, but if he also spends much of his non-working time sitting at home, the total daily pelvic compression time may become clinically decisive. In such a pattern, the pelvic system is not receiving meaningful offloading periods, movement variability, or circulatory reset. This can perpetuate pelvic floor overactivity, venous stasis, gluteal deconditioning, hip stiffness, abdominal wall shortening, and irritability of pressure-sensitive pelvic tissues.

Clinically, this answer often matters most when paired with **late-day worsening, evening pain, pelvic heaviness, bladder pressure, rectal fullness, hip stiffness, low back stiffness, and**

symptom relief with walking or lying down. In those combinations, the interpretation may extend beyond simple nerve compression and include **mechanical, vascular, and muscular persistence mechanisms**. The body is not only exposed to sitting as a trigger; it is deprived of the recovery conditions needed to reverse the effects of sitting. That distinction is very important in chronic pain.

This answer may also help explain why some patients describe symptoms that are "worse by the end of the day" even when no single dramatic trigger is obvious. The clinical meaning is cumulative load. The pelvis is being held for many hours in a state that may increase local pressure, reduce movement diversity, and sustain neuromuscular vigilance.

My job requires lifting heavy objects

This option points toward a different kind of background load: not compressive sitting, but **repeated increases in intra-abdominal pressure and trunk stabilization demand**. Heavy lifting can place significant strain on the abdominal wall, diaphragm, pelvic floor, lumbar spine, hip stabilizers, and fascial support structures. If those systems are poorly coordinated, chronically tense, or already sensitized, lifting may become a persistent biomechanical contributor to pelvic pain.

In interpretation, this answer is especially important when later sections show **worsening with heavy lifting, increased abdominal pressure, coughing, bending, hip pain, low back stiffness, pelvic floor pain during contraction, constipation-related pain, or a history of pelvic or spinal injury**. Together, these findings support a **biomechanical–pelvic floor** or **biomechanical–neural** phenotype. In such patients, the pain is often not due to "the pelvic organs alone," but to the repeated failure of the lumbopelvic system to distribute load without over-recruiting protective tension.

This answer should also make the evaluator think about movement patterns rather than only symptom location. A man who lifts at work may describe pain "behind the testicles," "in the prostate area," or "in the bladder region," but the actual perpetuating mechanism may involve poor force transfer across the abdomen, pelvis, hips, and spine. EAU guidance explicitly recognizes functional abnormalities and multisystem contributions in chronic pelvic pain phenotyping, which is highly relevant here.

I currently have or previously had a high sexual drive

This answer requires especially careful interpretation. A high sexual drive is **not itself a pathology** and should never be framed as inherently abnormal. In the context of this survey, its purpose is to determine whether the patient may have had **high cumulative exposure to sexual arousal and ejaculatory load**, because in some men with CPPS this can be clinically relevant. The key interpretive point is not libido as a trait, but whether high sexual drive has translated into repeated physiological stress on an already sensitive pelvic system.

The most responsible medical interpretation is therefore conditional. This answer becomes meaningful primarily when it later coexists with **pain during ejaculation, pain immediately or hours after ejaculation, worsening with frequent daily ejaculation, difficulty relieving sexual tension, penile hypersensitivity, compulsive sexual behaviour, pornography-driven overstimulation, edging-like patterns, or pain in the seminal vesicle region**. In such combinations, high sexual drive may indicate that the pelvis is being exposed to **repetitive ejaculatory loading without adequate symptom-free recovery time**. AUA materials on male chronic pelvic pain specifically note that pelvic floor myalgia/dysfunction is common and that **pain with ejaculation is a clinically important clue**.

However, it is important not to overstate the evidence. Current medical knowledge does **not** support a universal rule that high libido or frequent ejaculation is harmful for all men with CPPS, nor does it justify a blanket statement that it "damages" the prostate or seminal vesicles. What it does support is a **phenotype-based interpretation**: in some men, frequent ejaculation may aggravate pain; in others, regular ejaculation may relieve congestion-related symptoms; and in some, both too much and too little sexual activity may be destabilizing. Therefore, in Point 3, high sexual drive should be understood as a possible **exposure amplifier**, not a diagnosis and not a moral category.

A further nuance is important here. If a respondent later reports **seminal vesicle-region pain, reduced semen volume, weak ejaculation, or pain after multiple ejaculations in one day**, then high sexual drive may be relevant because it increases the probability of repeated symptomatic activation before the deeper ejaculatory apparatus has returned to baseline. That is a clinical inference about symptom load and recovery, not proof of structural injury in every case.

Under physiological conditions, male sexual function follows a balanced cycle of tension buildup and ejaculation, allowing adequate recovery of the pelvic floor, prostate, seminal vesicles, and associated neural pathways. However, in modern environments, frequent external stimulation may lead to ejaculation patterns that exceed the organism's recovery capacity, particularly in susceptible individuals. While frequent ejaculation is not inherently harmful, problems may arise when a chronic load–recovery imbalance develops.

One of the primary consequences is pelvic floor muscle overuse, leading to chronic hypertonicity, impaired relaxation, and trigger point formation. This creates a self-perpetuating cycle of pain → muscle guarding → further pain. Simultaneously, repeated stimulation may activate neurogenic inflammation, in which peripheral nerves release mediators such as substance P and CGRP, increasing local sensitivity and lowering pain thresholds.

Substance P plays a central role in this process by linking the nervous system, immune system, and local tissues. Elevated levels contribute to pain persistence, hypersensitivity, and activation of mast cells. Conversely, clinical improvement is often associated with reduced substance P signaling, which can occur through interventions targeting multiple pathways, including:

- relaxation of the pelvic floor (reducing nociceptive input),
- modulation of neural activity (e.g., neuromodulators, stress reduction),
- reduction of inflammatory signaling (e.g., short-term steroid response in selected cases),
- stabilization of mast cells and histamine pathways (e.g., antihistamines, quercetin, DAO).

In a subset of patients, mast cell activation (MCAS-like mechanisms) further amplifies this loop. Mast cells release histamine and other mediators that interact with substance P, reinforcing both immune and neural sensitization. This may manifest as burning pain, urgency, pressure, and sensitivity to stress, food, or mechanical stimuli, forming a histamine-reactive phenotype.

The convergence of muscle overuse, neurogenic inflammation, and immune activation can lead to a state of non-bacterial inflammatory pelvic pain, which clinically resembles prostatitis but lacks microbiological confirmation. As a result, patients are often repeatedly treated with antibiotics despite negative findings, delaying appropriate recognition of the underlying mechanisms.

In early stages, symptoms may remain reversible, but with persistent dysregulation, the condition may evolve into a chronic state characterized by sensitization, autonomic imbalance, and reduced regenerative capacity, particularly with increasing age.

From a clinical perspective, after repeated negative microbiological results, diagnostic focus should shift toward pelvic floor dysfunction, neurogenic sensitization, autonomic dysregulation, and immune/histamine-related mechanisms, rather than continued emphasis on infection.

In summary, chronic pelvic pain should be viewed as a multisystem disorder, in which behavioral factors such as sexual activity may act as modulators of underlying vulnerability. Improvement is most likely achieved through simultaneous modulation of muscular, neural, and immune pathways, leading to reduction of key mediators such as substance P and restoration of physiological balance within the pelvic system.

I currently have or previously had a low sexual drive

I currently have or previously had a low sexual drive

Low sexual drive has a distinct interpretive meaning and should be approached from a multidimensional perspective. In the context of chronic pelvic pain, reduced libido is often not a primary cause, but rather a **secondary adaptation to ongoing physiological and psychological stress within the pelvic system**.

In many patients, low sexual drive reflects the cumulative effects of chronic pain, anticipatory fear of symptom exacerbation, sleep disturbance, autonomic dysregulation, hormonal imbalance (e.g., reduced testosterone or altered prolactin/SHBG levels), and repeated negative sexual experiences. Over time, the sexual system may lose resilience, as arousal and ejaculation become associated with discomfort, fatigue, or worsening symptoms rather than relief.

From a physiological standpoint, low libido may also be linked to **mechanical and inflammatory factors within the prostate–seminal vesicle complex**. In patients with infrequent ejaculation, there may be a tendency toward **relative stagnation of prostatic and seminal vesicle secretions**, which in some individuals can contribute to increased intraductal pressure, local congestion, and irritation of surrounding tissues. In advanced or long-standing cases, imaging such as high-resolution MRI (e.g., 3T) may reveal **features consistent with chronic changes**, including post-inflammatory alterations, fibrosis, microcalcifications, or calcified deposits within the prostate. These structural findings may, in certain cases, interfere with normal fluid drainage and contribute to a **chronic low-grade inflammatory environment**.

Such conditions may also promote **neurogenic inflammation**, in which sensitized nerve endings release mediators (e.g., substance P, CGRP), further amplifying pain perception and local immune responses. In a subset of patients, persistent antigen exposure or tissue damage may contribute to **immune dysregulation**, including mechanisms resembling low-grade autoimmune or autoinflammatory activity, although this remains an area of ongoing research rather than a fully defined clinical entity.

Importantly, reduced sexual activity can itself reinforce this cycle. Limited ejaculation may contribute to **reduced clearance of secretions**, while ongoing pelvic floor tension and impaired relaxation may further hinder effective outflow. This creates a feedback loop involving congestion, inflammation, and neuromuscular dysfunction.

At the same time, low sexual drive may also reflect **behavioral and central adaptations**. If sexual activity has repeatedly triggered pain, the nervous system may begin to associate arousal with threat rather than reward. This leads to a form of **conditioned avoidance**, where libido decreases as a protective response. In such cases, reduced sexual drive should not be interpreted purely through hormonal or structural factors, but also as part of a **pain-conditioned neurobehavioral adaptation**.

Therefore, this response is most meaningful when considered in combination with other findings, such as erectile dysfunction, difficulty achieving orgasm, post-ejaculatory pain, pelvic floor dysfunction, imaging abnormalities, or evidence of inflammatory or immune-related activity. In this broader context, low sexual drive serves as an indicator that the pelvic system is operating under chronic strain, involving interactions between **mechanical, inflammatory, neural, immune, and psychological mechanisms**, rather than representing a single isolated dysfunction.

Other (please specify)

Although this option is technically open-ended, it has real clinical value. It allows the patient to report important contextual exposures that are not otherwise captured by the structured choices. Examples might include vibration-heavy work, prolonged motorcycle riding, extreme athletic training, repeated exposure to cold, stimulant use, an unusual sexual pattern, severe shift-work sleep disruption, or a long-standing gastrointestinal problem. Such information can be decisive when the standard categories only partially fit the case.

Summary of Point 3

Point 3 is a **background phenotype question**. Its purpose is to show the daily regulatory and mechanical environment in which the pelvic pain syndrome has developed. Significant job stress supports a **psychophysiological/autonomic loading model**. Prolonged sitting at work and at home supports a **mechanical-compressive and often neuropathic or myofascial model**. Heavy lifting supports a **biomechanical and pressure-related pelvic floor model**. High sexual drive is relevant when it is accompanied by later evidence of **repetitive ejaculatory overloading or sexual-triggered pain**, whereas low sexual drive more often reflects **secondary sexual suppression due to pain, fear, hormones, fatigue, or mood burden**. On its own, Point 3 does not diagnose the patient; it defines the functional terrain against which the rest of the questionnaire must be interpreted.

Point 4. Onset of the Disease. How did your symptoms begin?

(select all that apply)

In the current questionnaire, Point 4 includes the following options: **suddenly; gradually over several months; gradually over several years; after a urinary tract infection; after a sexually transmitted infection (STI); after intense sexual activity (multiple ejaculations in one day); after a long period of sexual abstinence; after severe stress; after prolonged sitting; after ejaculation combined with prolonged sitting; after frequent interrupted intercourse (coitus interruptus); after frequently delaying ejaculation during intercourse; after a pelvic injury; after spinal injury or spinal disease; after frequent cycling; after pelvic surgery; after urinary catheterization; after antibiotic treatment; after hormonal therapy; after intense physical exercise; after an episode of constipation; after gastrointestinal illness; after heavy lifting; after use of psychoactive substances or stimulants; symptoms triggered by cold exposure; other (please specify).**

Purpose of Point 4

Point 4 is one of the most clinically valuable questions in the entire survey because it attempts to identify the **initiating pathway** of the disorder. Chronic pelvic pain often becomes multi-mechanistic over time, but the way it began frequently reveals the **first system that destabilized**. In

clinical practice, this is extremely important. A pain syndrome that began after infection may evolve very differently from one that began after severe stress, a period of sexual overload, heavy sitting, constipation, pelvic injury, or gastrointestinal illness.

This question is therefore not simply historical. It is mechanistic. It asks: What was the original switch? Was the pelvis first challenged by inflammation, mechanical compression, sexual overloading, autonomic shock, bowel dysfunction, or structural trauma? Even if the patient now has a far more complex picture, understanding onset helps distinguish **primary trigger** from **secondary maintenance mechanisms**. EAU guidance explicitly notes that chronic pelvic pain classification should consider symptoms, signs, and investigative findings within a phenotype framework and that end-organ labels alone may be misleading.

Suddenly

A sudden onset strongly suggests that the patient can identify a relatively discrete initiating event or short time window in which the pelvic pain system changed state. Clinically, this often supports a trigger such as infection, acute stress, unusual sexual loading, mechanical compression, trauma, catheterization, stimulant exposure, or extreme exertion. Sudden onset does not tell us which trigger was involved, but it does make a **trigger-linked model** more likely.

This answer becomes particularly informative if later sections reveal that symptoms have remained persistent despite normal cultures or normal routine imaging. In such a case, the most likely interpretation is often that an acute event triggered a longer-lasting change in pelvic floor tone, peripheral nerve reactivity, sensory gain, or autonomic balance. In other words, the original event may have ended, but the pain system did not reset. That is a classic pattern in chronic pain evolution.

Gradually over several months

A gradual onset over several months suggests a process that built progressively rather than a single abrupt event. This pattern often fits **cumulative strain**, such as prolonged stress, chronic sitting, mounting pelvic floor overactivity, evolving bowel dysfunction, disturbed sleep, repeated but subthreshold sexual overloading, or slowly increasing urinary hypersensitivity.

This answer is particularly important because it shifts interpretation away from “What exact event caused this?” toward “What ongoing exposure progressively altered the pelvic system?” In many men, this is the most realistic onset pattern for psychophysiological or myofascial CPPS. If later questionnaire sections show occupational stress, breathing dysfunction, pelvic floor tightening, constipation, gradually worsening urinary urgency, and no clear infectious correlate, then this onset pattern strongly supports a **slowly consolidating functional pelvic pain phenotype**.

Gradually over several years

This answer suggests an even slower accumulation of dysregulation. Rather than one event or a short destabilization period, it implies that the disorder emerged through a long sequence of **unresolved micro-loads**: sitting, tension, bowel issues, poor recovery, cumulative sexual triggers, chronic autonomic overdrive, spinal compensation, or evolving post-inflammatory tissue consequences.

Clinically, gradual onset over years often indicates that by the time the patient seeks help, the syndrome is already deeply layered. If later sections show multiple symptom domains — urinary, sexual, bowel, neurological, mechanical, and psychological — this answer fits a chronic multi-axis evolution particularly well. It also increases the probability that some patients may already show

secondary tissue-level findings, not because the condition was "structural from the start," but because long-term dysfunction and recurrent flares may eventually leave detectable biological traces.

After a urinary tract infection

This option suggests a **post-infectious onset pathway**. It is extremely important not to overinterpret it as evidence of ongoing infection. The clinical significance lies in the possibility that an infection may have acted as the initial nociceptive or inflammatory trigger, after which the patient transitioned into persistent pain despite eradication of the organism. This is a well-recognized pattern in chronic pain medicine: the initiating injury resolves, but the pain system remains activated.

This answer becomes especially strong if later urine cultures are negative, but the patient continues to report urethral burning, bladder pressure, urgency, post-void pain, or pelvic floor tightening. In that setting, the likely interpretation is not "missed infection," but **post-infectious sensitization, pelvic floor guarding, residual inflammatory vulnerability, or pain now being maintained by noninfectious mechanisms**. If a respondent later reports meaningful benefit from short steroid courses or antihistamines elsewhere in the survey, that may support an inflammatory or immune-mediated persistence model, but that interpretation belongs to later treatment-response sections rather than this onset item alone.

After a sexually transmitted infection (STI)

This onset pattern is similar to post-UTI onset but places greater emphasis on the **urethra, ejaculatory tract, prostate region, and seminal vesicle region**. A prior STI may act as a direct inflammatory insult to these structures or may leave behind persistent local hypersensitivity even after microbiological cure.

This answer becomes highly relevant if later sections show **pain during or after ejaculation, seminal vesicle-region pain, burning in the urethra after ejaculation, reduced semen volume, weak ejaculation, or deep pain behind the testicles/prostate region**. In such a case, the clinical model may include post-infectious tissue irritability plus ongoing nerve and muscle amplification. Again, the correct interpretation is usually not "the infection is still there," but "the infection may have been the original trigger for a pain syndrome that later became self-maintaining."

After intense sexual activity (multiple ejaculations in one day)

This is a classic **sexual overloading onset pattern**. The item explicitly refers to multiple ejaculations in a short period, which increases the plausibility of acute functional overload of the pelvic floor, ejaculatory apparatus, peri-prostatic tissues, urethra, and local sensory nerves. In some men, especially those with an already reactive pelvic system, this may serve as the initiating event that converts normal physiology into prolonged symptom reactivity.

This answer should be interpreted carefully and professionally. It does not prove that ejaculation is inherently harmful, nor does it justify universal advice that men with CPPS should avoid ejaculation. Instead, it indicates that **in this respondent**, symptom onset may have followed unusually high ejaculatory load relative to his tissue and neuromuscular tolerance at that time. The interpretation becomes much stronger if later sections show **worsening with frequent daily ejaculation, post-ejaculatory pain, seminal vesicle-region pain, urethral burning after ejaculation, difficulty relieving sexual tension, or a high sexual drive associated with repeated symptom-provoking behaviour**. AUA sources also highlight that pain with ejaculation is

clinically meaningful and often points toward pelvic floor dysfunction/myalgia within male chronic pelvic pain.

After a long period of sexual abstinence

This answer suggests a different sexual mechanism from the previous one. Here the interpretive emphasis shifts toward **congestion, pressure build-up, altered sexual tension regulation, or destabilization when sexual activity resumes after prolonged inactivity**. In some men, especially those who later report relief after ejaculation and worsening after abstinence, this pattern may indicate a congestion-sensitive phenotype rather than an overuse phenotype.

Clinically, this item becomes most meaningful when combined with later responses showing **worsening after several days or weeks of abstinence, relief after ejaculation, pain in the seminal vesicle region, pelvic heaviness, or deep prostate-region fullness**. In that context, the onset interpretation leans toward a **stasis/congestion-related sexual phenotype**, even if nerve and muscle amplification later develop on top of it.

After severe stress

This is one of the most important onset options in the survey. Severe stress can act as a major autonomic transition point, shifting the body into a prolonged state of **hypervigilance, sympathetic dominance, altered breathing, pelvic floor guarding, and increased pain sensitivity**. In some men, the onset of CPPS after severe stress is not incidental but highly mechanistic: the pelvic system becomes the site where chronic stress physiology is expressed.

This answer becomes particularly powerful if later sections show **occupational stress, "on edge" feelings, difficulty relaxing, chest breathing, breath-holding, worsening during stress or anxiety, symptom amplification during body focus, poor sleep, and lower-abdominal cramping under stress**. In that combination, severe stress is not just a historical event; it likely marks the beginning of a chronic **neuro-autonomic / psychophysiological phenotype**. Modern pelvic pain frameworks explicitly recognize cognitive, behavioural, sexual, and emotional consequences as part of the phenotype, not as optional add-ons.

After prolonged sitting

This answer points strongly toward a **mechanical-compression onset pattern**. It suggests that the pelvis may have first become symptomatic under sustained perineal loading, pelvic floor overuse, pudendal-region pressure, or venous pooling associated with immobility.

Its significance rises sharply if later answers include **pain worsened by sitting, driving, cycling, leaning forward, perineal pain, pain in the anus or genital region, tingling or numbness while sitting, and a Nantes-like pudendal territory pattern**. In such cases, "after prolonged sitting" is one of the clearest possible clues to a **mechanical–neuropathic** or **mechanical–myofascial** onset.

After ejaculation combined with prolonged sitting

This is one of the most clinically meaningful combined triggers in the questionnaire because it joins two major systems of load: **sexual activation and mechanical compression**. Ejaculation involves pelvic floor contraction, autonomic activation, and deep pelvic organ engagement; prolonged sitting adds perineal pressure and impaired unloading. The combination can therefore act as a powerful trigger in susceptible patients.

If later responses show both **post-ejaculatory pain and sitting-triggered pain**, this onset answer supports a very characteristic **neuro-muscular pelvic overload phenotype**. In such patients, it is often not useful to ask whether the "real problem" is sexual or mechanical; the clinical reality is that the two triggers interact and mutually amplify each other.

After frequent interrupted intercourse (coitus interruptus)

This answer suggests a pattern of repeatedly initiating the sexual response but interrupting its completion. Clinically, this may matter because it can leave the patient in a state of **sustained arousal without full physiological resolution**, potentially increasing pelvic floor tension, deep muscular guarding, and unresolved sexual autonomic activation.

This interpretation becomes stronger if later sections show **difficulty relieving sexual tension, post-ejaculatory pain, worsening with frequent ejaculation, high sexual drive, or compulsive sexual behaviour**. The significance of this answer lies less in the moral or behavioural label and more in the repeated **failure of the sexual system to complete and recover normally**.

After frequently delaying ejaculation during intercourse

This item points toward an edging-like onset pattern. The likely interpretation is repeated exposure to **prolonged pelvic floor contraction, extended autonomic arousal, delayed completion of the ejaculatory reflex, and repeated sensory overloading of the pelvic sexual system**.

This is one of the places where the survey's attention to sexual physiology is especially advanced. The issue is not merely "how often sex occurred," but **how the sexual response was regulated**. If later responses show **difficulty relieving sexual tension, pain during ejaculation, penile hypersensitivity, post-ejaculatory pain, and worsening with frequent daily ejaculation**, this onset pattern strongly supports a **sexual overregulation / pelvic floor overload phenotype**.

After a pelvic injury

A pelvic injury suggests that the syndrome may have begun with a genuine nociceptive or structural insult. However, chronic pelvic pain after injury is rarely explained by the injury alone once symptoms persist over time. The longer-term clinical model often includes **protective muscle guarding, pain amplification, altered movement strategy, fear of loading, and secondary nerve irritability**.

This onset answer becomes particularly important if later sections show **pelvic floor spasm, hip dysfunction, rectal fullness, nerve symptoms, or imaging evidence of post-traumatic or post-inflammatory changes**. In such cases, the correct interpretation is often a **mixed tissue–nerve–muscle phenotype** rather than a simple "old injury" explanation.

After spinal injury or spinal disease

This answer immediately raises the relevance of the **lumbosacral spine, sacral roots, referred pain, and neural pathway involvement**. It suggests that the pelvis may not be the original site of pathology, even if it is now the main site of pain perception.

If later sections show **low back pain, sacral pain, radiating pain, numbness, tingling in the thighs or legs when sitting, suspected sacral nerve irritation, or abnormalities on lumbar/sacral MRI**, then this onset pattern strongly supports a **spine–pelvis neural interaction phenotype**. EAU guidance also emphasizes that the site where pain is perceived may not be the true

site of pain generation.

After frequent cycling

Cycling is a classic mechanical trigger for the **perineal–pudendal–pelvic floor axis** because of direct saddle pressure and repeated loading in the pudendal territory. When symptoms begin after frequent cycling, clinicians should strongly consider **perineal compression, neural irritation, soft tissue overload, and pelvic floor reactivity** as candidate mechanisms.

This answer becomes especially compelling if later sections show **pain with sitting, driving, genital/perineal pain, tingling while sitting, and a Nantes-like pattern**. It is one of the clearest mechanical onset items in the survey.

After pelvic surgery

Pelvic surgery may initiate chronic pelvic pain through multiple pathways: **scar formation, nerve irritation, altered local mechanics, post-operative guarding, vascular change, or persistent sensitization after tissue injury**. This answer strongly supports a biologically anchored onset, although the chronic persistence of symptoms often reflects more than postoperative tissue change alone.

If later sections show **myofascial symptoms, neuropathic symptoms, allodynia, altered bowel or urinary behaviour, or imaging findings**, then this onset pattern should be interpreted within a **post-surgical mixed phenotype** rather than reduced to either “scar pain” or “functional pain” alone.

After urinary catheterization

This answer suggests an onset pathway involving **urethral irritation, bladder neck irritation, local sensory nerve sensitization, or pelvic floor guarding triggered by instrumentation**. It becomes especially meaningful if later sections show **burning during or after urination, urethral burning after ejaculation, bladder hypersensitivity, urgency, and negative infection work-up**.

In such cases, the likely model is not necessarily structural damage, but **instrumentation-triggered sensory dysregulation** in a sensitive lower urinary tract/pelvic system.

After antibiotic treatment

This option can be interpreted in at least two clinically plausible ways. First, it may mean that the patient originally received antibiotics for suspected prostatitis or UTI, but symptoms persisted, suggesting that the condition either was never bacterial or became **post-infectious / post-treatment chronic pain**. Second, it may indicate that the treatment itself altered the bowel or immune environment, contributing to dysbiosis, bowel dysfunction, food reactivity, or histamine-related worsening.

This answer becomes more significant when combined with later reports of **bloating, IBS, food-triggered pelvic pain, low-FODMAP response, low-histamine response, or chronic nonbacterial cultures**. In those cases, “after antibiotic treatment” can be a clue that the syndrome shifted from a presumed infectious narrative into a **gut–immune–pelvic pain phenotype**.

After hormonal therapy

This answer suggests that symptom onset may have been linked to changes in the **androgen–estrogen balance, sexual function regulation, prostate/seminal vesicle physiology, smooth muscle tone, or general endocrine stability**. It is especially important because hormonal manipulations can affect libido, ejaculation, urinary dynamics, and pain sensitivity.

Its significance increases if later sections show **libido change, erectile dysfunction, altered semen volume, low or high sexual drive, abnormal testosterone/estradiol/SHBG/prolactin results, or symptom worsening with testosterone therapy or other hormone-related medications**. In this context, onset after hormonal therapy supports an **endocrine-sensitive phenotype** rather than a purely mechanical or purely infectious one.

After intense physical exercise

This answer suggests a load-related onset involving **pelvic floor overrecruitment, abdominal pressure, spinal or hip compensation, or exertion-triggered pain in a vulnerable pelvic system**. It is particularly relevant when the later profile includes **pain with heavy lifting, increased abdominal pressure, hip movement, lower back stiffness, pelvic muscle spasm, or symptom worsening with physical activity**.

The clinical interpretation here is often biomechanical rather than organ-specific. The patient may describe “prostate pain,” but the initiating mechanism may have been physical overload of the lumbopelvic–pelvic floor complex.

After an episode of constipation

This is a very important onset pattern because it directly links **bowel mechanics with pelvic pain onset**. Severe constipation can increase rectal pressure, strain the pelvic floor, provoke anal sphincter overactivity, increase local neural irritation, and alter pelvic sensory processing.

If later sections reveal **pain associated with constipation, difficulty passing stool, rectal fullness, anal pain, lower abdominal cramping under stress, bloating, and urinary cross-symptoms**, this answer strongly supports a **bowel–pelvic floor–pelvic pain phenotype**. It shows that the bowel may not be just an associated problem, but a primary trigger in the history of the syndrome.

After gastrointestinal illness

This answer suggests that the onset may have followed **intestinal infection, post-infectious IBS, dysbiosis, altered immune activation, or a disturbed gut–brain–pelvis axis**. In contemporary chronic pelvic pain thinking, this is highly plausible and clinically important.

It becomes especially meaningful if later sections show **IBS, bloating, food-triggered pelvic pain, FODMAP sensitivity, nausea during flare-ups, relief after bowel movement, or histamine-related food reactivity**. In such a pattern, gastrointestinal illness may have acted as the first destabilizer of a system that later expressed itself partly in the pelvis.

After heavy lifting

This answer overlaps somewhat with “after intense physical exercise,” but heavy lifting deserves distinct interpretation because it emphasizes **pressure transmission and abrupt load** more than general exertion. It suggests that a sudden or repeated increase in abdominal pressure and pelvic stabilization demand may have triggered the pain.

If later sections show **worsening with lifting, coughing, bending, pelvic floor contraction pain, low back stiffness, or hip–pelvis biomechanical factors**, then the onset is very consistent with a **pressure-triggered pelvic floor / lumbopelvic dysfunction phenotype**.

After use of psychoactive substances or stimulants

This onset pattern points toward a possible **autonomic, sensory, and neurochemical destabilization pathway**. Psychoactive substances and stimulants may acutely alter sympathetic tone, body awareness, anxiety, pain amplification, pelvic muscle tone, bladder activity, and sleep quality.

This answer becomes more plausible clinically if later sections show **palpitations, racing thoughts, sleep disturbance, stress sensitivity, cold extremities, heightened body monitoring, urinary urgency, or nerve-like pain**. It supports a model in which the onset involved a sharp disturbance of **neuro-autonomic regulation** rather than a local pelvic injury alone.

Symptoms triggered by cold exposure

Cold exposure as an onset or precipitating factor supports a **muscular, vascular, autonomic, or mast-cell-reactive** phenotype. Cold may increase muscle tension, reduce local blood flow, heighten sensory defensiveness, and in some susceptible patients act as a trigger for broader immune reactivity.

This answer becomes especially convincing when paired with later reports of **worsening in cold, relief with heat, pelvic floor spasm, hypersensitivity to cold, skin reaction to cold, or other autonomic features**. In such cases, cold should be interpreted not as a trivial exposure but as a real destabilizing input to a sensitive pelvic system.

Other (please specify)

This option captures unusual but sometimes highly revealing onset narratives, such as a specific medication, a unique athletic exposure, a major life event, a vaccination or infection not otherwise listed, a period of extreme sleep deprivation, or a highly specific sexual or postural history. In some patients, the most clinically meaningful onset clue will be found here rather than in the structured list.

Summary of Point 4

Point 4 is the survey's **primary trigger-identification question**. Its purpose is to determine how the pelvic pain syndrome first entered the patient's life: through infection, sexual loading, abstinence, stress, sitting, injury, bowel dysfunction, endocrine disturbance, mechanical overload, autonomic shock, or another route. A sudden onset suggests a discrete switch event; gradual onset suggests cumulative dysregulation; infection-related onset supports a post-infectious pathway; sexual overload and delayed/interrupted ejaculation support a **sexual–pelvic floor overload phenotype**; prolonged sitting and cycling support a **mechanical–pudendal phenotype**; severe stress supports a **neuro-autonomic / psychophysiological phenotype**; constipation and gastrointestinal illness support a **gut–pelvic floor / gut–immune phenotype**; and trauma, surgery, catheterization, or spinal disease support a **mixed tissue–nerve–muscle phenotype**. Point 4 is especially valuable because even when the present-day picture has become highly complex, the onset history often reveals the first system that failed.

Point 5. Pain Location. Do you experience pain in:

(select all that apply)

In the current version of the survey, Point 5 includes the following options: **the perineum; the testicles; the shaft of the penis; the tip of the penis (glans penis); the suprapubic region; the groin; the lower abdomen; the lower back (lumbar spine region); the sacrum; the anus; the inner thighs; the hips; pain suggesting the urinary bladder region; behind the testicles / prostate region; seminal vesicle area; other (please specify).**

Purpose of Point 5

Point 5 is designed to build an **anatomical and functional pain map**. In chronic pelvic pain, location does not simply tell us where the patient feels pain. It helps us infer **which structures, pathways, and physiological systems may be involved**. This is crucial because pelvic pain is often not generated exactly where it is felt. It may be **referred**, **radiating**, **centrally amplified**, or **secondarily maintained** by protective muscular tension or neural hypersensitivity. In male chronic pelvic pain, current guideline thinking explicitly recognizes that the site where pain is reported may differ from the site where the pathology or pain-generating process originally began.

The purpose of this item is therefore not descriptive alone. It is interpretive. A perineal pain pattern suggests something different from bladder-region pain, glans burning, low-back pain, or deep pain behind the testicles. Likewise, a single focal location has different implications from a wide pain map involving the perineum, penis, anus, thighs, hips, and lower back all at once. The broader and more distributed the pain map, the more likely one must consider a **mixed or system-level phenotype**, including pelvic floor overactivity, neuropathic spread, cross-sensitization between organs, or central amplification.

Location also becomes especially meaningful when interpreted together with later questionnaire sections. For example, deep pain “behind the testicles / prostate region” means something different if it is later associated with **pain after ejaculation, reduced semen volume, seminal vesicle-region pain, or partial relief from short steroid courses** than if it is associated primarily with **sitting pain, breathing dysfunction, and pelvic floor spasm**. Similarly, bladder-region pain carries different implications if later sections show **urgency, bladder pain on filling, relief after urination, or response to antihistamines**, compared with a case dominated by **pelvic floor clenching and bowel-related triggers**. This is why Point 5 should be treated as one of the survey’s main **structural-phenotypic anchors**.

The perineum

Perineal pain is one of the most classic and diagnostically important pain locations in male CPPS. It strongly raises suspicion of involvement of the **pelvic floor muscles, pudendal territory soft tissues, anal sphincter complex, deep myofascial structures, or the pudendal nerve and its surrounding fascial environment**. Clinically, perineal pain often sits at the centre of a **pelvic floor hypertonicity phenotype**, especially when it later coexists with sitting intolerance, a “golf ball” sensation, difficulty relaxing the pelvic floor, rectal fullness, post-ejaculatory worsening, or trigger-point sensitivity on pelvic floor examination.

At the same time, perineal pain is not exclusively muscular. If it is described as **burning, electric, prickling, or accompanied by allodynia**, the interpretation shifts more strongly toward a **neuropathic or pudendal neuralgia-like component**. If it is described as **deep pressure, heaviness, or fullness**, then vascular congestion, deep fascial tension, or pressure-related pelvic

loading may also need to be considered. Perineal pain is therefore a highly informative location because it can sit at the crossroads of **muscular, neural, vascular, and pressure-based mechanisms**.

The testicles

Testicular pain is common in chronic pelvic pain syndromes, but it is very often **referred pain rather than primary scrotal pathology**. This is a key point. In a patient with normal scrotal imaging and no ongoing infection, testicular pain may still be real and significant, but its origin may lie in the **pelvic floor, spermatic cord, inguinal region, lower abdominal wall, seminal tract, pudendal pathways, or lumbosacral neural structures**. Modern male chronic pelvic pain guidance explicitly recognizes the importance of referred pain patterns and overlapping pelvic phenotypes.

This location becomes particularly meaningful if later responses show **pain after ejaculation, seminal vesicle-region pain, groin pain, inner thigh pain, low-back or sacral pain, or worsening with sitting**. In that setting, testicular pain should not be interpreted narrowly as a scrotal problem alone. It often reflects a **deeper pelvic referral network**. If the patient later reports testicular pain specifically after ejaculation, then the symptom may also indicate that the sexual system is triggering pain that is subsequently perceived in the testes rather than exclusively in the prostate or seminal vesicle area.

The shaft of the penis

Pain in the shaft of the penis generally pushes interpretation toward a **sensory-neural or urethral–neurogenic axis** rather than a classic deep prostate-only model. The penile shaft contains dense sensory innervation, so pain in this area is often especially important when the patient later reports **burning, tingling, prickling, hypersensitivity of the penis, post-ejaculatory urethral burning, or rapid ejaculation driven by penile hypersensitivity**. In such cases, shaft pain may reflect **peripheral sensory amplification, pelvic nerve irritability, referred pain from the pelvic floor, or a sensitized genital sensory system**.

If shaft pain occurs together with bladder symptoms, it may also suggest a **cross-sensitized pelvic sensory phenotype**, in which urethral and bladder inputs are being processed as part of the same pain network. If it occurs with strong sitting-dependence and pudendal features, it may fit a neuropathic-pudendal pattern. Thus, penile shaft pain should not be dismissed as non-specific; in the right context it is one of the clearer clues that the **pain phenotype has a strong sensory-neural component**.

The tip of the penis (glans penis)

Pain in the glans penis is especially important because patients often interpret it as “infection,” “urethritis,” or “prostate inflammation,” even when repeated microbiology is negative. In chronic pelvic pain, glans pain frequently points toward a **distal sensory expression of a more proximal pelvic process**, particularly urethral hypersensitivity, pelvic floor overactivity, or genital sensory amplification. It often becomes especially meaningful when paired with **burning during urination, burning after ejaculation, urgency, bladder hypersensitivity, and negative cultures**.

Clinically, glans pain may reflect irritation of the **urethral sensory axis**, but it can also be part of a broader neuropathic picture if associated with **allodynia, electric-shock sensation, or hypersensitivity to clothing and touch**. If later sections show that symptoms worsen after acidic

foods, citrus, tomatoes, coffee, or histamine-rich foods, then glans pain may also be part of a **bladder–urethral irritative phenotype with sensory overgain**, rather than evidence of persistent infection.

The suprapubic region

Suprapubic pain pushes interpretation toward the **bladder–lower abdominal interface**. This location is especially important if later urinary items reveal **bladder pain, pressure when the bladder fills, urgency, relief after urination, or diagnosis of painful bladder syndrome / interstitial cystitis**. In such cases, suprapubic pain supports a **bladder-centred phenotype** within the broader CPPS spectrum.

However, suprapubic pain can also arise in patients with **lower abdominal wall tension, bowel distension, chronic abdominal guarding, or stress-related cramping**. That is why this location should not automatically be equated with bladder pathology. If suprapubic pain later coexists with bloating, constipation, reflux, or stress-induced lower abdominal pain, then the location may reflect a **shared abdominal–pelvic tension pattern** rather than isolated bladder disease. In contrast, if it is strongly linked to bladder filling and improves after voiding, bladder involvement becomes more likely.

The groin

Groin pain introduces a strong **musculoskeletal and referral** dimension. It can reflect adductor overload, hip dysfunction, inguinal strain, fascial tension, spermatic cord referral, lower abdominal tension, or pain spreading from the pelvic floor. When groin pain is present, the evaluator should think beyond the prostate and ask whether the pelvis is functioning within a larger **hip–groin–pelvic floor biomechanical chain**.

This answer becomes more meaningful if later sections show **hip tightness, pain with hip movement, inner thigh pain, lower back stiffness, worsening with walking, bending, or sitting cross-legged**. In that context, groin pain supports a **myofascial or biomechanical phenotype** rather than a purely organ-based one. It may also coexist with sexual or urinary symptoms, which is exactly why it is such an important location in a multidimensional pelvic pain survey.

The lower abdomen

Lower abdominal pain is one of the more integrative locations in the survey because it sits at the intersection of **bowel, bladder, abdominal wall, autonomic stress response, and pelvic floor function**. This location becomes particularly important if the patient later reports **bloating, constipation, IBS, urgency, suprapubic pressure, or cramping under stress**. It may indicate that the pelvic pain syndrome is not confined to the perineum or prostate region but is part of a broader **abdomino-pelvic pain network**.

Clinically, lower abdominal pain may represent abdominal wall overactivity, bowel-related pressure transmission, bladder discomfort, or stress-linked autonomic cramping. Therefore, this location is often a clue that the patient's phenotype is more **whole-system pelvic** than strictly genital or prostate-centred.

The lower back (lumbar spine region)

Lumbar pain broadens the interpretation toward the **lumbopelvic system**. It raises the possibility that chronic pelvic pain is being influenced by lumbar mechanics, root irritation, trunk stabilization patterns, or referred pain from the lower spine. This location becomes highly relevant if later sections show **bending sensitivity, heavy lifting aggravation, spinal injury history, sacral**

symptoms, radiating pain, thigh or leg tingling while sitting, or abnormalities on lumbar/sacral MRI.

Importantly, low back pain in this context does not mean the pelvic pain is "actually just the spine." Rather, it suggests that the **pelvis and spine may be participating in the same pain network**. In chronic pelvic pain, lumbar dysfunction can intensify pelvic floor guarding, and pelvic pain can in turn alter lumbar mechanics. This mutual reinforcement is clinically common.

The sacrum

Sacral pain is especially significant because it anatomically and functionally links the pelvis to the **sacral nerve roots, sacroiliac region, deep posterior pelvic structures, and the neural pathways relevant to bowel, bladder, and sexual function**. In many patients, sacral pain is a clue that the pelvic syndrome has a more **posterior neural or lumbosacral** component than appears at first glance.

If sacral pain later coexists with **perineal pain, anal pain, sitting-related worsening, radiating neural pain, or suspected sacral nerve irritation**, then the phenotype may include a significant **neural–mechanical posterior pelvic component**. This location should therefore be taken very seriously in differential interpretation.

The anus

Anal pain is highly relevant to **pelvic floor hypertonicity, anal sphincter involvement, deep pelvic myofascial pain, and pudendal territory pain**. It is especially informative when combined with **rectal fullness, pain associated with constipation, difficulty passing stool, pelvic floor muscle spasm, or pain worsened by sitting**. In those combinations, anal pain often indicates that the anorectal segment of the pelvic floor is part of the active pain phenotype.

If anal pain is described more as burning, electric, or allodynic, the interpretation shifts more toward a **neuropathic-pudendal component**. If it is described more as pressure or fullness, then deep pelvic floor tension or bowel-related pressure may be more prominent. Thus, the anus as a pain location is diagnostically rich and should not be considered a minor variant of perineal pain.

The inner thighs

Inner thigh pain often reflects **referred myofascial pain, adductor involvement, obturator-region discomfort, neural referral, or lumbopelvic biomechanical spread**. It is particularly meaningful when paired with **groin pain, hip tightness, sitting cross-legged aggravation, pain with hip movement, or numbness/tingling in the thighs when sitting**. In such cases, the patient likely has more than a purely organ-based pelvic pain syndrome.

Inner thigh pain helps reveal when the pain map is extending into the **musculoskeletal envelope around the pelvis**. This is often a sign of a myofascial or neural referral pattern rather than isolated prostate pathology.

The hips

Hip pain is clinically important because hip dysfunction can either contribute to pelvic pain or be mistaken for pelvic pain. The hip, groin, pelvic floor, and lower abdominal wall form a highly integrated movement and load-sharing system. Therefore, hip pain in CPPS may indicate that the pelvis is operating within a **broader biomechanical problem**, especially if it is associated with **hip**

tightness, groin pain, pain with hip movement, lower back stiffness, or pain worsened by walking or sitting cross-legged.

This location is especially valuable because it helps prevent over-localization of the syndrome to the prostate or bladder when the actual perpetuating mechanism may involve **hip–pelvis load transfer and myofascial overload**.

Pain suggesting the urinary bladder region

This answer strongly supports a **bladder-centred pain interpretation**, especially if later urinary items show **bladder hypersensitivity, urgency, pain when the bladder fills, pelvic pressure when the bladder is full, and relief after urination**. In such cases, bladder involvement may be central rather than incidental.

This location becomes even more significant if later sections reveal **histamine-related triggers, painful bladder syndrome / interstitial cystitis diagnosis, worsening after acidic foods, or benefit from antihistamines**. Current evidence and clinical practice increasingly recognize that a subset of pelvic pain patients have an **immune–sensory bladder phenotype**, in which mast-cell or histamine-related mechanisms may amplify bladder-region pain. H1 and H2 antihistamines are included in your updated survey because some patients report meaningful benefit, which can support that subtype when interpreted in context.

Behind the testicles / prostate region

This is one of the most clinically loaded pain locations in the survey because many patients describe deep pelvic pain in exactly these terms. However, this location should **not** be interpreted automatically as proof that the prostate itself is the sole source. Deep pain behind the testicles may arise from the **prostate, peri-prostatic tissues, seminal tract, levator ani complex, deep myofascial structures, pelvic congestion, or referred pain from nearby neural structures**.

Its interpretation depends heavily on what else the patient reports. If this location later coexists with **pain after ejaculation, weak ejaculation, reduced semen volume, seminal vesicle-region pain, abnormal semen inflammatory markers, or partial response to short steroid courses**, then a **deeper inflammatory / post-inflammatory or ejaculatory-tract phenotype** becomes more plausible. Current medical understanding supports that short steroid responsiveness, when it occurs, may suggest an inflammatory component in selected patients, though it is not diagnostic by itself and must be interpreted cautiously. Steroids are therefore best seen as a possible clue to **inflammatory amplification**, not proof of one specific disease mechanism.

By contrast, if deep pain behind the testicles/prostate region is more strongly associated with **sitting, stress, pelvic floor clenching, rectal fullness, or relief with pelvic floor physiotherapy**, then the same location may be better understood as **deep pelvic floor or referred myofascial pain** rather than primarily gland-driven pain. This is exactly why this questionnaire treats location as a starting point rather than a final conclusion.

Seminal vesicle area

This is one of the most sophisticated items in the entire location section because it specifically addresses the possibility that the pain phenotype involves the **ejaculatory reservoir system**, not just the prostate in a generic sense. Pain in the seminal vesicle area becomes highly significant if later sections show **pain during ejaculation, pain after ejaculation, worsening after abstinence, worsening after frequent daily ejaculation, reduced semen volume, or weak ejaculation**.

This location is exactly where the issue of **sexual load versus recovery time** becomes clinically relevant. Current evidence does not support a blanket claim that frequent ejaculation damages the seminal vesicles or prostate in all men. However, in a patient whose symptoms are repeatedly provoked by multiple ejaculations in a short period, who has high sexual drive, post-ejaculatory pain, and deep seminal-vesicle-region discomfort, it is clinically reasonable to infer that the **ejaculatory system may not be recovering adequately between symptom-provoking events**. This is best understood as a **patient-specific tolerance and recovery problem**, not as a universal biological rule.

This location may also become more biologically persuasive if semen testing later shows **leukocytes, cytokines, aggregates/clumps, or other evidence of chronic inflammatory or post-inflammatory change**. In that context, the pain may reflect a combination of **deep tissue irritability plus nerve and muscle amplification** rather than a purely "functional" complaint.

Summary of Point 5

Point 5 is the questionnaire's **pain topography map**. Its purpose is to identify where the pain is felt and, more importantly, what anatomical and physiological systems are likely involved. **Perineum, anus, and prostate-behind-the-testicles pain** often support a deep pelvic floor, pudendal, or peri-prostatic phenotype. **Glans and penile shaft pain** often strengthen a sensory-neural or urethral hypersensitivity interpretation. **Suprapubic and bladder-region pain** push interpretation toward a bladder-centred or bladder–sensory phenotype, especially if later urinary items and antihistamine response support that view. **Groin, hips, inner thighs, lumbar pain, and sacral pain** point toward biomechanical, neural, or referred musculoskeletal contributions. **Seminal vesicle-region pain** is particularly important in patients with high sexual drive, frequent ejaculations, abstinence-related symptoms, or post-ejaculatory worsening, because it may indicate an ejaculatory-system phenotype in which sexual load and recovery balance are clinically relevant. Point 5 therefore does not simply tell us where the pain is; it tells us how to begin organizing the pelvic pain system itself.

Point 6. These above symptoms most often occur:

In the current version of the survey, Point 6 includes the following options: **at the moment of ejaculation; a few hours after ejaculation; a few days after ejaculation; independent of ejaculation; after long periods of sexual abstinence; after frequent ejaculations; after prolonged sitting; after exposure to cold; Other – please write.**

Purpose of Point 6

Point 6 is one of the most clinically important temporal-pattern questions in the survey. Whereas Point 5 maps **where** pain is felt, Point 6 clarifies **when** the same symptoms most commonly occur. This is essential because in CPPS, the timing of symptoms often reveals more about mechanism than location alone. A pain syndrome that appears exactly at ejaculation is different from one that appears hours later, days later, after abstinence, after frequent ejaculation, after sitting, or independently of any sexual event. Each timing pattern suggests a different relationship between trigger, tissue response, nerve response, and regulatory failure.

This item is especially valuable because it helps identify whether the pain is behaving more like a **sexual-load phenotype**, a **congestion/abstinence phenotype**, a **mechanical sitting phenotype**, a **cold-reactive muscular/autonomic phenotype**, or a **more independent chronic pain phenotype** that no longer depends on one dominant trigger. In men with chronic pelvic pain, ejaculation can function almost like a physiological stress test for the pelvic floor, urethra, prostate region, and

seminal vesicle region. Likewise, prolonged sitting can function as a compression test. Point 6 therefore asks not simply when symptoms happen, but **what kind of challenge most predictably destabilizes the system**.

At the moment of ejaculation

Symptoms that occur exactly at the moment of ejaculation strongly suggest that the pain is linked to the **ejaculatory event itself**, rather than to a delayed downstream process. This usually raises the importance of **pelvic floor contraction, urethral/prostatic smooth muscle activity, peri-prostatic mechanical strain, seminal tract tension, or immediate sensory triggering during orgasmic contraction**. AUA pelvic pain materials specifically note that **pain with ejaculation is a significant clue to pelvic floor myalgia or pelvic floor dysfunction**, which is highly relevant here.

This answer becomes even more meaningful if later sections show **pain during ejaculation, pain in the seminal vesicle region, weak ejaculation, reduced semen volume, urethral burning after ejaculation, difficulty relieving sexual tension, or a history of frequent delayed ejaculation / interrupted intercourse**. In such combinations, the mechanism often involves a **sexual–pelvic floor overload pattern**. If this timing pattern is accompanied by later evidence of inflammatory findings in semen or partial response to short steroids, then a deeper inflammatory or post-inflammatory contribution may also be considered, but the temporal signature still strongly suggests that the act of ejaculation is the main immediate load event.

A few hours after ejaculation

Pain occurring a few hours after ejaculation suggests a **delayed reactive phase** rather than immediate orgasmic pain. This timing often indicates that ejaculation is functioning as the initiating event, but the actual pain may be driven by what follows: sustained pelvic floor spasm, delayed neural sensitization, bladder or urethral irritation, autonomic rebound, vascular fullness, or local inflammatory signalling.

Clinically, this pattern is especially important because it often marks a system that is **unable to recover cleanly after sexual activation**. The pain is not simply "during sex" pain. It is post-activation dysregulation. If the patient later reports **burning after ejaculation, urinary urgency after sexual activity, sitting-triggered worsening after sex, or deep pain behind the testicles/prostate region**, then "a few hours after ejaculation" becomes a strong clue that the sexual system is provoking a **delayed neuro-muscular or irritative flare**. If there is later benefit from neuromodulators, pelvic floor relaxation, antihistamines, or selected anti-inflammatory measures, that would fit well with this interpretation.

A few days after ejaculation

This timing pattern is particularly interesting because it implies that ejaculation triggers a response whose peak occurs **well after the sexual event**. This often suggests a more delayed biological or regulatory cascade than immediate muscular contraction alone. In some patients, such a delay may fit **post-ejaculatory inflammatory amplification, immune or histamine-related reactivity, persistent tissue irritability in deep pelvic structures, or delayed resurgence of protective muscle guarding**.

This answer deserves especially careful interpretation if the patient later reports **seminal vesicle-region pain, deep prostate-region pain, reduced semen volume, post-ejaculatory fatigue, strong food/histamine triggers, or partial benefit from short steroid courses or antihistamines**.

In such cases, it may support the idea that the sexual event is not only a mechanical or muscular stressor but may also initiate a more delayed **inflammatory, neuroimmune, or deep tissue irritative response**. This does not prove inflammation in every case, but it makes that layer more plausible than in patients who hurt only at the exact moment of ejaculation.

Independent of ejaculation

If symptoms most often occur independently of ejaculation, then ejaculation is unlikely to be the dominant trigger, even if it sometimes modifies symptoms. This answer shifts interpretation away from a primarily ejaculatory or seminal-tract model and toward a broader chronic pain phenotype in which symptoms may be maintained by **sitting, bladder activity, pelvic floor tension, bowel dysfunction, stress reactivity, neural sensitization, vascular fullness, or mixed multi-system dysregulation**.

This timing pattern is especially important because it often marks a syndrome that has become **less trigger-specific and more continuously self-sustaining**. If later sections show pain in the pudendal territory, allodynia, poor sleep, autonomic symptoms, bladder hypersensitivity, or bowel-triggered pain, “independent of ejaculation” supports the view that the pain system has moved beyond a predominantly sexual phenotype. In some patients, it may also indicate that ejaculation is a relatively minor modifier compared with stronger drivers such as stress, sitting, or urinary filling.

After long periods of sexual abstinence

This answer supports a **congestion / abstinence-sensitive phenotype**. The likely clinical meaning is that prolonged lack of ejaculation is associated with **increasing deep pelvic fullness, unresolved sexual tension, pressure in the seminal/prostatic region, or symptomatic build-up that improves only after release**. This pattern is fundamentally different from the “frequent ejaculation worsens me” phenotype.

This answer becomes much stronger if later sections also show **relief after ejaculation, pain in the seminal vesicle region, reduced semen volume, deep pain behind the testicles/prostate region, or pelvic heaviness/fullness**. In such cases, abstinence may be relevant not because abstinence is universally harmful, but because **this specific patient’s pelvic system appears to become unstable when sexual activity is absent for too long**. This may reflect a congestion-sensitive or pressure-sensitive pattern, especially when pain is described as fullness, heaviness, or deep pressure rather than purely burning or electric.

After frequent ejaculations

This answer suggests a **sexual overloading pattern**. In this phenotype, the issue is not that ejaculation occurs, but that ejaculation occurs too frequently for the patient’s pelvic system to recover comfortably between events. This interpretation is especially important in patients who also report **high sexual drive, intense sexual stimulation, pain during or after ejaculation, seminal vesicle-region pain, urethral burning after ejaculation, or difficulty relieving sexual tension**. In such combinations, the clinical model is one of repeated pelvic activation with **insufficient symptomatic recovery time**.

This is the point at which the issue of “high libido and frequent ejaculations” must be handled carefully and professionally. Current medical evidence does not justify saying that frequent ejaculation is categorically harmful for all men, nor that it inherently injures the prostate or seminal vesicles. However, in a patient whose symptoms clearly and repeatedly worsen after frequent ejaculations — particularly when paired with **deep pain in the seminal vesicle/prostate region, reduced semen volume, weak ejaculation, and onset after multiple ejaculations in one day** —

it is clinically reasonable to infer that the **ejaculatory system may not be regaining baseline tolerance between episodes**. That is a **phenotype-specific recovery problem**, not a universal biological rule.

If, in addition, later parts of the survey suggest benefit from **reducing ejaculation frequency**, that further strengthens this interpretation. If short steroid responsiveness is also reported, it may raise suspicion of an inflammatory or post-inflammatory amplification component in some patients, while benefit from antihistamines may suggest a neuroimmune or histamine-linked component in others. But those treatment-response clues must always be layered on top of this timing pattern rather than replacing it.

After prolonged sitting

This timing pattern strongly supports a **mechanical-compressive or sitting-dependent phenotype**. If symptoms most often occur after prolonged sitting, then the clinical reasoning should prioritize **perineal load, pelvic floor shortening/guarding, pudendal-region compression, venous pooling, and static lumbopelvic strain**. In many patients, this is one of the strongest single clues in the whole questionnaire.

Its significance becomes even greater if the patient later reports **perineal pain, anal pain, pudendal territory pain, symptom worsening with driving or cycling, leaning forward intolerance, tingling/numbness in the thighs or buttocks when sitting, or relief with offloading cushions**. In such cases, "after prolonged sitting" is a key signature of a **mechanical–neuropathic / mechanical–myofascial** pain system. This is precisely why the survey separates this timing pattern from ejaculation-related ones. It allows the evaluator to ask whether the pelvis is more destabilized by **compression** or by **sexual load**, or by both.

After exposure to cold

This answer supports a **cold-reactive muscular, autonomic, vascular, or mast-cell-reactive phenotype**. Cold exposure can increase pelvic floor tone, reduce local tissue comfort, alter vascular behaviour, and heighten sensory defensiveness. In some patients, it may also interact with broader immune reactivity.

This timing pattern becomes more convincing when later sections show **cold exposure as a trigger, relief from heat therapy, warm baths, sauna, pelvic floor spasm, chest-dominant breathing, stress reactivity, or skin reaction to cold / suspected histamine intolerance**. In such cases, cold is not just an unpleasant environmental factor; it may be a consistent physiological trigger for a system that responds with **guarding, vasoconstriction, sensory amplification, or immune irritability**.

Other – please write

The free-text option is important because some of the most informative timing patterns are highly individual. Patients may report that symptoms most often occur **after bowel movements, after bladder filling, at a particular time of day, after long travel, after stress-comedown, after certain foods, or only in a certain posture**. In difficult cases, these individualized timing clues can sometimes explain the syndrome more effectively than the standard checkbox items.

Summary of Point 6

Point 6 is the questionnaire's **temporal trigger-pattern section**. Its purpose is to identify **when the already-described symptoms most commonly emerge**, and thereby clarify which physiological

challenges most destabilize the pelvic system. Symptoms **at the moment of ejaculation** support an immediate ejaculatory/pelvic-floor contraction phenotype. Symptoms **a few hours after ejaculation** suggest delayed neuro-muscular or irritative post-activation reactivity. Symptoms **a few days after ejaculation** make inflammatory, neuroimmune, or deep tissue amplification more plausible. Symptoms **independent of ejaculation** support a broader chronic pain phenotype not dominated by sexual triggers. Symptoms **after long abstinence** support a congestion-sensitive pattern. Symptoms **after frequent ejaculations** support a sexual-overload pattern in which the prostate/seminal-vesicle/pelvic-floor system may not be recovering comfortably between episodes. Symptoms **after prolonged sitting** strongly support a mechanical-compressive or pudendal/myofascial phenotype. Symptoms **after exposure to cold** support a muscular, autonomic, vascular, or histamine-reactive pattern. In professional interpretation, Point 6 is invaluable because it shows **not only that pain exists, but under what physiological condition the system most predictably loses stability**.

Point 7. Nantes Criteria (Pudendal Neuralgia). Essential criteria

(select all that apply)

In the current version of the questionnaire, Point 7 includes the following options: **Pain independent of ejaculation; Pain in the territory of the pudendal nerve (perineum, genitals, anus); Pain worsens when sitting; Pain present upon waking; Pain that worsens throughout the day until evening; Pain in the evening; Pain only during the day; Continuous constant chronic pain, present throughout the day; Pain also at night.**

Purpose of Point 7

Point 7 is designed to identify whether the patient's symptom pattern is compatible with a **pudendal-territory or neuropathic pelvic pain phenotype**. Its function is not to establish a definitive diagnosis of pudendal neuralgia on its own. Rather, it acts as a **pattern-recognition section**. It asks whether the pain behaves in a way that is typical of pain involving the pudendal territory, sitting-related mechanical loading, day-long cumulative worsening, and a chronic neural or pressure-sensitive pattern. In modern chronic pelvic pain medicine, this kind of symptom clustering is highly important because pelvic pain that appears "prostate-like" may in fact be predominantly driven by **pelvic floor myalgia, pudendal irritation, myofascial compression, or chronic pain amplification**, rather than by primary gland pathology alone.

This section is also important because it distinguishes between pain that is primarily linked to **sexual events** and pain that has become more **position-dependent, territory-specific, and chronically self-sustaining**. This distinction matters clinically. A patient whose pain is clearly driven by ejaculation may fit a somewhat different phenotype from one whose pain is focused in the pudendal territory, worsened by sitting, and present independently of ejaculation. The latter pattern more strongly supports a **mechanical–neuropathic** or **pelvic floor–neuropathic** interpretation.

A second reason this point is so valuable is that it adds a **time-of-day dimension** to the neural pain hypothesis. Neural and pressure-sensitive pelvic pain often does not behave like acute injury pain. It may be mild in the morning, accumulate across the day, worsen with sitting or cumulative muscular guarding, and later become evening-dominant or continuous. For that reason, the temporal options in this section are not secondary details. They help determine whether the pain looks **activity-linked, cumulative, pressure-sensitive, continuous, or generalized across the full day-night cycle**.

Pain independent of ejaculation

This option is clinically important because it separates the pain from a primarily ejaculatory or seminal-tract trigger. If pain is independent of ejaculation, then ejaculation may still worsen symptoms at times, but it is **not necessary for the pain to exist**. This shifts interpretation away from a strictly sexual or post-ejaculatory phenotype and toward a more **autonomous pelvic pain pattern**, in which the pain is generated or maintained by mechanisms such as pudendal irritation, pelvic floor tension, bladder hypersensitivity, bowel cross-sensitization, central amplification, or chronic mechanical load.

This answer becomes especially meaningful if it later coexists with **perineal pain, pain in the anus or genitals, sitting-related worsening, electric-shock sensations, tingling, allodynia, or a broad pain distribution that no longer depends on one specific event**. In that combination, "independent of ejaculation" strongly supports the idea that the patient has moved beyond a purely sexual-triggered syndrome into a more persistent **pelvic neural or pelvic floor pain phenotype**. It also helps prevent over-attribution of all symptoms to sexual behavior alone, which is a common interpretive error in chronic pelvic pain.

It is also useful as a contrast variable. In patients with high sexual drive, frequent ejaculation, post-ejaculatory pain, deep seminal vesicle pain, or congestion-sensitive symptoms, pain independence from ejaculation suggests that **sexual load may be a modifier, but not the main engine** of the condition. That distinction is very important when trying to avoid overly narrow interpretations such as "the problem is only ejaculation frequency."

Pain in the territory of the pudendal nerve (perineum, genitals, anus)

This is one of the most diagnostically informative options in the entire questionnaire. The pudendal territory includes the perineum, genital area, and anus, which are among the most symptomatically rich regions in male chronic pelvic pain. If the pain falls primarily within this distribution, the evaluator must strongly consider **pudendal neuralgia-like pain, pelvic floor myofascial compression, perineal soft tissue overload, referred pain from deep pelvic floor structures, or neuropathic amplification within the pudendal sensory field**.

This answer is particularly significant because many patients interpret all genital or anal pain as "prostate pain," when in reality the pudendal territory may be the more anatomically accurate framework. If the pain is in the perineum, anus, and genitals together, and especially if it is worsened by sitting or described as burning, prickling, tingling, or electric, the profile becomes much more consistent with a **pelvic neuropathic / perineal pressure-sensitive syndrome** than with isolated gland disease.

At the same time, this answer should not be read too narrowly as "pure nerve damage." Pudendal-territory pain can also arise when the nerve is not structurally injured but is living within a **high-tension, inflamed, compressed, or hypersensitized pelvic environment**. In practical terms, this means the pain may still respond to pelvic floor physiotherapy, relaxation training, nerve blocks, or neuromodulators even if formal objective nerve injury is not demonstrated. That is one of the reasons modern guidelines treat pelvic pain phenotyping as more useful than rigid organ labeling.

Pain worsens when sitting

This is one of the strongest possible clues to a **mechanical–compressive pelvic pain phenotype**. Sitting places sustained pressure on the perineum, pelvic floor, pudendal territory, and surrounding

soft tissues, especially in men who already have chronic guarding, pelvic floor shortening, or deep tissue irritability. When a patient clearly reports that pain worsens when sitting, the evaluator should strongly consider **pudendal region loading, pelvic floor overactivity, venous congestion, or pressure-sensitive neural tissue** as major contributors.

This answer becomes especially powerful when combined with **driving pain, cycling intolerance, perineal pain, anal pain, genital pain, leaning-forward worsening, tingling or numbness in the thighs or buttocks when sitting, or improvement with offloading cushions**. In that context, the pain is behaving like a pressure-sensitive pelvic syndrome rather than a random or purely inflammatory complaint. In both AUA and EAU thinking, pelvic floor dysfunction and myofascial contributions are common in male CPPS, and pain with sitting is one of the clearest real-life markers that the pelvic pain phenotype may have a **mechanical and myofascial-neuropathic core**.

This option is also useful because it helps distinguish pudendal-type or pelvic floor-type pain from more **purely inflammatory or sexual-triggered** pain. A patient with deep post-ejaculatory pain but no sitting sensitivity may belong to a different phenotype from one who has comparable pain but is dramatically worse on any chair. That distinction shapes treatment thinking.

Pain present upon waking

Pain upon waking suggests that the patient does not begin the day from a fully pain-free baseline. This is clinically important because it implies that the condition is **already active before the day's physical exposures accumulate**. Such a pattern may indicate chronic overnight pelvic floor tension, poor restorative sleep, unresolved sensitization, persistent bladder or bowel irritation, or a pain state that has become partly independent of daytime activity.

This answer does not necessarily mean that the pain is at its worst in the morning. Rather, it means the pain is already present when the patient wakes, which often signals a more chronic and less fully resettable pain system. If this answer occurs together with **difficulty sleeping, pain also at night, stress-related sleep disruption, or continuous all-day pain**, it supports the interpretation of a syndrome that is no longer simply "triggered during the day," but has become more globally entrenched.

Pain that worsens throughout the day until evening

This option is highly characteristic of a **cumulative load phenotype**. It suggests that the patient's symptoms build as the day progresses, likely because of repeated or sustained exposure to the factors that destabilize the pelvis: sitting, movement strain, stress, sexual activation, poor posture, bowel loading, or cumulative pelvic floor guarding. This is a classic temporal pattern for **mechanical–myofascial–autonomic worsening**.

Clinically, this answer is especially useful because it implies that the pain is being maintained not only by static pathology but also by the **day's functional demands**. If it coexists with sitting-related worsening, work stress, prolonged sitting at home, pelvic heaviness, and evening pain, then the case for cumulative mechanical, muscular, or vascular burden becomes very strong. If it coexists with pain that is also present upon waking, then the interpretation shifts toward a syndrome that is already active in the morning and **intensified by daily accumulation**.

Pain in the evening

Evening-dominant pain usually reflects the endpoint of a cumulative daily process. While "pain worsens throughout the day until evening" emphasizes the trajectory, "pain in the evening" emphasizes the **time of maximum symptom expression**. Clinically, evening pain often supports

the same broad mechanisms: cumulative sitting, pelvic floor overuse, vascular heaviness, unresolved stress loading, or prolonged stimulation of a sensitized pelvic system.

This option becomes especially informative when paired with **pelvic heaviness, relief when lying down, prolonged sitting, driving, work stress, or symptom reduction on days of rest**. In such cases, evening pain may mark the daily culmination of mechanical, vascular, or autonomic stressors. If evening pain is present without strong sitting or activity links, it may also reflect a chronic pattern in which the patient is less able to down-regulate after the day is over.

Pain only during the day

Pain that occurs only during the day suggests that the syndrome is strongly linked to **wake-state exposures** rather than to continuous tissue irritation alone. This often supports an interpretation involving posture, sitting, work stress, movement load, bladder filling, bowel activity, or repeated sexual triggers occurring during waking hours. In other words, the pelvis behaves more like an **activity-dependent pain system** than a continuously self-generating one.

This answer becomes especially relevant if the patient later denies significant night pain and instead reports strong symptom links to **sitting, work, driving, stress, bladder filling, or ejaculation**. Such a pattern may be clinically useful because it suggests a system that still depends heavily on environmental or behavioural activation and may therefore remain more modifiable than a pain state that persists round the clock.

Continuous constant chronic pain, present throughout the day

This answer signals a more severe level of chronicity. It suggests that pain is no longer only a response to specific exposures but has become a **continuous feature of daily experience**. In chronic pelvic pain medicine, this usually increases the likelihood of a **sensitized, entrenched, multi-mechanistic phenotype**, especially if the patient also reports spread of pain, allodynia, persistent symptoms after triggers end, poor sleep, catastrophizing, or multiple system involvement.

Continuous all-day pain does not tell us whether the dominant driver is muscular, neural, inflammatory, bladder-related, or vascular. What it does tell us is that **the system is not returning to baseline during ordinary waking life**. This is clinically important because it often means that even if there is a localized pain generator somewhere in the pelvis, the patient's nervous system is now participating heavily in maintaining symptom persistence.

Pain also at night

Pain present at night is another marker of greater chronicity and reduced physiological reset. It suggests that the pain system remains active even when daytime mechanical exposures are reduced. This may support **sensitization, persistent bladder or bowel signalling, chronic inflammatory or post-inflammatory irritability, autonomic dysregulation, or unresolved pelvic floor overactivity during rest**.

Night pain is particularly important in interpretation because it weakens a purely "only from sitting" explanation. If the patient has both sitting-related worsening and night pain, then the syndrome likely includes both **mechanical triggers and persistent carry-over activation**. If night pain coexists with bladder symptoms, nocturia, pelvic pressure with bladder filling, or histamine-related food triggers, then a **bladder-sensory or immune-irritative component** becomes more plausible. If it coexists with severe stress, poor sleep, and continuous all-day pain, then the broader chronic pain system is likely deeply involved.

Summary of Point 7

Point 7 is the questionnaire's **pudendal-territory and chronicity-pattern section**. Its purpose is to determine whether the patient's pain behaves like a **pudendal / pelvic floor / sitting-dependent pelvic pain syndrome**, and whether it follows a daily pattern of accumulation, continuity, or night-time persistence. "Pain in the territory of the pudendal nerve" and "pain worsens when sitting" are among the strongest indicators of a **mechanical–neuropathic or pelvic floor–neuropathic phenotype**. "Pain independent of ejaculation" helps distinguish a more autonomous pelvic pain state from a primarily ejaculatory one. "Pain present upon waking," "pain that worsens throughout the day until evening," "pain in the evening," "pain only during the day," "continuous constant chronic pain," and "pain also at night" together define how deeply the pain has entered the patient's daily and nightly physiology. In professional interpretation, Point 7 is invaluable because it moves the questionnaire from symptom listing into **pattern-based pelvic pain phenotyping**.

Point 8. Character of Pain – my pain feels like:

(select all that apply)

In the current version of the questionnaire, Point 8 includes the following options: **Burning; Dull ache; Pressure or fullness; Throbbing; Electric-shock sensation; Cyclical flare-ups occur; Tingling; Prickling; Additional tingling in the thighs or buttocks when sitting; Additional numbness in the thighs or buttocks when sitting; Additional numbness in the legs when sitting; Additional tingling in the legs when sitting.**

Purpose of Point 8

Point 8 is designed to identify the **qualitative language of the pain**, which is one of the best clinical clues to mechanism. In chronic pelvic pain, the way a patient describes pain is not just subjective wording. It often signals whether the dominant process is more likely **neuropathic, myofascial, pressure-based, vascular, inflammatory, or mixed**. For this reason, pain quality is a fundamental part of phenotype interpretation in modern chronic pain medicine.

This section is also crucial because pain quality frequently reveals that two patients with apparently similar "prostate pain" are actually very different clinically. One may describe **burning, tingling, electric sensations, and sitting-induced thigh symptoms**, suggesting a neuropathic-pudendal phenotype. Another may describe **pressure, fullness, heaviness, and evening worsening**, suggesting congestion, deep tissue pressure, or pelvic floor overload. Another may describe **cyclical flares**, suggesting trigger-dependent instability rather than constant background pain. Thus, Point 8 gives the evaluator access not just to intensity, but to the **biological style of pain signalling**.

This point also helps contextualize later treatment response. For example, pain described as burning and electric may later correlate with benefit from **pregabalin, gabapentin, amitriptyline, lidocaine, ketamine, or nerve blocks**, which would strengthen a neuropathic interpretation. Pressure/fullness may later correlate more with **alpha-blockers, tadalafil, offloading, or vascular unloading**. If cyclical flare-ups later correlate with **short steroid benefit or antihistamine benefit**, that may suggest episodic inflammatory or neuroimmune amplification. Thus, although Point 8 is a symptom-description section, it often foreshadows the logic of later treatment interpretation.

Burning

Burning pain is one of the classic descriptors of **neuropathic or sensory-amplified pain**. In the pelvis, burning often points toward **nerve irritability, pelvic sensory hypersensitivity, urethral**

sensory overactivation, pudendal territory pain, or centrally amplified pain processing. It is especially important if later sections also show **tingling, electric-shock sensations, allodynia, penile or perineal hypersensitivity, light-touch pain, or pain persisting after the trigger ends**.

However, burning in pelvic pain is not automatically "pure neuropathy." It may also appear in patients with **bladder irritation, urethral irritation, histamine-related sensory activation, or post-inflammatory tissue irritability**. If burning coexists with **bladder pain, urgency, burning during urination, pain after urination, worsening after acidic foods, or later benefit from antihistamines**, then an **immune-irritative or bladder-sensory phenotype** becomes more plausible. In that sense, burning is a high-value descriptor because it often signals that the pain is being felt through a **sensitized sensory interface**, even if multiple upstream causes exist.

Dull ache

A dull ache usually supports a more **deep-tissue, muscular, pressure-based, or organ-adjacent** interpretation. It often corresponds to pain patients describe as soreness, persistent discomfort, or a non-electric deep pain rather than sharp or burning irritation. In male pelvic pain, a dull ache may arise from **deep pelvic floor tension, peri-prostatic discomfort, seminal tract loading, lower abdominal wall tension, or low-grade pressure in pelvic tissues**.

This descriptor becomes especially meaningful when paired with **deep pain behind the testicles/prostate region, seminal vesicle-region pain, pelvic heaviness, evening worsening, or pain that builds through the day**. In such cases, a dull ache often supports a **myofascial, pressure, or post-inflammatory deep pelvic phenotype** more than a purely neuropathic one. It can also occur in men whose sexual or sitting-related symptoms feel more like pressure accumulation than nerve firing.

Pressure or fullness

Pressure or fullness is one of the most clinically informative descriptors because it often points toward **congestion, pelvic floor overactivity, organ pressure sensitivity, venous pooling, deep fascial tension, or unresolved post-ejaculatory/deep pelvic loading**. In contrast to burning or electric pain, this descriptor usually makes the evaluator think first of **deep-volume or pressure phenomena** rather than pure nerve signalling.

This option becomes especially important if the patient also reports **pelvic heaviness, pain behind the testicles/prostate region, seminal vesicle pain, worsening after abstinence, bladder pressure when full, rectal fullness, or symptom relief when lying down**. In that context, "pressure or fullness" may indicate a **congestion-sensitive phenotype**, a **deep pelvic floor pressure phenotype**, or a **bladder/rectal pressure-sensory phenotype**. If such pain later responds to tadalafil, alpha-blockers, offloading, reduced sitting, or carefully selected short steroid courses, the interpretation may lean toward a mixed **deep-tissue + inflammatory or vascular + neural** phenotype.

Throbbing

Throbbing pain may suggest a **vascular, pulsatile, or pressure-fluctuating component**, although it is less specific than burning or electric-shock sensations. In a pelvic pain context, throbbing often becomes clinically relevant when paired with **pelvic heaviness, evening worsening, visible varicosities, heat sensitivity, post-activity pelvic pressure, or fullness relieved by lying down**. In that setting, a **vascular or congestion-related interpretation** becomes more plausible.

At the same time, throbbing may also appear in irritated tissues that are inflamed or pressure-loaded. If it coexists with a positive deep inflammatory history, abnormal semen inflammatory markers, or short-course steroid benefit, then a **deep inflammatory–pressure phenotype** may also be considered. It is therefore a useful descriptor, though less specific than some other pain-quality options.

Electric-shock sensation

This is one of the strongest single indicators of a **neuropathic pelvic pain phenotype**. Electric-shock sensation strongly suggests that pain is being experienced through an irritated or sensitized neural pathway rather than only as deep aching or pressure. In male pelvic pain, it should immediately raise suspicion of **pudendal irritation, pelvic nerve hypersensitivity, sacral pathway involvement, or high sensory gain in the pelvic pain network**.

Its significance increases greatly if the patient also reports **tingling, numbness, allodynia, pain radiating along nerves, sitting-induced thigh or buttock symptoms, pain in the pudendal territory, or positive response later to neuromodulating medications or nerve-targeted interventions**. In that combination, the pain profile is strongly compatible with a **neuropathic-pudendal or neural sensitization phenotype**.

Cyclical flare-ups occur

Cyclical flare-ups suggest that the pain is not entirely constant but instead behaves in a **trigger-responsive, oscillating manner**. This is highly important clinically because it means the condition may still be heavily influenced by **load-response patterns**, such as stress, sitting, ejaculation, abstinence, food triggers, bowel function, sleep disruption, or temperature exposure.

This answer becomes especially meaningful when the patient later identifies strong triggers in the ranking section or reports that symptoms worsen after **alcohol, histamine-rich foods, stress, prolonged sitting, frequent ejaculation, or poor sleep**. In some patients, cyclical flare-ups may support a **neuroimmune or inflammatory amplification phenotype**, especially if brief steroid responsiveness or antihistamine responsiveness is later reported. In others, it may reflect a more mechanical or behavioural trigger pattern. The key point is that cyclical flares suggest the system still has **identifiable instability points**, which can be clinically useful.

Tingling

Tingling is a classic sensory descriptor that supports a **neural or sensory-hypersensitivity phenotype**. It is not as dramatic as an electric-shock sensation, but it often indicates that the pain system is involving altered sensory transmission rather than only deep pressure or muscular aching. In pelvic pain, tingling may reflect **pudendal irritation, peripheral sensory hyperexcitability, or referred neural symptoms from the pelvis or lower spine**.

Its meaning becomes stronger if later sections show **tingling in the pelvis, radiating pain, perineal or penile hypersensitivity, allodynia, or sitting-related numbness/tingling in the thighs or buttocks**. In such cases, tingling suggests that the patient's pain map includes a meaningful **sensory-neural layer**, even if deep tissue pressure or myofascial symptoms are also present.

Prickling

Prickling is another neuropathic-leaning sensory descriptor, but it often implies a more **surface-oriented or sensory-irritative quality** than dull ache or fullness. In a pelvic context, prickling may

point toward **cutaneous-perineal hypersensitivity, urethral sensory irritation, genital sensory over-reactivity, or mild neural irritation**.

If it is accompanied by **burning, glans pain, shaft pain, sensitivity to clothing, or perineal hypersensitivity**, then the profile becomes increasingly consistent with **sensory overactivation** rather than purely deep pelvic pressure. Prickling may therefore help identify patients whose pain is not only deep and internal, but also experienced through an abnormally reactive sensory surface system.

Additional tingling in the thighs or buttocks when sitting

This is an extremely useful option because it links **sensory symptoms to posture**. Tingling in the thighs or buttocks specifically when sitting strongly supports a **compression- or position-dependent neural phenomenon**. This may reflect pudendal-region loading, posterior pelvic pressure transmission, sacral or gluteal neural sensitivity, or lumbopelvic mechanical contribution.

Clinically, this option becomes especially compelling when paired with **pain worsened by sitting, driving, cycling, sacral or low-back pain, or suspected sacral/pudendal involvement**. In such a case, the patient's pain system appears not only pelvic but also **positionally neurodynamic**, meaning that seating posture is altering symptom expression through a neural pathway.

Additional numbness in the thighs or buttocks when sitting

Numbness carries a stronger implication of altered sensory transmission than tingling alone. When it occurs in the thighs or buttocks specifically during sitting, it strongly supports a **compression-sensitive neural or lumbopelvic phenotype**. This may involve pudendal-related pressure phenomena, gluteal/sciatic-region irritation, or sacral/lumbar nerve contribution.

This answer is especially valuable because it suggests that sitting does not merely worsen pain through discomfort; it may be changing **nerve conduction or sensory perception** in a reproducible way. If later sections reveal lumbar or sacral pain, spinal disease history, or additional leg symptoms, then the evaluator should more strongly consider a **pelvis–spine interaction phenotype** rather than a purely local pelvic condition.

Additional numbness in the legs when sitting

This option broadens the symptom field further and often points toward a **lumbosacral or broader positional neural contribution** rather than a pain syndrome confined strictly to the perineum. If the legs become numb during sitting, the evaluator should think beyond the prostate and ask whether the patient's sitting posture is provoking a wider **spinal–pelvic–neural response**.

This answer is particularly important if later sections include **low-back pain, sacral pain, spinal injury or disease, radiating neural pain, or sitting-related worsening of pelvic symptoms**. In that context, the pain phenotype may involve a meaningful contribution from **lumbosacral neural structures**, even if the patient's main complaint remains pelvic.

Additional tingling in the legs when sitting

Tingling in the legs during sitting again supports a **position-sensitive neural phenotype**, though often with a slightly milder sensory emphasis than numbness. In practice, it suggests that sitting-related loading is not remaining local to the pelvis but is affecting a wider sensory field. This is

highly relevant because it indicates that the pelvic pain system may be part of a **broader seated neurodynamic problem** involving the spine, sacral roots, gluteal structures, or neural tension patterns.

If this answer is present together with **electric-shock sensation, low-back or sacral pain, pudendal-territory pain, or additional thigh/buttock symptoms**, then the case for a **mechanical –neuropathic–spinal overlap phenotype** becomes significantly stronger.

Summary of Point 8

Point 8 is the questionnaire's **pain-quality and sensory-phenotype section**. Its purpose is to determine **how the pain is experienced**, which often gives direct clues to mechanism. **Burning, tingling, prickling, and electric-shock sensation** strongly support a **neuropathic or sensory-amplified phenotype**, especially when paired with allodynia, perineal/penile hypersensitivity, and sitting-related spread. **Dull ache** suggests deeper tissue, muscular, or post-inflammatory discomfort. **Pressure or fullness** points more toward **congestion, pelvic floor pressure, bladder/rectal pressure sensitivity, or deep pelvic loading**. **Throbbing** may support vascular or inflammatory pressure-related interpretations. **Cyclical flare-ups** suggest a trigger-responsive unstable system rather than a strictly constant one. The sitting-dependent thigh, buttock, and leg tingling/numbness items are particularly important because they show that the pain phenotype may extend into a **mechanical–neuropathic–lumbosacral network** rather than remaining limited to the pelvis itself. In professional use, Point 8 is one of the most powerful tools in the survey for separating **deep pressure pain, muscular pain, sensory pain, vascular discomfort, and mixed pain phenotypes**

Point 9. Urinary Symptoms. Do you experience:

(select all that apply)

In the current version of the questionnaire, Point 9 includes the following options: **frequent urination during the day; frequent urination at night (nocturia); sudden urge to urinate; weak urinary stream; intermittent urinary stream; post-void dribbling; difficulty initiating urination; sensation of incomplete bladder emptying; pressure in the urinary bladder; burning during urination; pain in the urinary bladder; relief after urination; worsening after urination; episodes of urinary retention; pain after urination; bladder hypersensitivity; pain when the bladder fills; sudden urgency attacks; nocturnal urgency; pelvic pressure when the bladder is full; diagnosis of painful bladder syndrome / interstitial cystitis**. The questionnaire also separately asks: **How many times do you urinate at night? Mark the correct number: 0–10.**

Purpose of Point 9

Point 9 is designed to determine **how strongly the lower urinary tract is involved in the overall pain phenotype** and whether the urinary tract behaves more like a system that is inflamed, hypersensitive, obstructed, poorly coordinated, overactive, or cross-sensitized with the pelvic floor and pain system. This is an essential distinction in chronic pelvic pain. Men with CPPS commonly describe urinary symptoms, but modern guidance makes clear that such symptoms do **not automatically imply persistent infection or simple prostate obstruction**. They may reflect bladder pain syndrome–like features, pelvic floor dysfunction, outlet discoordination, sensory gain, chronic guarding, or central pain amplification.

AUA guidance specifically highlights that men with chronic pelvic pain should be assessed for **urinary symptoms, pain location, associated pelvic floor myalgia, and overlapping bladder pain phenotypes**, and EAU guidance similarly stresses the importance of a broad chronic pelvic pain framework rather than reduction to one organ label. In practical terms, Point 9 asks whether the bladder and urethra are merely passive neighbours of pelvic pain, or whether they are active participants in the syndrome.

This section is also important because urinary symptoms in chronic pelvic pain may have different meanings depending on **timing, pain quality, and treatment response**. For example, bladder pressure that improves after urination means something different from urethral burning that worsens after urination, and both differ from weak stream plus hesitancy, which may reflect outlet or pelvic floor discoordination. Likewise, if a patient later reports benefit from **alpha-blockers**, the urinary phenotype may include smooth-muscle/outlet involvement; if he reports benefit from **antihistamines such as hydroxyzine or H1/H2 blockers**, that may support a **bladder hypersensitivity / mast-cell / histamine-amplified phenotype**, especially when bladder pain and food-related irritability coexist. Antihistamines are already part of the current AUA/EAU-aligned chronic pelvic pain landscape because bladder pain and mast-cell-associated sensory amplification are recognized overlapping mechanisms in some patients.

Frequent urination during the day

Daytime frequency suggests that the bladder or bladder-sensory system is functioning in a **low-threshold state**, meaning that the urge to void occurs earlier and more often than expected. In chronic pelvic pain, this may result from true bladder hypersensitivity, pelvic floor tension around the outlet, autonomic overactivation, conditioned urgency, or a pain-avoidance pattern in which the patient voids more often because filling itself is uncomfortable. Clinically, this answer becomes more meaningful if later items also show **pain when the bladder fills, sudden urgency attacks, bladder pressure, or pelvic pressure when the bladder is full**, because that combination supports a **bladder-sensory phenotype** rather than frequency alone. AUA guidance emphasizes that urinary symptoms in men with chronic pelvic pain must be evaluated as part of the pain phenotype and not treated as evidence of a single simple cause.

Frequent urination at night (nocturia)

Nocturia is particularly important because it reflects not only the urinary tract, but also the interaction between **bladder behaviour, sleep physiology, autonomic tone, and chronic pain arousal**. In some men, nocturia suggests a more persistent bladder storage problem; in others, it reflects poor restorative sleep, hypervigilance, pelvic discomfort during filling, or incomplete nighttime down-regulation of the nervous system. It becomes more clinically relevant when paired with **night pain, nocturnal urgency, sleep problems, anxiety, or bladder pain on filling**. In that context, nocturia may be part of a broader **bladder–autonomic–sleep phenotype**, rather than just a symptom of aging or prostate enlargement. Modern chronic pelvic pain guidance encourages clinicians to interpret overlapping urinary and pain symptoms in exactly this broader way.

Sudden urge to urinate

A sudden urge to urinate suggests that the bladder sensory system may be **reactive, unstable, or over-interpreting filling signals**. In chronic pelvic pain, urgency does not always mean “overactive bladder” in the narrow sense. It may reflect pain-triggered voiding, pelvic floor guarding, bladder wall hypersensitivity, or central urgency amplification. This answer becomes especially informative when it occurs with **pain when the bladder fills, bladder pressure, relief after urination, or**

urgency attacks, because that cluster suggests a low-threshold bladder pain phenotype rather than isolated urinary frequency.

Weak urinary stream

Weak stream raises the possibility of **outlet resistance, smooth-muscle tension, bladder neck involvement, pelvic floor overactivity, or true mechanical obstruction**. In younger men with chronic pelvic pain, weak stream often points more toward **functional outlet dysfunction or pelvic floor discoordination** than to classic age-related enlargement. In older men, the interpretation may be more mixed. This option becomes especially important when combined with **difficulty initiating urination, intermittent stream, incomplete emptying, or retention episodes**. In those combinations, the urinary phenotype may include **outlet dysfunction**, and later benefit from **alpha-blockers** would make that interpretation stronger. AUA and EAU approaches both support careful symptom phenotyping rather than assuming that all chronic pelvic pain urinary symptoms are infectious.

Intermittent urinary stream

An intermittent stream suggests that urinary flow is being interrupted, either because of variable outlet resistance, pelvic floor guarding, inconsistent detrusor–sphincter coordination, or functional non-relaxation during voiding. In chronic pelvic pain, this often fits a **pelvic floor dysfunction phenotype**, especially if later sections show **difficulty relaxing the pelvic floor, perineal tension, rectal fullness, or pelvic muscle spasm**. If intermittent stream occurs together with weak stream and hesitancy, the case for a **functional voiding discoordination pattern** becomes stronger.

Post-void dribbling

Post-void dribbling often suggests incomplete final emptying of the urethra or poor pelvic floor / bulbospongiosus coordination. In chronic pelvic pain it may be another clue that the lower urinary tract is functioning in a **mechanically or coordinatively inefficient way**, rather than simply being inflamed. This item becomes more meaningful if paired with **weak stream, hesitancy, incomplete emptying, pelvic floor symptoms, or pain after urination**. It tends to support a more **functional pelvic floor–outlet phenotype** than an isolated bladder pain phenotype.

Difficulty initiating urination

Difficulty initiating urination is highly relevant because it often reflects **failure of coordinated relaxation** at the outlet. In the chronic pelvic pain setting, this may result from pelvic floor overactivity, guarding around the bladder neck, anxiety-related voiding inhibition, or true outlet dysfunction. The interpretation becomes especially strong if the patient also reports **weak stream, intermittent stream, incomplete emptying, or pelvic floor clenching**, because that cluster is highly compatible with a **pelvic floor / outlet discoordination phenotype**.

Sensation of incomplete bladder emptying

The sensation of incomplete emptying is particularly important because it may represent either a **true residual urine problem** or a **sensory mismatch**, where the patient feels incompletely emptied despite little objective residual. In chronic pelvic pain, that distinction matters greatly. A mismatch can arise from bladder hypersensitivity, pelvic floor guarding, persistent outlet tension, or chronic pain-related bodily vigilance. This symptom is therefore especially important when later interpreted together with **post-void residual testing, uroflowmetry, and pelvic floor findings**. It is one of the

clearest examples of why chronic pelvic pain must be evaluated with both symptoms and function in mind.

Pressure in the urinary bladder

Bladder pressure suggests that the bladder is being experienced not just as a reservoir but as an area of **mechanical, sensory, or inflammatory discomfort**. This may represent early bladder filling discomfort, low-threshold bladder sensation, pelvic floor pressure transmission, or a more explicit bladder pain phenotype. This answer becomes especially meaningful if it later coexists with **pain in the urinary bladder, pain when the bladder fills, pelvic pressure when the bladder is full, relief after urination, urgency, or diagnosis of painful bladder syndrome / interstitial cystitis**. In that context, the bladder is likely a major active participant in the pain syndrome.

Burning during urination

Burning during urination is one of the most easily misread symptoms in chronic pelvic pain. Patients and sometimes clinicians may assume it means infection, but when cultures are repeatedly negative, burning often points instead to **urethral hypersensitivity, bladder–urethral sensory amplification, residual post-inflammatory irritability, or histamine-associated irritative pain**. This item becomes especially important when paired with **glans pain, burning after ejaculation, acidic-food reactivity, histamine-related symptoms, or benefit from antihistamines**. In such cases, the burning may be part of a **bladder/urethral sensory phenotype** rather than persistent microbial disease.

Pain in the urinary bladder

This option is stronger than “pressure” or “burning” because it implies that the patient experiences the bladder itself as a pain site. This strongly supports a **bladder pain phenotype**, especially if later items confirm **pain on filling, urgency, nocturia, and relief after voiding**. In contemporary pelvic pain frameworks, bladder pain in men is increasingly recognized as a valid and important overlap condition rather than an uncommon curiosity. AUA guidance specifically incorporates male chronic pelvic pain overlap with bladder pain conditions and pelvic floor myalgia.

Relief after urination

Relief after urination is highly informative because it suggests that bladder filling itself may be a symptom driver. If voiding relieves pain, pressure, or urgency, then the bladder is likely functioning as a **triggering reservoir** whose filling increases discomfort until emptied. This pattern is strongly compatible with **bladder hypersensitivity or bladder pain syndrome–like physiology**, especially if paired with **pain when the bladder fills, urgency, nocturnal urgency, or bladder-region pain**.

Worsening after urination

Worsening after urination suggests a different mechanism from relief after urination. It implies that the act of voiding itself — or the immediate post-void state — triggers irritation, sensory rebound, pelvic floor spasm, or urethral discomfort. This answer becomes particularly meaningful if later sections show **burning after urination, urethral burning after ejaculation, pelvic floor tension, or post-void pain**. In such patients, voiding may be less a relief mechanism and more a **provoking event** for a sensitized outlet or urethral–pelvic floor interface.

Episodes of urinary retention

Retention episodes are clinically important because they raise the possibility of more severe **outlet dysfunction, pelvic floor non-relaxation, medication-related voiding impairment, or marked autonomic/smooth-muscle dysregulation**. In chronic pelvic pain, retention episodes do not automatically prove mechanical obstruction, but they do signal that urinary function needs serious attention. This option becomes especially important if later sections also reveal **decongestants or anticholinergic drugs worsening symptoms**, because those medication effects can support a vulnerability of the outlet/smooth-muscle system.

Pain after urination

Pain after urination suggests that the post-void state itself is painful, which may reflect **urethral irritation, outlet spasm, bladder neck sensitivity, or a sensory rebound after detrusor–outlet activity**. It is especially meaningful if combined with **worsening after urination, burning during urination, pelvic floor symptoms, or urethral burning after ejaculation**, because those patterns support a sensitized lower tract / pelvic floor interface.

Bladder hypersensitivity

This is one of the most directly interpretive options in the urinary section. It means that the patient experiences the bladder as **too reactive, too noticeable, or too easily symptomatic**, even when routine tests may be normal. In chronic pelvic pain, bladder hypersensitivity may be primary, secondary to chronic pelvic floor tension, related to prior inflammation, or amplified by immune and histamine pathways. If later sections show **pain on filling, urgency, relief after voiding, suprapubic pain, histamine-food triggers, allergic features, or benefit from antihistamines**, then a **bladder-sensory / neuroimmune phenotype** becomes especially plausible.

Pain when the bladder fills

Pain on filling is one of the strongest indicators of a **bladder pain phenotype**. It suggests that bladder distension itself is painful, which distinguishes this symptom from generic frequency or urgency. Clinically, this option becomes even more significant when paired with **relief after urination, urgency, suprapubic pain, bladder pressure, nocturia, or diagnosis of painful bladder syndrome / interstitial cystitis**. In that context, the bladder is not only associated with the pain — it is behaving like one of the core pain-generating organs in the phenotype.

Sudden urgency attacks

This answer points toward an episodic pattern of **rapid, forceful urgency**, which may suggest instability in bladder sensory processing, autonomic dysregulation, or bladder pain overlap. If paired with **daytime frequency, bladder pain, pressure on filling, or nocturnal urgency**, it strengthens a bladder-centred interpretation. If paired instead with **severe anxiety, body vigilance, and stress-triggered worsening**, autonomic amplification may be playing a larger role.

Nocturnal urgency

Nocturnal urgency differs from nocturia in that it emphasizes the **quality of the urge** rather than simply the number of voids. It may reflect a bladder that remains **sensory-reactive during the night**, poor autonomic down-regulation, or a pain syndrome in which bladder filling cannot be comfortably tolerated during sleep. This is especially important if combined with **night pain, bladder filling pain, poor sleep, and anxiety-related hyperarousal**.

Pelvic pressure when the bladder is full

This answer is particularly useful because it connects bladder filling not just to local bladder pain but to **broader pelvic pressure sensation**. This suggests that bladder distension may be transmitted through a **pelvic floor, fascial, deep pelvic, or vascularly sensitive environment**, rather than being experienced as bladder discomfort alone. It becomes especially important when paired with **pelvic heaviness, rectal fullness, deep perineal pressure, or pain behind the testicles/prostate region**, because it suggests that bladder filling is interacting with the whole pelvic container, not just the bladder wall.

Diagnosis of painful bladder syndrome / interstitial cystitis

This option indicates that bladder-centred pain has already been recognized clinically. In the setting of this survey, that does not exclude CPPS — it often identifies an **overlap phenotype** in which male chronic pelvic pain and bladder pain syndrome coexist. AUA guidance explicitly treats overlap between pelvic floor myalgia, chronic pelvic pain, and bladder pain conditions as clinically important. This answer gains even more weight if the patient also reports **pain with filling, urgency, frequency, nocturia, suprapubic pain, bladder-region pain, histamine sensitivity, or antihistamine benefit**.

How many times do you urinate at night? (0–10)

This scale quantifies the severity of **nighttime urinary disturbance**. A higher number is not just a frequency measure; it can indicate increased bladder sensitivity, sleep fragmentation, autonomic dysregulation, chronic hypervigilance, or poor nighttime symptom control. Low numbers may be compatible with mild or absent nocturnal involvement, while higher numbers suggest that the urinary/bladder component is substantial and materially affecting recovery. In chronic pelvic pain, this matters because poor sleep itself can further amplify pain sensitivity and autonomic imbalance. Current guidance strongly emphasizes the need to consider associated functional consequences and overlapping symptom domains in chronic pelvic pain.

Summary of Point 9

Point 9 is the questionnaire's **lower urinary tract and bladder phenotype section**. Its purpose is to determine whether the urinary tract is behaving mainly as a system of **frequency/urgency**, **bladder pain/hypersensitivity**, **outlet dysfunction/discoordination**, or a **mixed urinary–pelvic floor–sensory syndrome**. Frequency, urgency, pain on filling, relief after urination, and bladder pain support a **bladder-centred sensory phenotype**. Weak stream, hesitancy, intermittent flow, incomplete emptying, and retention point more toward **outlet or pelvic floor dysfunction**. Burning and pain after urination suggest a **urethral–sensory or post-void irritative phenotype**. High nocturia or nocturnal urgency raise the importance of **sleep disruption, autonomic tone, and persistent bladder involvement**. In professional interpretation, Point 9 is essential because it shows whether urinary symptoms are central to the chronic pelvic pain picture and, if so, **what kind of urinary involvement is most likely active**.

Point 10. Symptoms Related to Ejaculation and Pelvic Innervation

(select all that apply)

In the current version of the questionnaire, Point 10 includes the following options: **pain during ejaculation; pain immediately after ejaculation; pain several hours after ejaculation; pain the day after ejaculation; pain after ejaculation combined with sitting; relief after ejaculation;**

worsening after several days of abstinence; worsening after several weeks of abstinence; worsening with frequent daily ejaculation; pain in the seminal vesicle region; testicular pain after ejaculation; burning in the urethra after ejaculation; reduced semen volume; weak ejaculation; pain during nocturnal emission; decreased libido; increased libido; difficulty relieving sexual tension; penile hypersensitivity causing rapid ejaculation; difficulty achieving orgasm and ejaculation; erectile dysfunction.

Purpose of Point 10

Point 10 is one of the most important sections in the entire questionnaire because it examines how the pelvic pain system responds to **sexual activation, orgasm, ejaculation, post-ejaculatory recovery, abstinence, and sexual arousal regulation**. In male chronic pelvic pain, ejaculation is not just a sexual event. It is a complex physiological challenge involving the **pelvic floor muscles, autonomic nervous system, urethra, prostate region, seminal vesicles, smooth muscle contraction, and deep pelvic sensory pathways**. For that reason, ejaculation can function as a **natural stress test** of the male pelvic system. AUA materials emphasize that **pain with ejaculation** is especially important clinically and often points toward **pelvic floor myalgia/dysfunction**, although it can also coexist with other mechanisms.

This section is also crucial because it allows the evaluator to distinguish among several very different sexual phenotypes:
a predominantly **ejaculatory pain phenotype**,
a **post-ejaculatory delayed flare phenotype**,
an **abstinence/congestion phenotype**,
a **frequent-ejaculation overload phenotype**,
a **libido/regulation dysfunction phenotype**,
and a **broader sexual-function consequence phenotype** involving orgasm, erection, desire, and genital sensitivity.

This is also the section where the issue of **high sexual drive and frequent ejaculations** must be handled carefully and precisely. Current medical knowledge does **not** support a universal claim that frequent ejaculation is harmful for all men or inherently damages the prostate or seminal vesicles. However, in a subgroup of men with chronic pelvic pain, particularly those with **very strong sexual drive, repetitive ejaculations over short intervals, pain in the seminal vesicle/prostate region, reduced semen volume, weak ejaculation, and pain after ejaculation**, it is clinically reasonable to infer that the **deep ejaculatory structures may not be recovering comfortably between high-load events**. That is a **patient-specific tolerance and recovery problem**, not a universal biological rule. AUA guidance supports careful assessment of sexual symptoms, pelvic floor dysfunction, and treatment response rather than simplistic generalizations.

Finally, this section is important because it interacts with the treatment-response part of the survey. In some patients, symptoms in this domain may be reduced by **pelvic floor relaxation strategies, alpha-blockers, tadalafil, neuromodulators, selected antihistamines, or—in some carefully selected cases—brief steroid responsiveness suggesting inflammatory amplification**. The presence of benefit from antihistamines elsewhere in the questionnaire can be especially informative when sexual symptoms coexist with bladder hypersensitivity, histamine-rich food sensitivity, or immune-reactive phenotypes, while short-course steroid benefit may strengthen suspicion that **inflammatory or post-inflammatory amplification** contributes to deep post-ejaculatory pain in some men.

Pain during ejaculation

Pain during ejaculation strongly suggests that the pelvic system is being destabilized at the very moment of **ejaculatory contraction and orgasmic motor output**. This often raises the importance of **pelvic floor myalgia, pelvic floor non-relaxation, deep perineal muscle overactivity, urethral irritation, prostate-region tension, or seminal tract strain**. AUA pelvic pain guidance highlights pain with ejaculation as a clinically meaningful feature, often associated with pelvic floor dysfunction in men with chronic pelvic pain.

This answer becomes particularly important if paired with **pain in the seminal vesicle region, deep pain behind the testicles/prostate region, weak ejaculation, reduced semen volume, difficulty relieving sexual tension, or very strong sexual drive with repeated frequent ejaculation**. In such a pattern, the act of ejaculation itself appears to be overloading a sensitive pelvic system.

Pain immediately after ejaculation

Pain immediately after ejaculation suggests that the sexual event is completed, but the pelvic system enters an immediate **reactive post-ejaculatory state** rather than returning to baseline. Mechanistically, this may reflect **sustained pelvic floor contraction, smooth-muscle after-spasm, urethral irritation, neural flare, or deep tissue irritability**. It is especially meaningful when it coexists with **sitting-sensitive pain, urethral burning, or prostate-region/seminal-vesicle-region pain**, because that cluster suggests a vulnerable deep pelvic system that tolerates sexual activation poorly.

Pain several hours after ejaculation

Pain several hours after ejaculation supports the idea of a **delayed post-activation flare** rather than immediate orgasmic pain. This may indicate that ejaculation initiates a process — muscular, sensory, inflammatory, autonomic, or mixed — whose consequences emerge later. This option becomes especially important when combined with **burning in the urethra after ejaculation, urgency, bladder irritation, deep seminal-region discomfort, or cyclical flares**, because it suggests that the pelvic system is unable to recover smoothly after sexual activation.

Pain the day after ejaculation

Pain the day after ejaculation points toward an even more delayed **post-ejaculatory inflammatory, neuroimmune, deep tissue, or prolonged muscular recovery problem**. This is particularly relevant when symptoms are not limited to the sexual event itself but unfold over time. In a patient who also has **deep prostate/seminal-vesicle pain, reduced semen volume, weak ejaculation, and benefit from anti-inflammatory strategies**, this timing pattern may raise suspicion that there is more than momentary muscular spasm involved. It can fit a phenotype in which **ejaculatory load is followed by prolonged recovery difficulty**, potentially involving deep tissues, local inflammation, or neuroimmune amplification.

Pain after ejaculation combined with sitting

This is one of the most informative combined options in the questionnaire because it joins two major trigger systems: **sexual activation and perineal mechanical loading**. If pain occurs after ejaculation and is especially problematic with sitting, that strongly supports a **deep pelvic floor / pudendal / pressure-sensitive phenotype**, in which the sexual system and the mechanical system amplify one another. This pattern is especially suggestive of patients whose pelvic tissues are already highly vulnerable, so that post-ejaculatory pelvic activation makes them less tolerant of seated compression afterward.

Relief after ejaculation

This answer indicates a different sexual phenotype: instead of ejaculation provoking pain, ejaculation may temporarily relieve symptoms. That often supports a **congestion-sensitive or abstinence-sensitive pattern**, especially if the patient later reports worsening after several days or weeks of abstinence, pelvic heaviness, deep pressure, or seminal vesicle-region discomfort. In such cases, the issue may be **build-up and release** rather than ejaculatory overloading. This should not be overgeneralized, but in this phenotype ejaculation may function as a temporary decompression event.

Worsening after several days of abstinence

This answer suggests that the pelvic system becomes symptomatic when sexual activity is absent for long enough that **tension, fullness, congestion, or sensory anticipation** accumulate. Clinically, this often fits a **congestion-sensitive sexual phenotype**, particularly when paired with **relief after ejaculation, deep prostate-region pain, seminal vesicle-region pain, or pelvic fullness**. It is distinct from the frequent-ejaculation overload pattern and should be interpreted separately.

Worsening after several weeks of abstinence

This is a stronger and longer-term version of the previous pattern. It suggests that prolonged sexual inactivity may be associated with **progressive symptom accumulation**, which may be experienced as pelvic pressure, deep discomfort, or unstable sexual-pelvic regulation. When combined with relief after ejaculation, it strengthens the interpretation of an **abstinence/congestion-sensitive phenotype**, though it still must be interpreted in the full context of the patient's other symptoms and not as a universal rule.

Worsening with frequent daily ejaculation

This is one of the key items for identifying a **sexual-overload phenotype**. It indicates that frequent ejaculation is not neutral for this patient; rather, it appears to exceed the comfortable recovery capacity of the pelvic system. This is especially important if it occurs together with **very strong sexual drive, pain during or after ejaculation, pain in the seminal vesicle region, reduced semen volume, weak ejaculation, and difficulty relieving sexual tension**. In such a cluster, the clinical interpretation is that the patient's deep ejaculatory system may be repeatedly activated **before symptoms have settled from the previous event**. That is the most medically careful way to frame the issue of high libido and frequent ejaculation in chronic pelvic pain.

This does **not** mean that the questionnaire is claiming that ejaculation is universally harmful or that frequent ejaculation inevitably injures the prostate or seminal vesicles. Rather, it identifies a subgroup of patients in whom **sexual load and recovery time appear clinically mismatched**. That is a nuanced and phenotype-based interpretation consistent with current chronic pelvic pain thinking.

Pain in the seminal vesicle region

This is one of the most sophisticated items in the whole sexual section because it specifically points beyond generic "prostate pain" to the **deeper ejaculatory storage/transport system**. Pain in this region becomes highly meaningful when combined with **pain during ejaculation, pain after ejaculation, reduced semen volume, weak ejaculation, abstinence worsening, or frequent-ejaculation worsening**.

This location may support the idea that symptoms are arising from a **deep ejaculatory phenotype**, in which the seminal vesicles and adjacent structures are functionally or inflammatory-reactively involved. If semen testing later shows inflammatory markers, that further strengthens the biological plausibility of this item. If anti-inflammatory strategies — including short steroid response in selected individuals — appear helpful elsewhere in the survey, the seminal-vesicle region becomes even more significant clinically.

Testicular pain after ejaculation

Testicular pain after ejaculation often suggests a **referred post-ejaculatory pain pattern** rather than primary testicular disease, especially when scrotal work-up is unrevealing. In chronic pelvic pain, the testes may become the site where deeper pelvic or ejaculatory pain is perceived. This is especially relevant if it occurs with **groin pain, seminal-vesicle pain, deep prostate-region pain, perineal pain, or pelvic floor tension**, because then the testes may be functioning as part of a broader pelvic referral map.

Burning in the urethra after ejaculation

This symptom strongly supports a **urethral–sensory irritative phenotype** linked to sexual activity. It may indicate post-ejaculatory urethral hypersensitivity, irritative sensory gain, or overlap between sexual and bladder/urethral pain pathways. It becomes especially significant when paired with **burning during urination, glans pain, bladder hypersensitivity, acidic-food worsening, or benefit from antihistamines**, because that broader constellation may support a **bladder/urethral sensory or histamine-amplified subtype** rather than infection.

Reduced semen volume

Reduced semen volume is clinically important because it suggests that the ejaculatory system may not be functioning normally from a **volume, pressure, or deep-secretory standpoint**. It does not, by itself, identify the cause. However, if it coexists with **pain in the seminal vesicle region, weak ejaculation, post-ejaculatory pain, or worsening with frequent ejaculation**, it supports the idea that the deep ejaculatory structures deserve close attention in the phenotype. In that setting, the issue may involve **deep tract irritation, altered contractility, congestion, or reduced tolerance of repeated ejaculatory load**.

Weak ejaculation

Weak ejaculation may indicate a problem in **ejaculatory force generation, pelvic floor output, deep smooth-muscle activity, or sexual-system coordination**. In chronic pelvic pain, it often matters most when it appears alongside **pain during ejaculation, reduced semen volume, deep pelvic pain, and difficulty achieving orgasm and ejaculation**. In such cases, the questionnaire is identifying a **sexual function phenotype that is not limited to pain**, but includes reduced ejaculatory performance and possibly poor recovery of the deep sexual apparatus.

Pain during nocturnal emission

Pain during nocturnal emission is highly significant because it shows that the sexual system can be painful even during an **unconscious, spontaneous ejaculatory event**. This weakens explanations based only on conscious anxiety during sex and suggests that the underlying pelvic apparatus itself is reactive. Clinically, this strengthens the case for a **true sexual-pelvic physiological vulnerability**, whether muscular, inflammatory, sensory, or mixed.

Decreased libido

Decreased libido in the context of chronic pelvic pain syndrome (CPPS) should be interpreted as a secondary, system-level adaptation, rather than an isolated symptom. It often reflects that the **pelvic, neuroendocrine, and sexual systems are already dysregulated** by ongoing pain and repeated activation of **inflammatory and stress pathways**.

One of the central mechanisms involves the **association between sexual activity and pain**. When ejaculation or arousal repeatedly leads to discomfort—especially **delayed pain hours or days later**—the nervous system begins to **downregulate sexual drive as a protective response**. This is not purely psychological; it reflects **neuroplastic changes in pain–reward circuits**, where previously rewarding stimuli become linked with **threat or nociception**.

From a peripheral perspective, decreased libido may also be associated with **chronic pelvic congestion and impaired outflow of prostatic and seminal vesicle secretions**. In some patients, repeated cycles of **incomplete drainage, increased viscosity of secretions, or ductal obstruction** may contribute to **stasis of seminal fluid**. Over time, this may promote **local inflammatory processes**, including **non-bacterial inflammation, microcalcifications, fibrosis, and structural remodeling**. Imaging such as **high-resolution MRI (including 3T)** may reveal features consistent with **congestion, post-inflammatory changes, or structural irregularities** within the prostate and surrounding tissues.

These local processes may be accompanied by **neurogenic inflammation**, where repeated activation of pelvic nerves leads to release of mediators such as **substance P and CGRP**, increasing **tissue sensitivity and maintaining chronic pain**. In parallel, a subset of patients may develop **immune-mediated or mast cell–related responses**, where inflammatory signaling becomes **amplified and self-sustaining**. While not strictly autoimmune, these mechanisms may resemble **localized immune dysregulation**, contributing to symptoms even in the absence of infection.

At the systemic level, decreased libido is often reinforced by **chronic stress, autonomic imbalance, and hormonal dysregulation**. Persistent pain may **disrupt sleep, elevate cortisol, and negatively affect testosterone signaling**. Additionally, **pelvic floor overactivity and chronic muscle tension** may impair coordination between **arousal, erection, and ejaculation**, further reducing sexual function.

Clinically, decreased libido becomes particularly meaningful when it coexists with:

- **pain during or after ejaculation**
- **erectile dysfunction or reduced erection quality**
- **delayed or weakened ejaculation**
- **pelvic heaviness or deep pressure sensations**
- **fatigue, sleep disturbance, or mood changes**

In such combinations, it indicates that the **sexual system is no longer functioning independently**, but has become **integrated into the chronic pain process**. Sexual desire is therefore reduced not as a primary disorder, but as a **consequence of repeated physiological strain, impaired recovery, and ongoing pelvic dysregulation**.

In summary, decreased libido in CPPS is a multifactorial outcome, involving:

- **pelvic congestion and fluid stasis**

- **inflammatory and post-inflammatory tissue changes**
- **neural sensitization**
- **immune and histamine-related activation**
- **autonomic and hormonal imbalance**

It reflects an **adaptive but clinically significant response**, where the body reduces sexual drive because the pelvic system is **no longer able to tolerate normal activation without triggering symptoms**.

Increased libido

Increased libido is not inherently pathological and should not be considered abnormal. However, within the context of chronic pelvic pain syndrome (CPPS), it may have clinical relevance as an **exposure-related factor**, reflecting a higher frequency of sexual activation and ejaculatory events. Its importance arises when it interacts with an already sensitized pelvic system, potentially contributing to a **load–recovery imbalance**.

Under physiological conditions, male sexual function follows a balanced cycle of arousal, ejaculation, and recovery. This cycle allows adequate normalization of the pelvic floor muscles, prostate, seminal vesicles, vascular circulation, and associated neural pathways. In some individuals, however, particularly those with pre-existing pelvic sensitivity, frequent or repetitive sexual stimulation may exceed the system's capacity to fully recover between events.

Two distinct but sometimes overlapping mechanisms may emerge:

1. Sexual Overload / Repetitive Activation

In patients with high libido and frequent ejaculation, repeated activation of the pelvic floor and deep pelvic structures may lead to:

- **pelvic floor muscle overuse**, resulting in chronic hypertonicity, impaired relaxation, and trigger point formation,
- **increased intrapelvic pressure and mechanical strain**,
- **neurogenic inflammation**, driven by repeated activation of sensory nerve endings.

This creates a self-reinforcing cycle: pain → muscle guarding → increased nociceptive input → further pain.

2. Seminal Fluid Stasis and Congestion

Conversely, in some individuals, irregular ejaculation patterns or incomplete emptying of the seminal vesicles may contribute to:

- **local fluid stasis**,
- increased intraluminal pressure within the seminal vesicles and prostate,
- **chemical irritation of surrounding tissues**,
- and a tendency toward **low-grade, non-bacterial inflammatory responses**.

This mechanism is particularly relevant in patients reporting deep pelvic pressure, fullness, or delayed post-ejaculatory discomfort.

Neurogenic and Immune Mechanisms

At the neural level, repeated stimulation may lead to **sensitization of peripheral and central pain pathways**. A key mediator in this process is **substance P**, along with other neuropeptides such as CGRP. These molecules:

- increase vascular permeability,
- enhance pain signaling,
- and promote interaction between nerves and immune cells.

Substance P also activates **mast cells**, linking the nervous and immune systems. This interaction may amplify inflammation even in the absence of infection.

In a subset of patients, this process evolves into a **neuroimmune amplification loop**, where:

- mast cells release histamine and pro-inflammatory mediators,
- histamine further sensitizes nerve endings,
- and pain perception becomes heightened and more persistent.

Clinically, this may present as burning pain, urgency, pressure, and sensitivity to stress, food, or mechanical stimuli — forming a **histamine-reactive phenotype**.

Inflammatory and Autoimmune-like Components

Although CPPS is classified as a non-bacterial condition in the majority of cases (with confirmed infection present in only approximately 5–10%), persistent local irritation and neuroimmune activation may lead to a state of **chronic sterile inflammation**.

In some patients, repeated tissue stress, microtrauma, and prolonged immune activation may contribute to:

- **dysregulated immune responses**,
- local production of inflammatory cytokines,
- and, in certain cases, **autoimmune-like reactions**, where the immune system maintains inflammation without an external pathogen.

This does not necessarily represent classical autoimmune disease, but rather a **self-sustaining inflammatory state** driven by internal signaling pathways.

Clinical Interpretation

The relevance of increased libido in this context is therefore conditional. It becomes clinically meaningful when it coexists with:

- pain during or after ejaculation,
- worsening with frequent sexual activity,

- difficulty achieving symptom-free recovery,
- pelvic floor tension,
- or deep pelvic/prostate-region discomfort.

In such cases, increased libido may function as a **modulator of symptom load**, increasing the frequency of pelvic activation and thereby amplifying underlying dysfunction.

Importantly, current medical evidence does **not** support the claim that high libido or frequent ejaculation is inherently harmful or universally damaging to the prostate or seminal vesicles. Instead, it supports a **phenotype-based interpretation**, where sexual activity may:

- aggravate symptoms in some individuals,
- relieve congestion in others,
- or have a bidirectional effect depending on frequency and individual physiology.

Summary

Chronic pelvic pain should be understood as a **multisystem disorder**, involving dynamic interactions between muscular, neural, vascular, and immune processes. Increased libido, within this framework, is not a cause of disease but a **potential amplifier of underlying vulnerability**, particularly when combined with impaired recovery, neurogenic sensitization, and local inflammatory or immune dysregulation.

Effective management therefore requires addressing multiple pathways simultaneously — including pelvic floor function, neural regulation, and immune balance — to restore physiological equilibrium within the pelvic system.

Difficulty relieving sexual tension

This item suggests that sexual arousal does not resolve smoothly and may remain physiologically "unfinished," leading to persistent pelvic activation, pressure, or autonomic tension. This can be clinically important because the body may remain in a partially **activated pelvic state** even after stimulation ends. If this answer coexists with **pain during ejaculation, pain after ejaculation, high libido, interrupted intercourse history, or delayed ejaculation patterns**, it supports a **sexual regulation / pelvic overactivation phenotype**.

Penile hypersensitivity causing rapid ejaculation

This answer suggests that the sexual system is characterized by **sensory over-reactivity**, such that penile input reaches threshold rapidly. In chronic pelvic pain, this may fit a broader **genital sensory amplification phenotype**, especially if there is also **shaft or glans pain, burning, prickling, allodynia, or urethral sensitivity**. It indicates that sexual dysfunction in CPPS may be driven not only by pain avoidance or low libido, but also by **excessive sensory gain**.

Difficulty achieving orgasm and ejaculation

This option suggests impaired sexual completion rather than only painful sexual completion. It may reflect **pelvic floor overcontrol, altered autonomic transition, fear of pain, reduced ejaculatory force, endocrine factors, medication effects, or deep sexual-system dysregulation**. This is

especially important when paired with **weak ejaculation, decreased libido, erectile dysfunction, pain during ejaculation, or psychological overcontrol**, because it suggests a broad sexual dysfunction phenotype within chronic pelvic pain.

Erectile dysfunction

Erectile dysfunction is highly relevant because it shows that the syndrome affects not only pain and ejaculation but also the **arousal phase of sexual function**. In chronic pelvic pain, ED may reflect autonomic dysregulation, pelvic floor overactivity, pain avoidance, poor sleep, anxiety, reduced libido, vascular factors, or medication effects. It becomes especially important when interpreted together with **decreased libido, pain after ejaculation, tadalafil response, stress sensitivity, and endocrine context**. Chronic pelvic pain guidelines and sexual medicine guidance both support evaluating erectile symptoms in the broader sexual and pelvic phenotype rather than in isolation.

Summary of Point 10

Point 10 is the questionnaire's **sexual physiology and ejaculatory pain phenotype section**. Its purpose is to identify how the pelvic pain system responds to **arousal, orgasm, ejaculation, abstinence, frequency of ejaculation, and the deeper sexual apparatus**. Pain during or immediately after ejaculation supports a **pelvic floor / ejaculatory-load phenotype**. Pain several hours or the next day after ejaculation supports a **delayed flare phenotype**, potentially involving inflammatory, sensory, or neuroimmune amplification. Relief after ejaculation and worsening after abstinence support a **congestion-sensitive phenotype**. Worsening with frequent daily ejaculation supports a **sexual-overload phenotype** in which recovery between ejaculatory events may be insufficient for that particular patient. Pain in the seminal vesicle region, reduced semen volume, and weak ejaculation strengthen the importance of the **deep ejaculatory tract**. Urethral burning after ejaculation supports a **urethral–sensory irritative phenotype**. Libido, sexual tension, orgasm difficulty, and erectile dysfunction show that chronic pelvic pain often becomes a full **sexual-function syndrome**, not just a pain syndrome. In professional interpretation, Point 10 is indispensable because it identifies whether sexuality is a major trigger, a consequence, or both — and what specific sexual-pelvic mechanism seems most likely to be active.

Point 11. Mechanical and External Triggers. Symptoms of pain worsen with:

(select all that apply)

Purpose of Point 11

Point 11 is designed to identify whether the pain behaves as a **mechanically provoked, posture-sensitive, load-sensitive, or environmentally reactive syndrome**. This is one of the most important interpretive sections in the entire questionnaire because chronic pelvic pain is often not constant in the same way throughout the day. Instead, in many patients it is **modulated by position, pressure, movement, load transfer, temperature, and cumulative tissue stress**. In clinical terms, this point helps determine whether the pelvis is acting like a pain system that is especially vulnerable to **compression, sustained posture, abdominal pressure, musculoskeletal loading, or vascular-environmental influences**.

This question is also essential because it helps separate different pelvic pain phenotypes that may otherwise sound similar. A patient with deep pelvic pain may describe it as "prostate pain," but if it is strongly triggered by sitting, driving, cycling, tight clothing, coughing, bending, or lifting, the pain is behaving much more like a **pelvic floor / pudendal / musculoskeletal / pressure-sensitive syndrome** than a purely gland-based disorder. Current chronic pelvic pain guidance explicitly supports this broader interpretive model and recommends that clinicians evaluate **musculoskeletal and functional abnormalities** as part of the chronic pelvic pain work-up.

A further reason this section matters is that it identifies the **real-life physical environment** in which pain is sustained. Men with chronic pelvic pain may repeatedly expose the pelvis to the exact conditions that worsen symptoms, often without recognizing the pattern. Point 11 therefore serves as a bridge between anatomy and everyday behaviour. It asks whether pain is worsened by the kinds of load and postures most likely to affect the **pelvic floor, pudendal region, lower abdominal wall, hips, sacrum, and pelvic venous outflow**.

Prolonged sitting

This is one of the strongest items in the entire questionnaire. If pain worsens with prolonged sitting, the evaluator should strongly consider a **mechanical-compressive pelvic phenotype**, especially involving the **perineum, pelvic floor, pudendal territory, and deep posterior pelvic tissues**. Sitting places sustained load on the perineum and often also promotes pelvic floor shortening, gluteal underuse, hip flexion tightness, and reduced pelvic movement variability. In a sensitized patient, that combination can provoke pain very reliably.

Clinically, this answer becomes especially meaningful when it is later combined with **perineal pain, anal pain, genital pain, tingling or numbness while sitting, driving pain, cycling pain, or a Nantes-like pattern of pudendal-territory symptoms**. In that context, prolonged sitting strongly supports a **pudendal / myofascial / mechanical pelvic floor phenotype**. Current expert guidance on male chronic pelvic pain also emphasizes pelvic floor myalgia and dysfunction as common features in men with CPPS.

Driving (seat pressure)

Driving is not simply another version of sitting. It adds **seat pressure, limited positional adjustment, vibration, trunk fixation, hip flexion, and often prolonged immobility**. If pain worsens while driving, that often means the pelvis is especially reactive to **focused seated compression and static loading**, rather than just to ordinary sitting time alone.

This answer is particularly informative in patients who also report **pain worsened by sitting, pain in the perineum or anus, posterior testicular/prostate-region pain, or symptoms spreading into the thighs and buttocks**. In such cases, driving pain strongly supports a **seat-pressure–dependent mechanical phenotype**, and in some men may raise concern for pudendal or sacral pathway irritability.

Cycling (pressure on seat)

Cycling is one of the most classically relevant triggers in pelvic pain because it produces direct, repeated **saddle pressure on the perineum and pudendal region**. If cycling worsens pain, the evaluator should think seriously about a **compression-sensitive pelvic phenotype**, especially if the patient also reports perineal pain, genital pain, anal pain, or sitting intolerance.

This answer is especially compelling because the mechanical logic is clear: saddle pressure directly loads the same region that is frequently symptomatic in pelvic floor and pudendal-related pain. In

the right clinical context, cycling sensitivity is one of the strongest real-world clues that the pelvis is behaving as a **pressure-sensitive neural–myofascial system**.

Tight clothing

Worsening with tight clothing suggests that the pain system is reactive not only to large-scale posture and load, but also to **local external compression**. This may support several overlapping interpretations: a **cutaneous or superficial sensory hypersensitivity**, a **pudendal or genital allodynic phenotype**, or a **pressure-sensitive pelvic floor / mast-cell-reactive environment**.

This answer becomes particularly important if it later coexists with **glans pain, penile hypersensitivity, perineal hypersensitivity, light touch causing pain, dermographism, or pressure as an immune trigger**. In those cases, tight clothing may reveal that the patient's pelvis is not only mechanically vulnerable but also **sensory-amplified or pressure-reactive at the skin/fascial interface**.

Pressure from the trouser belt on the stomach

This item is especially interesting because it suggests that pain can be worsened not only by direct perineal pressure, but also by **abdominal compression and increased pressure transmission into the pelvis**. A tight trouser belt may increase lower abdominal wall tension, alter diaphragmatic breathing, affect bowel comfort, and mechanically transmit pressure downward into the pelvic floor and bladder region.

This answer becomes more significant if later parts of the questionnaire show **lower abdominal pain, bladder pressure, bloating, constipation, difficulty breathing into the abdomen, or pelvic pressure when the bladder is full**. In that context, the pelvis may be part of a larger **abdomino-pelvic pressure and guarding phenotype** rather than a pain syndrome confined to the perineum alone.

Sitting cross-legged

Pain worsened by sitting cross-legged suggests a positional sensitivity that likely involves **pelvic alignment, hip rotation, adductor tension, and asymmetric load transfer through the pelvis**. This can be especially relevant in men whose chronic pelvic pain overlaps with **hip tightness, groin pain, inner thigh pain, or lower back stiffness**.

This answer often supports a **biomechanical or myofascial phenotype**, particularly when combined with other musculoskeletal signs. It suggests that pelvic pain is sensitive not only to pressure, but also to **how the pelvis is being held in space**.

Leaning forward

Leaning forward can increase pressure through the lower abdomen, compress the pelvic outlet, change sacral and lumbar loading, and increase contact pressure on the perineum if sitting at the same time. If pain worsens in this position, it often suggests a **pressure-transmission or posture-sensitive phenotype**, especially involving the **perineum, pelvic floor, or posterior pelvic structures**.

This answer becomes especially meaningful if it coexists with **driving pain, prolonged sitting pain, deep perineal pressure, or bladder-related discomfort**, because that combination suggests that forward posture may intensify existing pelvic loading rather than being a neutral movement.

Increased abdominal pressure

This item is crucial because it directly tests whether the pelvis reacts badly when **intra-abdominal pressure rises**. Such pressure increases occur during straining, lifting, bracing, certain exercises, coughing, and even breath-holding. If pain worsens in that context, the evaluator should think strongly about a **pelvic floor, lower abdominal wall, outlet, or lumbopelvic stabilization phenotype**.

This answer becomes particularly important if later sections reveal **difficulty relaxing the pelvic floor, pain with muscle contraction, constipation-related pain, difficulty passing stool, or heavy-lifting sensitivity**. It supports the idea that the pelvis is failing to tolerate pressure in a coordinated way and may be responding with **spasm, guarding, outlet dysfunction, or pain amplification**. EAU guidance specifically discusses pelvic floor overactivity and the importance of pelvic floor evaluation and treatment in chronic pelvic pain.

Heavy lifting

Heavy lifting is one of the most clinically meaningful triggers because it combines **increased abdominal pressure, pelvic stabilization demand, spinal loading, and muscular recruitment**. If lifting worsens pain, it often supports a **lumbopelvic–pelvic floor biomechanical phenotype**, especially in patients who also report low-back pain, sacral pain, groin pain, or tension during exertion.

Lifting-related pain is especially important in men whose symptoms have a strong occupational component or began after exertion. In chronic pelvic pain, the issue is often not “lifting damaged the prostate,” but rather that lifting reveals a **poorly coordinated pressure-management system** involving the abdomen, diaphragm, pelvic floor, hips, and spine.

Bending

Pain worsened by bending suggests that the pelvic system may be sensitive to **forward-flexion mechanics, abdominal compression, lumbar movement, or pelvic floor load transfer**. Bending-related worsening often fits a more **musculoskeletal or pressure-sensitive phenotype** than a purely bladder- or gland-dominated one.

This answer gains significance if it appears with **low-back pain, sacral pain, increased abdominal pressure sensitivity, or deep pelvic floor symptoms**, because together these findings suggest that movement itself is part of the pelvic pain network.

Standing

Standing worsens pain in a somewhat different way from sitting. If standing is a major aggravator, clinicians may need to consider **vascular congestion, static muscular overuse, posture-related pelvic floor load, or deep tissue heaviness** rather than only seated compression.

This answer becomes especially important when paired with **pelvic heaviness, fullness, improvement when lying down, visible varicose veins, or evening worsening**, because that combination raises suspicion for a **pelvic venous or congestion-related phenotype**. Standing may then be aggravating symptoms through prolonged gravitational loading rather than direct

compression. Pelvic venous insufficiency is recognized as a contributor to chronic pelvic pain, including in men, although it is less commonly identified than in women.

Walking

Walking is an especially interesting trigger because in some patients movement improves symptoms, while in others it worsens them. If walking worsens pain, this may suggest **musculoskeletal loading, hip–pelvis dysfunction, deep tissue friction/activation, or a lumbopelvic instability pattern**.

This option becomes particularly important if later parts of the survey show **hip pain, groin pain, lower back stiffness, pain with hip movement, or spinal injury history**. In those cases, walking-related worsening supports a **biomechanical or referred pain phenotype** rather than a purely seated compression pattern.

Lying down

Pain that worsens when lying down is less common and therefore especially informative. In many chronic pelvic pain syndromes, lying down relieves pressure. If it instead worsens pain, the evaluator should think more carefully about **persistent neural pain, bladder filling at rest, autonomic hypervigilance, or a pain state no longer dependent on upright load alone**.

This answer becomes most significant when interpreted alongside **night pain, bladder filling pain, nocturnal urgency, poor sleep, or continuous all-day pain**. It often suggests that the syndrome is more entrenched and not simply explained by daytime mechanics.

Coughing

Coughing is a very useful mechanical trigger because it briefly but sharply increases **intra-abdominal pressure**. If pain worsens with coughing, it supports the idea that the pelvic system is vulnerable to **pressure spikes**, which often implicates the pelvic floor, lower abdominal wall, outlet, or lumbopelvic stabilization pattern.

This answer is especially useful when paired with **constipation-related pain, heavy-lifting sensitivity, difficulty relaxing the pelvic floor, or pressure from a trouser belt**, because that broader picture supports a **pressure-sensitive pelvic phenotype** rather than a random symptom pattern.

Heat exposure

Heat sensitivity in chronic pelvic pain is highly informative because it can point toward **vascular dilation, inflammatory reactivity, mast-cell / histamine sensitivity, or worsening of deep pelvic congestion**. While heat therapy helps some patients, worsening with heat suggests a different physiology.

This answer becomes especially important if the patient also reports **pelvic heaviness, fullness, standing-related worsening, visible varicosities, flushing, or histamine-related food and allergy triggers**. In that setting, heat may be aggravating a **vascular or neuroimmune-reactive phenotype** rather than simply “loosening muscles.”

Cold exposure

Cold exposure often worsens symptoms in patients with **muscular guarding, autonomic over-reactivity, vasoconstrictive sensitivity, or cold-triggered sensory amplification**. In practical terms, cold may provoke pelvic floor tightening, decrease comfort in already sensitive tissues, and intensify protective guarding.

This answer becomes particularly meaningful if later sections show **hypersensitivity to cold, pelvic floor spasm, chest-dominant breathing, stress-related holding patterns, skin reaction to cold, or benefit from heat therapy**. In that context, cold sensitivity supports a **muscular–autonomic–sensory phenotype** and sometimes, in selected patients, may also overlap with **histamine- or mast-cell–reactive physiology**.

Summary of Point 11

Point 11 is the questionnaire's **mechanical, positional, and environmental trigger section**. Its purpose is to determine whether the pain behaves like a syndrome that is particularly sensitive to **pressure, posture, abdominal loading, musculoskeletal movement, or temperature**. **Prolonged sitting, driving, cycling, and leaning forward** strongly support a **mechanical-compressive / pudendal / pelvic floor phenotype**. **Increased abdominal pressure, heavy lifting, coughing, bending, and belt pressure** support a **pressure-sensitive pelvic floor and lumbopelvic stabilization phenotype**. **Standing, heat exposure, pelvic heaviness, and relief when lying down** begin to raise the possibility of **vascular congestion**. **Walking, hip-related symptoms, and cross-legged sitting** support a **musculoskeletal–biomechanical phenotype**. **Cold exposure** supports a **muscular–autonomic or sensory-reactive phenotype**. In professional interpretation, Point 11 is essential because it shows whether pain is being actively shaped by the body's physical environment, not only by organs or abstract pain mechanisms.

Point 12. Problems with pelvic blood supply

(select all that apply)

Purpose of Point 12

Point 12 is designed to determine whether the patient's symptom picture includes a meaningful **vascular or pelvic congestion component**. This is an especially valuable section because vascular mechanisms are often under-recognized in male chronic pelvic pain, yet they can substantially alter how pain feels, when it worsens, and why certain positions or activities aggravate symptoms. In other words, this point asks whether the pelvis may be behaving not only as a muscular or neural pain system, but also as a **pressure-loaded venous compartment** with impaired or vulnerable drainage.

This matters because vascular-type pelvic symptoms often feel very different from classic neuropathic or myofascial pain. Instead of burning, electric shocks, or sharp trigger-point pain, vascular-predominant discomfort is more often described as **heaviness, fullness, pressure, deep aching, worsening later in the day, and relief when the pelvis is unloaded**. Those qualities can easily be misinterpreted by patients as "prostate pressure" or "deep pelvic inflammation," even when the true pattern is more compatible with venous pooling or congestion physiology.

Point 12 is also important because vascular and non-vascular mechanisms are **not mutually exclusive**. A man may have pelvic venous congestion and also develop secondary pelvic floor guarding, pudendal irritation, bladder pressure sensitivity, or post-ejaculatory pain. Likewise, a patient with chronic pelvic floor tension may describe "pelvic fullness," but the true driver may still be primarily muscular rather than venous. This section therefore does not diagnose one condition in

isolation. It identifies whether a **vascular layer** is plausible and deserves to be integrated into the overall pelvic pain phenotype.

Vascular / Pelvic Congestion Symptoms

This option functions as a broad umbrella signal that the patient recognizes symptoms suggestive of **pelvic vascular loading or venous congestion**. It is less specific than the more focused items below, but it is still clinically meaningful because some patients intuitively perceive their symptoms as "congestion-like" even before they can describe them precisely. In such men, the pain is often experienced as **deep, heavy, pressure-like, internal, loaded, or worsening after prolonged standing or sitting**, rather than primarily burning or stabbing.

This response becomes especially important when it is accompanied by the more specific vascular descriptors in the same section, such as **pelvic heaviness or pressure, fullness in the pelvis, improvement on lying down, and visible varicose veins**. When these features cluster together, the vascular interpretation becomes much stronger. Although male pelvic congestion remains less frequently described than female pelvic congestion in the literature, recent reports and reviews increasingly acknowledge that **pelvic venous disorders in men are real, under-recognized, and capable of producing chronic pelvic pain**.

Pelvic heaviness or pressure

This is one of the most informative vascular items in the whole questionnaire. Pelvic heaviness or pressure typically suggests that the patient experiences pain less as sharp irritation and more as **deep burden, pelvic loading, or an internal sense of weight**. That symptom quality is highly compatible with a **venous congestion phenotype**, although it can also overlap with deep pelvic floor tension, post-ejaculatory fullness, or bladder/rectal pressure sensitivity.

This answer becomes more compelling if it occurs together with **standing-related worsening, evening pain, deep pain behind the testicles/prostate region, seminal vesicle-region fullness, or relief when lying down**. In that context, heaviness is not just a vague subjective description; it suggests that the pelvis may be functioning like a **poorly unloading deep compartment**, in which pressure accumulates over time. It is also important because some men describe this sensation as "inflamed prostate pressure," even though the phenomenology may actually fit better with congestion or pelvic compartment loading.

Pain improves when lying down

This is one of the strongest vascular clues in the entire questionnaire. Improvement when lying down strongly suggests that symptoms are at least partly relieved by **removing gravitational load and improving venous return from the pelvis**. In a congestion-sensitive patient, the transition to recumbency may function as a natural decompression test.

Clinically, this answer becomes especially significant if the patient also reports **pelvic heaviness, fullness, standing-related worsening, visible scrotal varicosities, or evening accumulation of symptoms**. That combination strongly supports a vascular contribution. By contrast, if lying down worsens symptoms rather than relieving them, then a simple venous-congestion interpretation becomes less likely and the evaluator may need to think more about **bladder filling at rest, night pain, central sensitization, or persistent neuropathic pain**. This is why this item has high differential value: it does not merely describe a comfortable position, it tests whether **offloading the pelvis changes the pain meaningfully**.

Feeling of fullness in pelvis

Fullness in the pelvis is closely related to heaviness and pressure, but it often conveys a slightly different internal quality. Rather than weight alone, it suggests a **crowded, loaded, distended, or internally "filled" sensation**. In the right context, this strongly supports a **venous pooling or deep congestion interpretation**.

This answer becomes particularly relevant if the patient also reports **worsening after abstinence, deep seminal vesicle-region discomfort, pain behind the testicles/prostate region, standing-related worsening, or relief after lying down**. In that pattern, pelvic fullness may represent an interaction between **vascular congestion and the deep ejaculatory / peri-prostatic system**. It can also coexist with sexual-triggered symptoms, especially in men who feel that repeated sexual activation or prolonged abstinence changes the internal deep-pelvic "pressure state." This does not prove a single mechanism, but it does suggest that the symptom is not merely superficial pain; it is a **deep pelvic compartment sensation**.

Visible varicose veins (e.g., scrotum)

Visible varicose veins, especially in the scrotal region, provide one of the most concrete clues in this section. Their presence suggests that the venous system may be abnormal not just functionally but also anatomically or hemodynamically. In men, scrotal venous enlargement may coexist with broader pelvic venous insufficiency or reflux patterns, although the exact relationship can vary between patients.

This answer becomes especially powerful when it is paired with **pelvic heaviness, fullness, standing-related worsening, and improvement when lying down**. In that combination, the case for a **vascular-loading phenotype** becomes substantially stronger. It is still important to remember that visible varicosities do not automatically explain all of the patient's symptoms. They may be one contributor within a broader chronic pelvic pain syndrome that also includes pelvic floor overactivity, sexual-triggered pain, or sensory amplification. But when present, they should prompt serious consideration of **venous contribution rather than defaulting automatically to a prostate-only narrative**.

Summary of Point 12

Point 12 is the questionnaire's **vascular and pelvic congestion section**. Its purpose is to identify whether the patient's pelvic pain may include a meaningful **venous, pressure, or blood-flow related component**. **Pelvic heaviness or pressure** and **feeling of fullness in the pelvis** support a deep pressure-loaded phenotype. **Pain improves when lying down** is especially valuable because it suggests that unloading and venous drainage meaningfully affect symptoms. **Visible varicose veins**, especially in the scrotum, strengthen the plausibility of a pelvic venous contribution. In professional interpretation, Point 12 is most useful in patients whose pain feels **deep, heavy, full, pressure-like, posture-dependent, and worse with prolonged upright loading or late in the day**. It should not be used to replace other explanations, but to identify whether a **vascular layer** is helping shape the overall pain phenotype.

Point 13. Breathing Pattern

(select all that apply)

Purpose of Point 13

Point 13 is designed to identify whether the patient's chronic pelvic pain is being maintained in part by an **abnormal breathing-regulation pattern**, especially one that promotes chronic pelvic floor guarding, abdominal tension, autonomic overactivation, and poor down-regulation. This is a very important section because the diaphragm, abdominal wall, and pelvic floor do **not** function as isolated systems. They form a mechanically and neurologically linked pressure-regulation unit. Disrupted breathing patterns can therefore change pelvic floor behaviour, pain sensitivity, and symptom flares.

Current EAU management guidance explicitly recognizes pelvic floor overactivity as a contributor to chronic pelvic pain and recommends learning pelvic floor relaxation in appropriate patients. The clinical relevance of Point 13 is that breathing is one of the most important gateways into pelvic floor relaxation or, conversely, pelvic floor over-recruitment. If breathing is chronically shallow, upper-chest dominant, braced, or stress-held, then the pelvic floor may remain in a partially guarded state for long periods.

This point is also essential because it helps connect the pelvic pain syndrome to the patient's **autonomic style of regulation**. In many men with chronic pelvic pain, the body behaves as if it is continuously preparing for effort or threat: the chest leads the breath, the abdomen remains held, the breath is intermittently suspended under stress, and full exhalation/relaxation never truly occurs. When that pattern is present, the pelvis is not just hurting; it is being maintained inside a **breathing-linked state of tension and vigilance**.

Chest-dominant breathing

Chest-dominant breathing suggests that the patient relies more heavily on the **upper chest, accessory respiratory muscles, and thoracic expansion** than on a coordinated diaphragmatic-abdominal breathing pattern. In practical terms, this often reflects a body that is breathing in a more **guarded, effortful, or vigilance-related mode** rather than in a relaxed regulatory mode.

This is clinically important because chest-dominant breathing often correlates with **abdominal holding, reduced diaphragmatic descent, impaired pelvic floor excursion, and chronic stress physiology**. In a patient with chronic pelvic pain, that can support the maintenance of **pelvic floor hypertonicity**, especially if later sections also show perineal tension, involuntary pelvic floor clenching, difficulty relaxing the pelvic floor, stress-worsened symptoms, or lower-abdominal cramping under stress. Rather than being a superficial lifestyle observation, chest-dominant breathing may identify a patient whose pelvis is embedded in a **whole-body guarding strategy**.

This answer becomes even more meaningful if the patient later reports benefit from **breathing exercises, meditation, pelvic floor relaxation training, or psychotherapy**, because that suggests the breathing pattern is not incidental but part of the pain-maintaining mechanism. It may also matter in patients with bladder symptoms, because poor abdominal–diaphragmatic coordination can change how pressure is transmitted into the pelvic floor and lower urinary tract.

Difficulty breathing into abdomen

Difficulty breathing into the abdomen is one of the clearest clues that the patient may have impaired **diaphragmatic-abdominal expansion** and reduced pressure-distribution flexibility. In a healthy coordinated pattern, diaphragmatic breathing allows the abdomen and pelvic floor to participate in a dynamic pressure cycle. If a patient feels unable to breathe into the abdomen, this often means the body is functioning in a state of **abdominal bracing, diaphragmatic restriction, or pelvic floor co-contraction**.

Clinically, this answer becomes highly relevant when paired with **lower abdominal pain, pressure from a trouser belt, increased abdominal pressure as a pain trigger, constipation-related pelvic pain, difficulty passing stool, or feelings of pelvic fullness/pressure**. In such cases, the inability to breathe comfortably into the abdomen may indicate that the patient's abdomino-pelvic pressure system is **chronically over-controlled and poorly distributed**. That can contribute to bladder symptoms, bowel symptoms, pelvic floor tension, and deep pelvic pain.

This answer also has therapeutic implications. If diaphragmatic breathing later proves helpful in treatment-response sections, that strengthens the interpretation that poor abdominal breathing was part of the active phenotype rather than a neutral observation. It suggests that breathing retraining may improve symptoms not by "relaxing the patient psychologically" alone, but by changing the **mechanics and neuroregulation of the pelvis itself**.

Breath holding during stress

Breath holding during stress is one of the most revealing psychophysiological items in the whole survey. It suggests that the patient's body responds to stress not only emotionally or cognitively, but also through a **motor-pressure holding pattern**. Breath holding often coexists with **jaw clenching, abdominal bracing, anal or pelvic floor tightening, and transient increases in intra-abdominal pressure**, all of which can be highly relevant in chronic pelvic pain.

This answer becomes especially significant when later sections show **stress-triggered symptom worsening, difficulty relaxing, pelvic floor clenching, lower-abdominal cramps under stress, urinary hesitancy, or pain worsened by abdominal pressure, lifting, and coughing**. In such a pattern, breath holding is not just a stress habit; it is likely part of the direct physiological route through which stress is translated into pelvic pain. In many patients, this is one of the clearest examples of how the nervous system, respiration, and pelvic floor become linked in a chronic pain syndrome.

This is also one of the best examples of why modern chronic pelvic pain care is multidisciplinary. The problem is not merely "stress" in an abstract psychological sense. It is the fact that stress is being expressed through **breathing interruption and pressure dysregulation**, which can directly load the pelvic system.

Feeling unable to fully relax breathing

This option suggests that the patient perceives breathing itself as never fully settling into a relaxed, effortless state. This often reflects a more **persistent background of autonomic activation**, in which even at rest the respiratory system remains guarded, incomplete, or subtly effortful.

Clinically, this is very important because it suggests that the patient may have difficulty reaching the kind of **deep down-regulation** that allows the pelvic floor to let go. If the breath never fully relaxes, then the abdomen, pelvis, and pelvic floor may remain in a low-grade state of readiness. This answer becomes especially meaningful when paired with **difficulty relaxing the pelvic floor, pain that worsens during mental stress, poor sleep, chest-dominant breathing, and symptom improvement with breathing exercises or relaxation-based therapies**. In that context, the breathing pattern is not just a symptom; it is a marker of a **chronically overactivated regulatory state**.

This option may also indicate why some patients feel that they are "never fully off," even when they are not consciously anxious. Their breathing pattern may be revealing the body's version of chronic vigilance. That is exactly the kind of information a phenotype-based questionnaire should capture,

because it helps explain why symptoms may persist even when obvious external triggers are not present.

Summary of Point 13

Point 13 is the questionnaire's **breathing-regulation and pressure-management section**. Its purpose is to identify whether the patient's pelvic pain is being maintained in part by **poor diaphragmatic function, abdominal bracing, stress-linked breath interruption, and incomplete respiratory relaxation**. **Chest-dominant breathing** supports a guarded, upper-body, vigilance-linked breathing style. **Difficulty breathing into the abdomen** suggests impaired diaphragmatic-abdominal pressure distribution and often supports an abdomino-pelvic dysfunction phenotype. **Breath holding during stress** is especially important because it reveals a direct pathway through which stress may be translated into pelvic pressure, muscle guarding, and pain. **Feeling unable to fully relax breathing** suggests a more chronic background of autonomic overactivation and incomplete down-regulation. In professional interpretation, Point 13 is essential because it helps identify patients whose chronic pelvic pain is not only a local pelvic problem, but also part of a broader **respiratory–autonomic–pelvic floor dysregulation pattern**.

Point 14. Pelvic Floor Muscle Symptoms

(select all that apply)

Purpose of Point 14

Point 14 is one of the most important sections in the entire questionnaire because it asks whether the pelvic pain syndrome is being generated, amplified, or maintained by the **pelvic floor and the broader lumbopelvic musculoskeletal system**. In modern chronic pelvic pain medicine, this is not a secondary issue. Male chronic pelvic pain frequently includes a **pelvic floor myalgia / overactivity phenotype**, and current AUA educational summaries of the 2025 guideline specifically state that **pelvic floor myalgia or dysfunction is very common in CPPS**, with pain during ejaculation being one especially strong clue.

The purpose of this point is therefore not simply to ask whether the patient "has tight muscles." It is to determine whether the patient's pain behaves like a condition in which the pelvic floor has become a **final common pathway** for multiple other inputs: stress, urinary symptoms, bowel dysfunction, sexual overactivation, pelvic guarding, altered breathing, sitting intolerance, or neural irritation. When these muscles become chronically overactive, shortened, painful, uncoordinated, or unable to relax, they can produce local pain, referred pain, urinary dysfunction, bowel dysfunction, and sexual symptoms. The EAU guideline specifically notes that pelvic floor overactivity is relevant in prostate pain and bladder pain phenotypes and that learning relaxation can help interrupt the **pain–spasm–pain cycle**.

This section is also important because it helps distinguish between pain that is primarily **felt in the pelvis** and pain that is actively **generated by the pelvic musculoskeletal container**. A man may describe "prostate pain," "bladder pain," or "pain behind the testicles," but if he also reports perineal tension, involuntary clenching, pain with pelvic muscle contraction, constipation-associated pelvic pain, rectal fullness, and hip/lower-back stiffness, then the deeper interpretation becomes much more consistent with a **pelvic floor–myofascial phenotype**.

Tension in the perineum

Tension in the perineum is one of the clearest direct indicators that the patient experiences the pelvic floor not only as a pain region, but as a **tonically activated structure**. Clinically, this often corresponds to a sensation of tightness, holding, pressure, or guarded discomfort in the area between the scrotum and anus. In chronic pelvic pain, this frequently supports a **pelvic floor hypertonicity phenotype**, especially if accompanied by sitting pain, anal pain, bladder pressure, post-ejaculatory pain, or stress-related symptom worsening.

Perineal tension is especially important because it often functions as a **linking symptom**. It can connect urinary complaints, bowel complaints, and sexual pain into one coherent mechanical/functional model. If the patient later reports benefit from **pelvic floor physiotherapy, trigger point work, diaphragmatic breathing, or learning to relax the pelvic floor**, that interpretation becomes even stronger.

Involuntary pelvic floor muscle clenching

This item suggests that the pelvic floor is not just tight when the patient consciously notices it, but that it may be **automatically and repeatedly recruited without intention**. That is extremely important clinically. It means the patient may be living in a pattern where the pelvic floor behaves like a chronic guarding muscle group, much like jaw clenching or shoulder tension in other stress-related conditions. In chronic pelvic pain, this involuntary clenching can contribute to ischemic discomfort, trigger-point formation, outlet dysfunction, and referred pain into the perineum, rectum, penis, or prostate region.

This answer becomes especially meaningful if it coexists with **breath holding during stress, chest-dominant breathing, difficulty relaxing, urinary hesitancy, post-ejaculatory pain, or constipation-related pain**. In that pattern, the pelvic floor is acting as part of a **whole-body guarding strategy**, not merely as a local painful tissue.

Sensation of muscles pulling upward

A sensation of the muscles “pulling upward” is very characteristic of a pelvic floor that is being held in an **elevated, shortened, over-recruited state**. Patients often describe this as an internal lifting, gripping, or drawing-in sensation. Clinically, this strongly supports a hypertonic pelvic floor phenotype and may be particularly relevant when the patient also reports stress-triggered worsening, abdominal bracing, or difficulty breathing into the abdomen.

This upward-pulling sensation is also important because it helps distinguish simple pain from **dynamic dysfunctional tension**. It suggests that the patient can perceive the pelvic floor as an active mechanical force, not merely a passive pain location. That often has major treatment implications, because it increases the likelihood that relaxation-based physiotherapy and breathing retraining will matter clinically.

Difficulty relaxing pelvic floor muscles

This is one of the most important options in the entire section. Difficulty relaxing the pelvic floor strongly suggests that the patient’s problem is not just intermittent spasm but **impaired down-regulation** of the pelvic system. In practical terms, this means the pelvic floor may stay partially “on” even when it no longer needs to contract. That alone can maintain chronic pain, urinary dysfunction, bowel dysfunction, and post-ejaculatory discomfort.

The EAU management guidance specifically states that for patients with pelvic floor dysfunction it is very helpful to learn to relax the muscles when the pain starts, and that relaxation may interrupt the **pain–spasm–pain cycle**. This means that a positive answer to this item has both diagnostic and therapeutic value. If the patient also later reports benefit from **pelvic floor muscle relaxation**

training, pelvic floor physiotherapy, breathing exercises, meditation/yoga, or Wise-Anderson-type approaches, then this item becomes a central organizing clue for the case.

Pain when contracting the muscles

Pain on contraction suggests that the pelvic floor is not only overactive but also **painful when recruited**, which is highly consistent with myalgia, trigger-point sensitivity, or severe irritation of the muscular–fascial system. It indicates that the muscles are participating in the pain itself, not just reacting secondarily.

Clinically, this becomes especially important if the patient later reports **pain during ejaculation, pain with increased abdominal pressure, pain during bowel movements, or worsening with lifting/coughing**, because all of those situations recruit the pelvic floor. When contraction is painful, such activities can act like repeated micro-provocation tests for the same dysfunctional muscle system.

Pain when stretching the muscles

Pain with stretching suggests that the pelvic floor and surrounding myofascial structures may be **shortened, irritated, or trigger-point active**, so that lengthening the tissues provokes discomfort. This may occur in men whose pelvic floor has been chronically over-recruited for months or years and no longer tolerates normal movement or decompression comfortably.

This item is particularly relevant when paired with **hip tightness, lower-back stiffness, pain with hip movement, sitting intolerance, and constipation-related symptoms**, because it supports a broader **lumbopelvic myofascial phenotype** rather than a purely focal pelvic complaint. It may also help explain why some patients initially worsen with stretching-based therapies before improving, or why careful, graded treatment is needed.

Pain associated with constipation

This answer strongly supports a clinically important **bowel–pelvic floor overlap**. Constipation can increase rectal pressure, prolong straining, trigger anal sphincter overactivity, and increase guarding in the pelvic floor. When pelvic pain is clearly associated with constipation, the evaluator should think not only about bowel function but also about the pelvic floor's role in **defecatory dysfunction and pressure sensitivity**.

This item becomes especially meaningful when paired with **difficulty passing stool, rectal fullness, lower abdominal pressure, abdominal-breathing difficulty, or stress-related lower abdominal cramps**. In that setting, constipation is not simply a separate symptom; it may be one of the mechanisms that repeatedly loads and destabilizes the pelvic system.

Difficulty passing stool

Difficulty passing stool is one of the strongest clues to a **non-relaxing pelvic floor / defecatory dysfunction phenotype**. In chronic pelvic pain, bowel emptying may be impaired not because of primary bowel disease alone, but because the pelvic floor fails to relax or even contracts paradoxically during attempted evacuation.

This symptom is particularly important because it directly links the pelvic pain syndrome to **functional outlet dysfunction**, similar in concept to some urinary symptoms such as hesitancy or incomplete emptying. If a patient has both urinary and bowel emptying difficulty, pelvic floor discoordination becomes a much more plausible unifying explanation.

Feeling of fullness in the rectum

Rectal fullness is often described by patients as a “ball,” “pressure,” “something there,” or a persistent sense that the rectum is not relaxed or not empty. In chronic pelvic pain, this is one of the most characteristic sensations of a **deep pelvic floor / anorectal tension phenotype**. It can reflect chronic levator ani tension, outlet guarding, incomplete relaxation, constipation-linked pressure, or referred pelvic floor pain.

This answer is especially meaningful when it coexists with **anal pain, constipation-related pain, pain with sitting, and difficulty relaxing the pelvic floor**, because together those items create a highly coherent **anorectal pelvic floor overactivity pattern**.

Episodes of pelvic muscle spasm

Episodes of pelvic muscle spasm strongly support a muscular phenotype in which symptoms are not only chronic but may also become **episodically sharply worse through transient over-contraction**. Spasm implies a more active contractile event than generalized tension alone and can account for sudden flares, post-trigger worsening, or pain after ejaculation, sitting, or bowel strain.

This answer becomes particularly important if later responses show **cyclical flares, stress-triggered worsening, pain during ejaculation, or worsening with increased abdominal pressure**, because those patterns are all compatible with a pelvic system that periodically enters overt spasm. The EAU guideline’s emphasis on relaxation and physical treatment is highly relevant here because patients with spasm often benefit most when therapy addresses both muscle overactivity and its neural/autonomic drivers.

Musculoskeletal Factors: Hip tightness

Hip tightness suggests that the pelvis is operating inside a **larger musculoskeletal chain problem** rather than in isolation. Tight hips can alter pelvic tilt, load transfer, groin tension, and pelvic floor recruitment. In many men with chronic pelvic pain, hip stiffness is not just a coincidental finding; it may be one of the reasons the pelvis remains mechanically overloaded.

This item becomes more important when paired with **groin pain, inner thigh pain, pain with hip movement, sitting cross-legged intolerance, walking-related worsening, or lower-back stiffness**. In that setting, the pelvic pain phenotype often extends into a broader **hip–pelvis–pelvic floor biomechanical network**.

Lower back stiffness

Lower-back stiffness supports the idea that the pelvis and spine are functioning as a **linked lumbopelvic pain unit**. In chronic pelvic pain, lumbar stiffness may contribute to altered pelvic mechanics, poor pressure handling, and compensatory pelvic floor recruitment.

Its importance increases when combined with **sacral pain, pain with bending, heavy lifting, walking sensitivity, or thigh/leg symptoms**, because then the case becomes stronger for a **lumbopelvic musculoskeletal contribution** rather than a strictly pelvic-organ explanation.

History of pelvic or spinal injury

A history of pelvic or spinal injury suggests that the pain system may have been shaped by a prior **structural or nociceptive insult**, even if the current syndrome is now more complex. Such injuries

can leave behind altered movement patterns, persistent guarding, neural irritability, or chronic tissue sensitivity.

Clinically, this item is important because it reminds the evaluator that a chronic pelvic floor phenotype may coexist with a **post-traumatic lumbopelvic phenotype**. If the patient also reports lower-back stiffness, sacral pain, pain radiating along nerves, or hip dysfunction, the injury history becomes especially relevant.

Pain with hip movement

Pain with hip movement further strengthens the interpretation that the pelvis is part of a **larger movement dysfunction pattern**. It suggests that the pain system is sensitive not only to static tension but also to **dynamic hip–pelvis interaction**, which may implicate myofascial restrictions, groin/adductor tension, hip pathology, or lumbopelvic compensation.

When this answer appears alongside **hip tightness, groin pain, inner thigh pain, walking sensitivity, or lower-back stiffness**, it often supports a **musculoskeletal phenotype that is feeding into pelvic floor symptoms**, rather than a syndrome limited to the prostate, bladder, or urethra.

Summary of Point 14

Point 14 is the questionnaire's **pelvic floor and lumbopelvic musculoskeletal phenotype section**. Its purpose is to determine whether the patient's pain is being generated or maintained by **pelvic floor overactivity, poor relaxation, painful contraction, bowel-linked pelvic dysfunction, trigger-point behaviour, and broader hip–spine–pelvis mechanics**. The strongest indicators of a pelvic floor myalgia/overactivity phenotype are **perineal tension, involuntary clenching, upward pulling sensations, difficulty relaxing, pain with contraction, rectal fullness, constipation-related pain, and pelvic muscle spasm**. The additional hip and lower-back items extend the interpretation into a **wider biomechanical pelvic pain network**. In professional use, Point 14 is critical because it often identifies the pelvis not just as the place where pain is felt, but as the **active mechanical engine** of the syndrome.

Point 15. Neurological Symptoms

(select all that apply)

Purpose of Point 15

Point 15 is designed to identify whether the chronic pelvic pain syndrome includes a meaningful **neuropathic, centrally sensitized, autonomic, or broader pain-amplification component**. This is a crucial section because many men with CPPS do not have pain that is purely inflammatory or purely muscular. Instead, their pain may involve **abnormal sensory processing, nerve irritation, pain spread beyond the original site, exaggerated responses to normal stimuli, persistent pain after triggers end, or systemic autonomic dysregulation**. Modern chronic pelvic pain guidelines explicitly emphasize the importance of understanding both **peripheral and central pain mechanisms** in these patients.

This section is also important because it helps distinguish between pain that is primarily **generated by local pelvic tissues** and pain that is increasingly being **interpreted and amplified by the nervous system itself**. That distinction is fundamental. A man may begin with a local trigger such

as infection, pelvic floor tension, sexual overloading, or sitting compression, but over time the syndrome may acquire features of **neuropathic pain or central sensitization**, at which point symptoms spread, become disproportionate, persist after triggers, and no longer map neatly onto one local structure.

Another reason this section is important is that it may explain why **some patients respond better** to **neuromodulators, nerve blocks, intravenous or topical lidocaine/ketamine** or broader pain management strategies than to prostate-specific therapies alone. In extreme neuropathic pain, **nerve resetting** through local anesthesia (infiltration), epidural anesthesia or spinal anesthesia (intradural) is sometimes requiredIt can also help explain why some men appear to have a much higher symptom burden than would be expected from routine imaging or basic examination. That does not make the pain less real. Rather, it suggests that the pain phenotype includes an important **nervous-system component**.

Tingling in the pelvis

Tingling in the pelvis is one of the classic features of a **sensory-neural phenotype**. It suggests that the pain is not being experienced purely as pressure or aching, but includes altered sensory transmission. In a pelvic context, tingling may reflect **pudendal irritation, local sensory overactivity, referred neural symptoms, or a sensitized pelvic sensory field**.

This symptom becomes especially meaningful when combined with **electric-shock sensations, neuropathic burning, hypersensitivity of the perineum or penis, radiating pain, or sitting-related spread into the thighs or buttocks**. In that context, tingling is not a minor symptom; it is an indicator that the pain system includes a significant **neural signalling component**.

Electric shock sensations

Electric-shock sensations are one of the strongest indicators of a **neuropathic pain phenotype**. Patients who use this descriptor are often experiencing pain through a sensory mechanism that behaves more like **nerve irritation or sensitized nerve firing** than simple deep tissue discomfort. In the pelvis, this raises the importance of the **pudendal nerve, sacral pathways, or other sensitized neural routes**.

This answer becomes highly significant when paired with **pain radiating along nerves, allodynia, numbness, tingling, suspected pudendal neuralgia, or sitting-triggered leg and buttock sensory changes**. In such cases, the pain phenotype is strongly compatible with **pelvic neuropathic pain or mixed mechanical–neuropathic pain**.

Neuropathic burning pain

Neuropathic burning pain is another very high-value sensory descriptor. Burning pain in chronic pelvic pain often indicates that the patient is not only experiencing discomfort from tissues but through a **sensitized neural interface**. This may involve the pudendal territory, urethral/genital sensory pathways, post-inflammatory nerve irritability, or broader central pain amplification.

Burning pain becomes especially important if the patient also reports **glans pain, shaft pain, urethral burning, allodynia, light-touch pain, or bladder hypersensitivity**. In some men, burning also overlaps with **histamine-sensitive bladder or urethral pain**, which is why later benefit from **antihistamines, hydroxyzine, H1 or H2 blockers** can become clinically relevant. AUA guidance includes bladder-pain overlap and multisystem chronic pelvic pain phenotyping, which is consistent with this interpretation.

Numbness

Numbness suggests a stronger sensory disturbance than pain quality alone. It may indicate **altered nerve conduction, nerve compression, sensory pathway dysfunction, or severe sensory gain disruption**. In the pelvis, numbness is especially important when it affects the perineum, penis, thighs, buttocks, or legs during sitting or pelvic loading.

This answer becomes more meaningful in combination with **tingling, electric-shock sensations, radiating pain, suspected pudendal neuralgia, suspected sacral nerve irritation, or sitting-related sensory changes**. In those combinations, numbness strongly supports a **neuromechanical or lumbosacral–pelvic neural phenotype**.

Hypersensitivity of the perineum or penis

Hypersensitivity of the perineum or penis indicates that the genital–perineal sensory system is functioning at a **lower threshold**, meaning that ordinary touch, pressure, arousal, or friction may be experienced as unusually intense or uncomfortable. This may reflect **peripheral sensitization, pudendal-territory hypersensitivity, genital sensory amplification, or allodynia-like mechanisms**.

This answer is especially important if it appears together with **burning, prickling, light-touch pain, penile hypersensitivity causing rapid ejaculation, tight-clothing sensitivity, or post-ejaculatory urethral burning**. In that setting, the patient is likely expressing a **genital sensory amplification phenotype**, which can affect pain, sexuality, and pelvic tension simultaneously.

Pain radiating along nerves

Radiating pain suggests that the symptom is following a **neural pathway**, rather than remaining fixed in one local deep structure. This can be highly relevant in differentiating true deep pelvic-organ pain from pain that is spreading through **pudendal, sacral, obturator, or lumbosacral neural routes**.

If this answer coexists with **electric sensations, numbness, sacral suspicion, thigh/buttock/leg sensory symptoms, or a history of spinal injury**, the probability of a meaningful **neuropathic or referred neural phenotype** increases substantially.

Suspected pudendal neuralgia

This item identifies that the patient recognizes the possibility of a **pudendal distribution pain syndrome**, especially if the pattern includes perineal/genital/anal pain worsened by sitting. In itself, this is not a formal diagnosis, but it is clinically useful because patients often only endorse it when their symptoms closely fit a pudendal pattern.

This answer becomes especially meaningful when combined with **pain in the pudendal territory, sitting-related worsening, hypersensitivity of the perineum or penis, electric-shock sensations, or relief with offloading**. In such a pattern, pudendal neuralgia becomes a clinically important interpretive framework, even if the final phenotype remains mixed with pelvic floor overactivity or myofascial compression.

Suspected sacral nerve irritation

Suspected sacral nerve irritation broadens the neurological interpretation beyond the pudendal territory and points toward **posterior pelvic neural pathways, sacral roots, or lumbosacral**

contribution. This may be especially relevant in men whose pain includes **low-back pain, sacral pain, leg symptoms, numbness, or autonomic features**.

This item becomes particularly important if it coexists with **pain radiating along nerves, sitting-related leg symptoms, spinal injury history, or heat/cold autonomic intolerance**. In such a case, the chronic pelvic pain syndrome may be interacting with a broader **sacral or lumbosacral neural phenotype** rather than remaining purely perineal.

Hypersensitivity to cold

Cold hypersensitivity suggests that the nervous system or local tissues respond abnormally strongly to **cold exposure**, which may be a sign of **neuropathic irritability, sensory amplification, autonomic dysregulation, or even mast-cell-linked reactivity in selected patients**.

This symptom becomes more meaningful when paired with **cold exposure as a trigger, burning pain, tingling, autonomic symptoms, or skin reactions to cold elsewhere in the survey**. In such cases, cold hypersensitivity supports a **sensory-autonomic reactivity phenotype** rather than a purely deep structural pain pattern.

Pain feels disproportionate to physical findings

This is one of the most important central-sensitization clues in the whole questionnaire. It suggests that the patient experiences the pain as **stronger, broader, or more disabling than would be predicted by routine imaging, examination, or laboratory findings alone**. This does not mean the pain is “not real.” It means the nervous system may be amplifying the pain signal beyond the apparent tissue burden.

In modern chronic pelvic pain medicine, this is highly relevant because central sensitization is increasingly recognized as part of some chronic pelvic pain phenotypes. A positive answer to this item becomes especially meaningful when combined with **pain spread, allodynia, persistence after stimulus removal, migraine, fibromyalgia, sleep problems, or high symptom monitoring**.

Pain spreads beyond original location

Spread of pain beyond the original site strongly supports the possibility that the syndrome is no longer purely local. Instead, it suggests **sensory expansion, referred pain evolution, or broader central pain amplification**. In chronic pelvic pain, this is often a marker that the nervous system is increasingly involved in shaping the symptom map.

This item becomes particularly important if the patient later reports **migraine, fibromyalgia, allodynia, pain disproportionate to findings, or broader body sensitivity**, because together those answers support a **generalized pain amplification phenotype** rather than isolated pelvic disease.

Light touch causes pain (allodynia)

Allodynia is one of the most important markers of a **sensitized nervous system**. If light touch causes pain, then the threshold for perceiving harmless input as painful has been significantly lowered. In the pelvis, this may manifest as pain from clothing, light genital contact, seat pressure, or superficial touch.

Clinically, this answer is especially meaningful when it coexists with **perineal or penile hypersensitivity, burning, prickling, tight-clothing aggravation, or pressure reactivity**. It

strongly supports a **sensory amplification / neuropathic phenotype**, and often indicates that treatment should include pain-modulation strategies rather than only organ-targeted therapies.

Pain persists after stimulus is removed

Pain that persists after the provoking stimulus has ended is another strong sign that the pain system has become **self-sustaining or slow to switch off**. This is highly relevant in chronic pelvic pain because many patients initially have trigger-linked symptoms that later become harder to terminate even after the trigger is gone.

This item becomes especially important in combination with **cyclical flares, post-ejaculatory delayed pain, post-sitting pain, allodynia, or pain disproportionate to findings**, because those patterns suggest impaired recovery and **persistent post-trigger sensitization**.

Heart palpitations

Heart palpitations introduce an **autonomic nervous system dimension**. In chronic pelvic pain, palpitations may signal that the syndrome is not confined to the pelvis but is occurring in a person with broader dysautonomia, sympathetic overactivation, or stress-reactive physiology.

This answer becomes more meaningful if paired with **breath holding during stress, chest-dominant breathing, dizziness on standing, cold hands/feet, heat intolerance, anxiety, or poor sleep**. In that context, pelvic pain may be embedded within a broader **autonomic overactivation phenotype**.

Cold hands/feet

Cold hands and feet often suggest a state of **autonomic vasoconstrictive dominance or dysregulation**. While not specific to pelvic pain, this symptom becomes relevant when interpreted alongside pelvic symptoms because it suggests that the patient's body may be maintaining a broader **stress-linked or dysautonomic physiological pattern**.

When combined with **palpitations, breath-holding, anxiety, dizziness, or cold hypersensitivity**, this item strengthens the case for a **neuro-autonomic phenotype** rather than a purely local pelvic disorder.

Excessive sweating or lack of sweating

Abnormal sweating suggests **autonomic dysfunction or sensory-autonomic dysregulation**. Either excessive sweating or reduced sweating may indicate that the sympathetic system is not regulating the body normally.

In chronic pelvic pain, this symptom becomes especially relevant when paired with **palpitations, dizziness, cold hands/feet, heat intolerance, or strong stress sensitivity**, because it supports the idea that the syndrome is being maintained in part by **autonomic instability**, not just local tissue pain.

Dizziness when standing

Dizziness on standing raises the possibility of **orthostatic/autonomic dysregulation**, which may be relevant in a subset of patients with chronic pain, central sensitization, or broader autonomic imbalance.

This answer becomes more meaningful when it coexists with **palpitations, cold extremities, sweating abnormalities, or strong stress-related physiological symptoms**, because then the pelvic pain may be embedded in a wider **dysautonomic phenotype**.

Heat intolerance

Heat intolerance, like cold hypersensitivity, may indicate **autonomic dysregulation, vascular instability, sensory amplification, or mast-cell-linked symptom amplification** in selected cases.

It becomes especially important when paired with **heat exposure worsening pain, palpitations, sweating abnormalities, flushing, or histamine-related symptoms elsewhere in the survey**. In such a patient, the nervous system and vascular/immune systems may be interacting strongly.

Migraine

Migraine is clinically important because it is often associated with **central sensory amplification, autonomic dysregulation, and a sensitized nervous system**. In a chronic pelvic pain context, migraine does not explain pelvic pain by itself, but it increases the plausibility of a **broader pain-amplification phenotype**.

If the patient also reports **allodynia, pain spread, disproportionate pain, fibromyalgia, sensory hypersensitivity, or strong stress sensitivity**, migraine becomes a powerful supporting clue that the pain system may be globally amplified rather than only locally irritated.

Fibromyalgia

Fibromyalgia is another major clue to a **central sensitization / generalized pain processing phenotype**. In men with chronic pelvic pain, its presence strongly suggests that pelvic symptoms may be part of a broader system of pain amplification rather than a purely local pelvic disease.

If fibromyalgia coexists with **pain spread, allodynia, migraine, sleep problems, disproportionate pain, and multiple symptom domains**, then the pelvic pain should be interpreted inside a **centralized pain framework** as well as a local pelvic one.

Teeth Clenching During the Day

Daytime teeth clenching reflects increased **baseline muscle tension** and heightened **central nervous system activity**. In the context of **CPPS (Chronic Pelvic Pain Syndrome)**, it suggests a generalized pattern of **muscular overactivation** and **stress-related guarding**. This is commonly associated with **central sensitization**, where the nervous system maintains a persistent state of **increased excitability**.

Waking Up with a Tense or Painful Jaw

Waking with **jaw tension or pain** indicates **nocturnal bruxism** and sustained **muscle contraction during sleep**. In **CPPS patients**, this may reflect a chronic **upregulation of the nervous system** that persists even during rest. It is often linked to **central sensitization** and impaired **downregulation of muscle tone**.

Pain in the Temporomandibular Joint (TMJ) Area

Pain in the **temporomandibular joint (TMJ)** region may indicate dysfunction within the **craniofacial musculoskeletal system**. In **CPPS**, TMJ pain can be part of a broader pattern of

widespread pain and **muscle tension**, supporting the presence of **central sensitization** and altered **pain processing**.

Temporal Pain / Tension Headaches

Temporal headaches or **tension-type headaches** are commonly associated with increased **muscle tone** and **stress-related mechanisms**. In individuals with **CPPS**, these headaches may reflect shared pathways of **pain amplification** and **central sensitization**, indicating that abnormalities in **pain processing** are not limited to the pelvic region.

A Feeling of a "Stiff Face"

A subjective sensation of **facial stiffness** suggests increased **muscle tone** or altered **sensory perception** in the **craniofacial region**. In the setting of **CPPS**, this may represent a manifestation of **central sensitization**, where normally non-painful sensations are perceived as **abnormal or uncomfortable** due to heightened **neural sensitivity**.

Groin Pain

Groin pain is a common symptom in **CPPS** and may arise from **muscular**, **fascial**, or **neural sources**. It can also represent **referred pain** from **pelvic floor dysfunction**. In cases of **central sensitization**, the pain may persist without clear **structural pathology** and may become **amplified** or more **diffuse**.

Inner Thigh Pain

Pain in the **inner thigh** often reflects involvement of the **adductor muscles** or shared **nerve pathways** with the pelvic region. In **CPPS**, this may indicate **myofascial dysfunction** or **referred pain**. **Central sensitization** can contribute to increased **pain intensity** and **spread beyond the original site**.

Tightness or Pain in the Buttocks

Tightness or pain in the buttocks is frequently associated with **pelvic floor dysfunction** and involvement of muscles such as the **gluteals** or **piriformis**. In **CPPS**, this symptom may reflect both **local muscular issues** and **referred pain patterns**. **Central sensitization** may further **amplify these sensations** and contribute to their **chronic persistence**.

Summary of Point 15

Point 15 is the questionnaire's **neuropathic, autonomic, and central sensitization section**. Its purpose is to determine whether the chronic pelvic pain syndrome includes a meaningful **neural-pain layer**, a **broader autonomic dysregulation layer**, or a **pain-amplification / central sensitization layer**. **Tingling, electric-shock sensations, neuropathic burning, numbness, hypersensitivity, radiating pain, and suspected pudendal or sacral irritation** all support a **neuropathic or neuromechanical phenotype**. **Pain disproportionate to findings, pain spread, allodynia, and pain persisting after the trigger ends** strongly support a **sensitized pain**

phenotype. **Palpitations, cold extremities, sweating abnormalities, dizziness on standing, and heat intolerance** point toward **autonomic involvement**. **Migraine and fibromyalgia** further strengthen the possibility that the pelvic pain exists within a broader system of sensory amplification. In professional interpretation, Point 15 is essential because it identifies whether the patient's pain is still mostly local and tissue-linked, or whether it has become substantially shaped by the **nervous system's own processing and regulation**

Point 16. Neurodevelopmental traits and psychological factors. Do you experience:

(select all that apply)

Purpose of Point 16

Point 16 is designed to identify whether the patient's chronic pelvic pain exists within a broader pattern of **attention regulation differences, sensory over-responsivity, stress-reactivity, compulsive monitoring, fear-based behavioural adaptation, and chronic autonomic overactivation**. This point is not included to suggest that pelvic pain is "imagined," "merely psychological," or secondary in importance to physical symptoms. Its purpose is the opposite: to determine whether the patient's nervous system is functioning in a way that may **increase vulnerability to pelvic floor guarding, symptom amplification, trigger sensitivity, and chronicity**. That is consistent with modern chronic pain science, which recognizes that attention, expectation, arousal, sensory processing, and trauma can all influence the persistence and intensity of pain without making that pain any less real.

In practical terms, Point 16 helps determine whether the patient has a phenotype in which the pelvis is not only exposed to local triggers such as sitting, ejaculation, bowel pressure, or bladder filling, but is also embedded in a nervous system that is **highly vigilant, difficult to down-regulate, prone to symptom monitoring, vulnerable to stress-triggered muscular recruitment, or unusually sensitive to sensory input**. This is especially important in chronic pelvic pain because the pelvic floor is highly responsive to emotional state, breathing pattern, body vigilance, and perceived threat. The AUA guideline emphasizes multimodal assessment, and the EAU guideline highlights central sensitization and psychosocial factors as relevant parts of chronic pelvic pain evaluation.

A second reason this point matters is that it helps distinguish between **primary symptom generators** and **maintenance mechanisms**. A man may originally develop pain after infection, sexual overloading, prolonged sitting, or bowel dysfunction, but if he later develops constant symptom checking, fear of worsening, inability to relax, poor sleep, sensory hypersensitivity, and marked stress reactivity, then the condition may now be maintained partly by a **pain–attention–arousal loop**. Point 16 is therefore a map of the **regulatory environment** in which the pelvic pain lives.

Difficulty maintaining attention on tasks

This item suggests possible problems with **sustained attention and cognitive persistence**. In the context of chronic pelvic pain, it becomes relevant because patients who struggle to hold steady

attention often also struggle to maintain consistent self-regulation, pacing, and body-state monitoring without becoming overwhelmed. This does not mean attentional difficulty causes pelvic pain directly. Rather, it may indicate a nervous system that is more vulnerable to **fragmented regulation, variable stress processing, and unstable symptom focus**. A 2025 systematic review found that individuals with ADHD show significantly greater atypical sensory processing, including sensory sensitivity and sensory avoidance, compared with controls.

Clinically, this item becomes more meaningful if it coexists with **easily distracted by external stimuli, frequent mind-wandering, procrastination, difficulty organizing and planning, racing thoughts, and increased sensory sensitivity**. In that pattern, chronic pelvic pain may be occurring in someone whose attentional system is already taxed, which may affect how strongly he perceives triggers, how consistently he follows therapeutic routines, and how easily he becomes physiologically overloaded.

Easily distracted by external stimuli

This suggests heightened sensitivity to **environmental input** and reduced filtering of irrelevant stimuli. In chronic pelvic pain, this matters because the same nervous system that struggles to ignore noise, movement, pressure, or visual clutter may also struggle to ignore **internal body sensations**. Such patients may react more strongly to sitting pressure, clothing, bladder filling, bowel signals, or genital sensations. Research on ADHD and sensory processing supports the idea that attentional dysregulation often coexists with atypical sensory filtering.

This item becomes especially important when paired with **increased sensitivity to sensory stimuli, discomfort with certain textures, hypersensitivity to stimuli, and tendency to overanalyze bodily sensations**. In that cluster, pelvic pain may be shaped not only by peripheral pelvic events but by a brain/body system that treats incoming stimuli as unusually salient.

Frequent mind-wandering

Frequent mind-wandering may reflect attentional instability, difficulty sustaining directed mental effort, or a nervous system that readily shifts between internal themes. In chronic pain, this can matter in two apparently opposite ways: it may reduce focus on pain at times, but it may also promote **repetitive shifting back toward symptoms, worries, health interpretations, and future predictions**.

Its meaning becomes more clinically relevant when it appears with **overanalyzing symptoms, catastrophizing, frequent symptom monitoring, and feeling that attention increases pain intensity**. In that situation, mind-wandering may not be neutral distraction; it may become a route through which the mind repeatedly returns to pain-related cues.

Difficulty completing tasks

This item may indicate impaired executive persistence, fatigue, cognitive overload, or reduced tolerance for sustained effort. In chronic pelvic pain, difficulty completing tasks often gains clinical importance when symptoms themselves interfere with concentration, sitting tolerance, sleep, and emotional regulation. It may also reflect a broader attentional phenotype when it occurs alongside procrastination and distractibility.

This answer matters because patients who struggle to complete tasks may also struggle to implement **graded exercise, breathing practice, pelvic floor relaxation work, elimination diets,**

symptom diaries, or consistent therapy attendance. It therefore has implications not only for pain interpretation but for how treatment plans should be designed.

Tendency to procrastinate

Procrastination may reflect executive dysregulation, avoidance under stress, task paralysis, or fear-based postponement. In chronic pelvic pain, it may become especially important if the patient also avoids activity because he fears symptom worsening. In that context, procrastination may not be a generic personality issue but part of a **pain-avoidance cycle**.

If paired with **fear of symptoms worsening, avoiding activity, depression, anxiety, and difficulty completing tasks**, it may signal that chronic pain has begun to reshape behaviour around avoidance and perceived lack of control.

Feeling mentally anxious or "on edge"

This is one of the most directly relevant psychophysiological items. Feeling “on edge” suggests a state of **sympathetic readiness, vigilance, and incomplete internal safety**, which can directly translate into pelvic floor tightening, breath holding, urinary urgency, and increased pain reactivity. Chronic pelvic pain guidelines recognize the importance of psychosocial and stress-related contributors in symptom persistence.

This item becomes especially important if it coexists with **difficulty relaxing, stress-worsened symptoms, body-focused worsening, palpitations, cold hands/feet, or sleep difficulty**. In that cluster, the pelvis is likely functioning within a broader **autonomic overactivation phenotype**.

Difficulty relaxing, even when there is time to rest

This is one of the strongest indicators of a **persistent overactivated regulatory state**. It suggests that rest conditions exist externally, but the body and mind fail to enter a true restorative mode. In chronic pelvic pain, that often matters because pelvic floor relaxation, down-regulation of bladder urgency, and reduction of symptom vigilance all depend on the nervous system’s ability to switch into a less defensive state.

If this item appears with **chest-dominant breathing, breath holding during stress, pelvic floor clenching, difficulty sleeping, and stress as a trigger**, it strongly supports a **psychophysiological pelvic pain phenotype**.

Impulsive behaviors, including sexual behavior

This answer suggests a pattern of **rapid action under urge**, sometimes without adequate pacing or self-regulation. In chronic pelvic pain, that becomes particularly relevant if impulsivity includes sexual behavior, because frequent or poorly regulated sexual activation may increase exposure to **ejaculatory loading, post-ejaculatory flares, or reduced recovery time** in susceptible patients.

This item becomes especially important if it coexists with **high sexual drive, tendency to seek stimulation, compulsive sexual behavior, pornography addiction, pain during ejaculation, pain after ejaculation, worsening with frequent daily ejaculation, or seminal vesicle-region pain**. In such a pattern, impulsive sexual behaviour may help explain how the patient repeatedly re-enters a symptom-triggering sexual state before the deep ejaculatory system has comfortably returned to baseline. That is a **phenotype-specific recovery issue**, not a universal statement about sexuality.

Tendency to seek stimulation (e.g.through pornography and sex)

This item points toward a nervous system that may require or pursue **high-intensity input** to feel engaged, soothed, or regulated. In the context of chronic pelvic pain, it becomes clinically important when sexual stimulation is part of that pattern, because repeated genital/pelvic activation can become a trigger in some men, especially those with **high drive, frequent ejaculation, delayed ejaculation practices, or post-ejaculatory pain**.

This answer is best interpreted as an **exposure amplifier**, not as pathology by itself. It becomes significant when it is linked to a pelvic system that already shows poor recovery after stimulation.

Difficulty organizing and planning

Difficulty organizing and planning often suggests executive dysfunction, cognitive overload, or poor tolerance of complex self-management demands. In chronic pelvic pain, that matters because treatment often requires coordinated behavioural strategies: scheduling movement breaks, pacing sexual activity, tracking food triggers, doing physiotherapy exercises, and implementing sleep or stress interventions.

This item has practical importance because patients with this trait may need more structured treatment plans and simpler routines. It does not define the pain mechanism by itself, but it can shape **how chronic pain becomes persistent through poor recoverability and inconsistent regulation**.

Excessive focus on specific topics (including symptoms or health)

This answer suggests a tendency toward **narrow, sustained fixation**, which can become highly relevant when the topic of fixation is health or symptoms. In chronic pelvic pain, sustained health focus can increase body vigilance, symptom checking, trigger anticipation, and catastrophic interpretation.

It becomes especially meaningful when paired with **frequent symptom monitoring, overanalyzing symptoms, fear of worsening, and feeling that attention increases pain intensity**. In that cluster, symptom-focused cognition may itself become part of the pain-maintaining system.

Difficulty switching attention

Difficulty switching attention suggests that once the mind is captured by something — including pain or fear of pain — it may be hard to disengage. In chronic pelvic pain, this trait is especially important because it can intensify **perseverative symptom focus**, slow emotional recovery after flares, and reduce flexibility in attention away from bodily threat signals.

If combined with **body focus worsening symptoms, symptom monitoring, catastrophizing, and repetitive thought patterns**, it strongly supports a **cognitive amplification phenotype**.

Excessive anxiety and sensitivity

This item suggests generalized **threat sensitivity**, emotional reactivity, and reduced tolerance for bodily or environmental stress. In chronic pain, such a profile often increases the likelihood that

pain will be perceived as more alarming, more difficult to regulate, and more behaviourally restricting.

When it coexists with **palpitations, sleep difficulty, symptom catastrophizing, and stress-triggered worsening**, it strengthens the interpretation of a **high-gain psychophysiological phenotype**.

Racing thoughts

Racing thoughts suggest cognitive overactivation and difficulty settling mental activity. In chronic pelvic pain, that can contribute to **sleep disturbance, bodily vigilance, poor relaxation, and persistent anticipation of symptoms**. It often indicates that the patient is not merely stressed occasionally, but may be living in a continuously activated mental state.

This answer becomes especially relevant with **difficulty relaxing, anxiety, body-focused worsening, and pain that feels stronger when attention is directed inward**.

Difficulty relaxing

Although similar to the earlier item, this broader wording captures a more generalized inability to settle, physically or mentally. In chronic pelvic pain, it often points toward a **persistent inability to downshift from guarded mode**, which can keep the pelvic floor and pain system activated.

Pornography addiction

This item matters because compulsive pornography use may increase the frequency and intensity of **sexual stimulation, prolonged arousal, edging-like patterns, repeated ejaculation, and symptom-triggering pelvic activation** in susceptible men. In pelvic pain, the problem is not pornography in the abstract, but the possibility that it drives **high-frequency stimulation with insufficient physiological recovery**.

This answer is especially relevant if it coexists with **high libido, compulsive sexual behavior, pain after ejaculation, worsening with frequent daily ejaculation, and deep seminal-vesicle or prostate-region pain**. In that pattern, it supports a **sexual-overload / poor-recovery phenotype**.

Compulsive sexual behavior

This item broadens the previous one beyond pornography to a more general pattern of **repetitive, hard-to-regulate sexual activation**. In chronic pelvic pain, it becomes highly relevant when sexual activation and ejaculation clearly worsen symptoms. It may help explain why the patient continues to provoke a pain-prone pelvic system despite recognizing the consequences.

Again, the medically careful interpretation is not that sexuality is pathological, but that **behavioural drive and tissue recovery may be poorly matched in this patient**.

Increased sensitivity to sensory stimuli (touch, sound, light)

This item points toward a generalized **sensory processing vulnerability**. In chronic pain, broad sensory sensitivity often increases the plausibility of central sensitization or generalized sensory amplification. In ADHD and related neurodivergent phenotypes, atypical sensory processing is also more common.

In pelvic pain, this answer becomes highly relevant because it suggests the patient's nervous system may already be functioning at a higher-than-usual sensory gain, making pelvic sensations more intrusive and difficult to ignore.

Discomfort with certain textures (e.g. clothes, pressure in the groin)

This is a particularly important item because it connects neurodevelopmental/sensory processing with **local pelvic symptom expression**. If the patient is uncomfortable with clothes or pressure in the groin, this may indicate **allodynia, cutaneous hypersensitivity, genital sensory amplification, or pressure-reactive pelvic tissue**.

This item becomes especially strong if paired with **tight clothing worsening pain, allodynia, hypersensitivity of the perineum or penis, and reaction to pressure**. It can represent either a **sensory-processing phenotype**, a **local pain amplification phenotype**, or both.

Anxiety when routine is disrupted

This suggests reduced tolerance for unpredictability and increased distress when the usual regulatory structure breaks down. In chronic pain, this can matter because pain itself is often unpredictable, and patients with low tolerance for routine disruption may experience flares as especially destabilizing.

Tendency to overanalyze bodily sensations

This is one of the most important cognitive-maintenance items in the questionnaire. Overanalyzing bodily sensations can increase vigilance, threat interpretation, and symptom amplification. In chronic pelvic pain, this often becomes part of a self-reinforcing loop in which normal or mildly abnormal sensations are repeatedly re-scanned, reinterpreted, and re-felt more intensely.

Preference Loneliness

A preference for loneliness may reflect sensory overload, social exhaustion, mood burden, neurodivergent preference, or chronic pain adaptation. In chronic pelvic pain, it can matter because isolation may reduce immediate overload, but it can also reduce social buffering and increase symptom-focused attention.

Feeling overwhelmed in social situations

This suggests a nervous system that becomes overloaded by social complexity, stimulation, or interpersonal demand. In chronic pain, overwhelm can translate into increased autonomic arousal and symptom reactivity.

Intense focus on specific interests

This can reflect neurodivergent attentional style. In itself it is not pathological. In chronic pelvic pain, it matters mainly if the same style of intense focus is recruited toward symptoms, bodily changes, or health concerns.

Repetitive thought patterns

Repetitive thought patterns may contribute to rumination, persistent symptom analysis, and difficulty disengaging from bodily distress. In chronic pain, repetition of pain-related thinking is associated with greater persistence and distress.

Heightened awareness of bodily sensations (interoception)

Heightened interoception means internal bodily signals are noticed strongly and often. In chronic pelvic pain, this can be a double-edged phenomenon: it may help patients identify triggers, but it can also increase the salience of bladder filling, bowel pressure, genital sensations, and pelvic floor tension.

Hypersensitivity to stimuli (light, sound)

This overlaps with the broader sensory-sensitivity items and again suggests a **high-gain sensory phenotype**, which may make pelvic sensations harder to filter out or contextualize.

I have trouble maintaining relationships with others

Both items speak to chronic interpersonal strain, possible neurodevelopmental differences, social stress, or the social consequences of chronic pain. They matter because chronic pelvic pain often becomes more severe and more isolating when it disrupts relational stability and support.

I have or have had episodes of depression

Depression is highly relevant in chronic pain because it is associated with increased pain burden, fatigue, lower motivation, sleep disturbance, and reduced recovery capacity. It should not be treated as a dismissal of symptoms, but as an important layer of the phenotype.

I have or have had anxiety disorders

This strongly supports a **psychophysiological pain-maintenance phenotype**, especially if paired with body vigilance, symptom fear, breath holding, and stress-triggered worsening. Anxiety disorders can increase anticipatory fear and pelvic floor guarding.

I have or have had obsessive-compulsive disorder (OCD)

OCD or obsessive-compulsive tendencies can be highly relevant when chronic pelvic pain becomes linked to **checking, reassurance seeking, bodily overanalysis, and intrusive symptom-related mental loops**.

Symptoms worsen during periods of psychological stress or anxiety

This is one of the strongest items in the whole section. If symptoms reliably worsen during stress or anxiety, that strongly supports a **stress-reactive pelvic phenotype** in which autonomic arousal, pelvic floor recruitment, body vigilance, or bladder urgency intensify pain.

Symptoms worsen when focusing on the body

This suggests that attention itself modulates symptoms. In chronic pain science, this is highly relevant because focusing on symptoms can increase their salience and perceived intensity. It does not mean the symptoms are imagined; it means the nervous system is **attention-sensitive**.

Symptoms improve with distraction or engagement

This is the complementary pattern. If symptoms improve when attention is drawn outward into meaningful engagement, that supports a role for **attention-based amplification** in symptom persistence.

Tendency to catastrophize symptoms

Catastrophizing is clinically important because it is associated with worse pain outcomes, greater perceived threat, increased avoidance, and stronger autonomic arousal. In chronic pelvic pain, it can turn flares into high-threat events that prolong recovery.

Fear of symptoms worsening

Fear of worsening can drive avoidance, hypervigilance, reduced activity, and more intense self-monitoring. In chronic pain, that often contributes to persistence rather than protection.

Feeling of loss of control over bodily sensations

This item captures a particularly distressing chronic pain experience: that the body feels intrusive, unpredictable, and difficult to regulate. This often strengthens symptom vigilance and perceived threat.

Frequent symptom monitoring

Frequent monitoring is a major maintenance factor in chronic pain because it repeatedly directs attention toward the body and reinforces symptom salience. In pelvic pain, this may include checking urinary patterns, genital sensations, perineal pressure, or pain fluctuations.

Avoiding activity for fear of symptoms worsening

Avoidance is clinically important because it can reduce conditioning tolerance, increase deconditioning, and reinforce fear. In pelvic pain, this may affect exercise, sexual activity, sitting, travel, and even daily tasks.

Overanalyzing symptoms

This overlaps with symptom monitoring and catastrophizing, and together these items suggest a strong **cognitive amplification loop** around the pain.

Feeling that attention increases the intensity of pain

This is one of the clearest markers that the patient subjectively recognizes **attention-modulated pain amplification**. That is highly important clinically because it suggests the pain system is sensitive not only to peripheral triggers but also to **top-down cognitive focus**.

When I'm stressed, I experience cramps/pain in my lower abdomen

This item reveals a strong **stress-to-body translation pathway**. In many patients, lower-abdominal tightening and pelvic discomfort are how stress is somatically expressed. That can be highly relevant for pelvic floor, bowel, and bladder symptoms.

Experiencing improvement after intensely focusing on something exciting

This item suggests that meaningful engagement can temporarily override pain salience. That supports a strong role for **attention, arousal allocation, and cognitive capture** in the symptom phenotype.

Difficulty sleeping

Sleep difficulty is critically important in chronic pain because poor sleep worsens pain sensitivity, autonomic instability, emotional regulation, and recovery. In pelvic pain, it often amplifies both sensory and muscular symptoms.

I am or have been a victim of violence

I have experienced post-traumatic stress disorder

These items are extremely important because trauma and PTSD are linked to chronic pain persistence, hypervigilance, sleep disturbance, catastrophizing, and dysregulated body-state processing. PTSD and chronic pain frequently co-occur, and trauma can shape how pain is perceived and maintained.

Everyday life stresses me out

This suggests high baseline stress load and low recovery capacity, both highly relevant in chronic pelvic pain.

I don't feel stressed because it's a daily occurrence for me

This item is clinically subtle and important. It may indicate that high stress has become so normalized that the patient no longer labels it consciously, even though the body may still be functioning in a chronically stressed state.

I'm not an anxious person

This answer does not rule out stress-linked pelvic pain. Some patients do not identify as anxious yet still have strong physical patterns of breath holding, pelvic guarding, and autonomic overactivation. It is therefore interpretively important but not exclusionary.

Stress motivates me and allows me to grow

This item is also subtle. It may reflect resilience, but in some patients it may also indicate chronic functioning in a **high-activation mode** that feels productive yet carries physiological cost. In

chronic pelvic pain, that matters because performance-oriented stress can still drive pelvic tension and nervous-system overactivation.

Summary of Point 16

Point 16 is the questionnaire's **neurodevelopmental, psychological, and pain-regulation section**. Its purpose is to determine whether chronic pelvic pain exists within a broader phenotype of **attention dysregulation, sensory hypersensitivity, compulsive symptom focus, fear-based avoidance, trauma-related vulnerability, autonomic arousal, and poor down-regulation**. The most clinically important clusters are:
a **neurodivergent/sensory profile** built around distractibility, sensory sensitivity, interoceptive intensity, and executive difficulty;
a **psychophysiological stress-reactive profile** built around anxiety, inability to relax, stress-linked worsening, breath-related and body-related reactivity;
and a **cognitive amplification profile** built around catastrophizing, symptom monitoring, overanalysis, and fear of worsening.
In professional interpretation, Point 16 is essential because it shows whether the pelvic pain is living inside a nervous system that is likely to **amplify, maintain, or repeatedly re-trigger it**, even when the original peripheral trigger was somewhere else.

Point 17. Immunological / MCAS-Related Symptoms. Please indicate what aggravates your pelvic floor pain symptoms:

(select all that apply)

According to the latest uploaded questionnaire, Point 17 includes: **Alcohol; Coffee; Spicy foods; Fermented foods: kefir, yogurt; Foods high in histamine: blue cheese, red wine, sauerkraut, mackerel, tuna, salami, etc.; Sour foods; Artificial sweeteners, energy drinks; Citrus fruits and tomatoes; Intense physical activity; irregular sleep; Symptoms after pressure (mast cell trigger); Allergic rhinitis; Hay fever; Asthma**.

Purpose of Point 17

Point 17 is designed to determine whether the patient's pelvic pain shows a pattern compatible with **immune reactivity, histamine intolerance, mast-cell–related symptom amplification, or a broader allergy-reactive phenotype**. This section is particularly important because chronic pelvic pain is not purely muscular, neurological, or prostate-related in every patient. A subset of men appear to have symptoms that are aggravated by **histamine-rich foods, alcohol, sleep disruption, pressure, and allergic conditions**, suggesting a possible **bladder–pelvic floor–immune interface**.

Modern urology literature on bladder pain syndrome and pelvic pain increasingly recognizes that **mast cells may play a role in symptom generation or amplification** in some patients, especially those with bladder pain, urgency, food-triggered flares, allergic symptoms, and pressure sensitivity. The EAU chronic pelvic pain management section states that mast cells may play a role in primary bladder pain syndrome and notes that histamine receptor antagonists have been used, although evidence for benefit is variable and hydroxyzine has not shown clear superiority in every study. In

other words, a histamine or mast-cell–related interpretation is clinically plausible in selected patients, but it should be used as a **phenotype clue**, not as a blanket explanation for all CPPS.

This section is also highly relevant to the later medication-response items in your survey. If a patient reports that pelvic/bladder symptoms are worsened by **histamine-rich foods, alcohol, irregular sleep, pressure, or allergic seasons**, and later also reports benefit from **antihistamines, hydroxyzine, H1 blockers, H2 blockers, quercetin, DAO supplements, luteolin, vitamin C, or a low-histamine diet**, then the immunological interpretation becomes much stronger. Likewise, if a patient reports benefit from **short steroid courses**, that may support the idea that inflammatory amplification is present in at least some flares, but steroids are not diagnostic and should not be used to overstate an immune hypothesis. In current evidence, they remain a **supportive clinical clue**, not a definitive classification tool.

Alcohol

Alcohol is one of the most important items in this section because it can act as both a **bladder irritant and a histamine-related trigger**. In selected patients, alcohol may increase flushing, urgency, bladder discomfort, pelvic pressure, and post-trigger pain flares. It may also worsen sleep quality, which in turn can amplify pain sensitivity and autonomic instability.

This answer becomes especially meaningful when combined with **bladder pain, urgency, burning, histamine-rich food sensitivity, flushing, hives, or benefit from antihistamines or low-histamine diet**. In such a pattern, alcohol is not just a “bad lifestyle factor”; it may be functioning as a real **immune-irritative or sensory trigger**.

Coffee

Coffee can aggravate symptoms through several mechanisms: **bladder stimulation, urgency amplification, autonomic activation, and sensory irritability**. In some patients it may function mainly as a bladder irritant; in others, it may worsen an already high-arousal autonomic phenotype.

This answer becomes more significant if the patient also reports **frequency, urgency, bladder pressure, pain when the bladder fills, palpitations, anxiety, and poor sleep**. In such cases, coffee may amplify both the bladder and the nervous system.

Spicy foods

Spicy foods may aggravate chronic pelvic pain through **bladder irritation, bowel irritation, sensory amplification, and in some patients histamine-like flare behaviour**. They are especially relevant in patients who experience symptoms as burning, urgency, pelvic heat, or post-meal flares.

If paired with **burning during urination, glans/urethral burning, bladder pain, or GI symptoms**, this supports a **food-triggered sensory-irritative phenotype**.

Fermented foods: kefir, yogurt

Fermented foods are especially important because they may be relevant in a **histamine-sensitive phenotype**. Not every patient reacts to them, but in those who do, they can help identify a pattern in which the immune/sensory system is unusually reactive to histamine-containing or histamine-liberating foods.

If this item is present along with **foods high in histamine, alcohol sensitivity, flushing, hives, itching, allergic rhinitis, or benefit from antihistamines**, that strengthens the interpretation of a **histamine-related pelvic pain subtype**.

Foods high in histamine: blue cheese, red wine, sauerkraut, mackerel, tuna, salami, etc.

This is one of the most direct histamine-phenotype questions in the whole survey. If the patient clearly worsens after these foods, that strongly raises the possibility of **histamine intolerance, mast-cell–related symptom amplification, or a bladder/pelvic floor pain state that is sensitive to histamine-related inputs**.

This answer becomes especially important if it clusters with **alcohol sensitivity, fermented-food sensitivity, skin flushing, hives, itching, allergic disease, and later benefit from antihistamines, quercetin, DAO, low-histamine diet, or hydroxyzine**. In that context, the survey is identifying not just "food triggers," but a possible **MCAS-like or histamine-amplified pain phenotype**.

Sour foods

Sour foods may function as **direct bladder irritants**, particularly in patients with bladder pain, urgency, and urethral burning. Their relevance may be more irritative than immunologic in some patients, although overlap is possible.

If paired with **burning urination, bladder filling pain, glans/urethral burning, and citrus/tomato sensitivity**, this item supports a **bladder-sensory irritative phenotype**.

Artificial sweeteners, energy drinks

These may aggravate symptoms through a mix of **bladder irritation, autonomic stimulation, sensory gain, and sleep disruption**. Energy drinks are particularly relevant because they combine stimulants with additives that may intensify urinary and autonomic symptoms.

This item becomes stronger when paired with **urgency, bladder pain, palpitations, anxiety, poor sleep, and frequency**.

Citrus fruits and tomatoes

This item points strongly toward a **bladder/urethral irritative phenotype**, because these foods are commonly reported as triggers in patients with bladder pain and urinary burning. In selected patients they may also overlap with histamine or sensory reactivity.

If paired with **pain when the bladder fills, burning during urination, urgency, bladder hypersensitivity, or post-ejaculatory urethral burning**, the significance becomes much greater.

Intense physical activity

This item is important because exercise can aggravate symptoms through more than one route: **mechanical overload, pressure increases, autonomic activation, heat generation, and—in some patients—mast-cell or inflammatory flare behaviour**. It therefore sits at the interface of musculoskeletal and immunological interpretation.

If this answer appears together with **heat intolerance, sweating abnormalities, muscular flares, or histamine-type symptoms**, it may support a broader **reactive phenotype**, not just simple biomechanical overload.

Irregular sleep

Irregular sleep is extremely important in both pain science and immune regulation. Poor or irregular sleep can increase **pain sensitivity, autonomic instability, inflammatory signalling, and perceived trigger intensity**. In patients with histamine-like or bladder-reactive symptoms, bad sleep may lower the threshold for flares.

This answer becomes especially meaningful if the patient also reports **difficulty sleeping, nocturia, anxiety, widespread reactivity, or flare-ups after food and stress**. In that pattern, sleep irregularity may be a major **amplifier** of the overall syndrome.

Symptoms after pressure (mast cell trigger)

This is one of the most sophisticated items in the entire questionnaire. It suggests that external pressure itself may trigger symptoms in a way that is not purely mechanical, but possibly **pressure-reactive at a mast-cell / sensory / inflammatory interface**. Mast cells are known to be activated by various physical and immune signals, and this kind of item is especially useful when pressure sensitivity seems disproportionate to simple mechanical load.

This answer becomes highly meaningful if it coexists with **tight clothing sensitivity, reaction to pressure elsewhere, bladder pain, burning, histamine-food sensitivity, flushing, or benefit from antihistamines**. In that context, pressure may be acting not only as compression but as a trigger within a **mast-cell–sensitive pelvic environment**.

Allergic rhinitis

Allergic rhinitis supports an **atopic or immune-reactive background**. By itself it does not prove that pelvic pain is histamine-mediated, but it increases the plausibility of an immune-sensitive phenotype, especially when combined with food-triggered flares and other allergic symptoms.

Hay fever

Hay fever serves a similar role. It suggests that the patient's immune system may already be prone to **seasonal allergic over-reactivity**, which becomes relevant when the pelvic pain pattern also looks histamine-sensitive or antihistamine-responsive.

Asthma

Asthma is particularly important because it suggests a more systemic **allergic / inflammatory / mast-cell-relevant background**, not just local nasal allergy. In the right context, it can strengthen the interpretation of a broader immune-reactive phenotype.

This answer is most meaningful when paired with **allergic rhinitis, hay fever, histamine-food sensitivity, flushing, hives, itching, or benefit from antihistamines**.

Summary of Point 17

Point 17 is the questionnaire's **immunological, histamine-related, and MCAS-like aggravation section**. Its purpose is to determine whether the patient's pelvic pain behaves like a syndrome that is

aggravated by **histamine-rich foods, bladder irritants, allergic disease, pressure-reactivity, irregular sleep, or broader immune reactivity**. The strongest clues to a histamine/MCAS-like subtype are **worsening after alcohol, fermented foods, high-histamine foods, pressure, and irregular sleep**, especially when those occur together with **allergic rhinitis, hay fever, asthma**, and later benefit from **antihistamines, hydroxyzine, H1/H2 blockers, low-histamine diet, quercetin, DAO, or related interventions**. Short steroid responsiveness, if present elsewhere in the survey, may add weight to an **inflammatory amplification layer**, but it should be interpreted as supportive rather than definitive. In professional interpretation, Point 17 is essential because it identifies a subset of men whose chronic pelvic pain may be shaped not only by muscles, nerves, or sexual triggers, but also by a **bladder–pelvic floor–immune / histamine-reactive phenotype.**

Point 18. I often experience this:

(select all that apply)

Purpose of Point 18

Point 18 is designed to identify whether the patient has a background pattern suggestive of **ongoing immune reactivity, atopy, histamine sensitivity, mast-cell–related symptoms, or non-specific mediator-driven hypersensitivity**. Point 17 asks what **aggravates** pelvic symptoms; Point 18 asks what the patient **commonly experiences as part of his baseline physiology**. That distinction is crucial. A patient may report that alcohol or high-histamine foods aggravate symptoms in Point 17, but Point 18 shows whether that happens in the context of a wider body-wide pattern of **allergic, histaminergic, cutaneous, or pressure-reactive symptoms**.

This section is particularly important because chronic pelvic pain that overlaps with **bladder pain, urethral burning, pressure sensitivity, food-triggered flares, or irregular-sleep flares** may in some patients be occurring inside a broader **MCAS-like / histamine-amplified phenotype**. Current EAU guidance on chronic pelvic pain notes that mast cells may play a role in bladder pain syndrome and discusses histamine receptor antagonists among treatment approaches, although evidence remains mixed and should not be overstated.

The purpose of Point 18 is therefore not to diagnose mast cell activation syndrome from a questionnaire alone. Rather, it helps determine whether the patient's pelvic pain may exist within a **broader immune-reactive background**. This is especially relevant if the patient later reports benefit from **antihistamines, hydroxyzine, H1/H2 blockers, low-histamine diet, quercetin, DAO supplements, luteolin, or vitamin C**, because those later treatment-response items gain much more interpretive value when they occur in a patient who already shows allergic, flushing, itching, dermographic, or pressure-reactive features. Likewise, if a patient reports partial benefit from **short steroid courses**, that may support the idea of inflammatory amplification in at least some flares, but steroids should still be interpreted as a **supportive clue** rather than proof of one immune mechanism.

Seasonal allergies manifesting as nasal or sinus congestion

Seasonal allergy symptoms suggest an **atopic background**, meaning the immune system is already prone to exaggerated responses to environmental triggers. In the context of chronic pelvic pain, this does not prove that pelvic pain is allergy-driven, but it increases the plausibility that the patient's body is generally **reactive to mediator release**, especially if pelvic or bladder symptoms worsen during periods of higher allergen exposure or when other histamine-related triggers are also present.

This answer becomes much more meaningful when combined with **alcohol sensitivity, fermented-food sensitivity, histamine-rich food sensitivity, itching, hives, dermographism, or benefit from antihistamines**. In that cluster, the patient's pelvic symptoms may be embedded in a broader **immune-reactive phenotype**, rather than existing as an isolated urological complaint.

Food intolerances

Food intolerances are highly relevant because they often strengthen the connection between the **gut, immune system, and pelvic pain**. Some patients use the term loosely, while others describe very clear and repeatable patterns. In chronic pelvic pain, this answer becomes especially important because it may indicate that symptoms are modulated by **dietary chemistry, intestinal permeability, fermentation, histamine handling, or broader gut–immune irritability**.

This item becomes especially meaningful if paired with **gluten sensitivity, dairy sensitivity, FODMAP sensitivity, bloating, IBS, nausea during flare-ups, or worsening of pelvic symptoms after meals**. In such cases, "food intolerances" may help identify a **gut–immune–pelvic phenotype** rather than a purely prostate or bladder phenotype. Recent work continues to support links between CP/CPPS and gut microbiome alterations, although this remains an evolving area rather than a settled diagnostic pathway.

Skin flushing

Skin flushing is a particularly important mediator-related symptom. It often suggests **histamine release or broader vasoactive mediator activity**, especially when it occurs episodically and in association with alcohol, specific foods, heat, stress, or exercise. In the context of pelvic pain, flushing can be a very useful clue that the patient's pain may not be only muscular or neurological, but may also be embedded in a **histamine-reactive body state**.

This answer becomes much stronger if it coexists with **hives, itching without rash, dermographism, suspected histamine intolerance, or food-triggered pelvic flares**. When those symptoms cluster, the case for an **MCAS-like or histamine-amplified phenotype** becomes more coherent.

Hives

Hives are one of the most classic signs of **allergic or mast-cell–related mediator reactivity**. In the pelvic pain setting, hives do not directly locate the pain source, but they strongly suggest that the patient may have a system that is prone to **abnormal mediator release**, which could also influence bladder pain, urethral burning, pelvic pressure, or food-related flares.

This item becomes particularly significant if the patient also reports **flushing, histamine-food sensitivity, dermographism, itching without rash, or later benefit from antihistamines or hydroxyzine**. In such a pattern, pelvic pain may be occurring within a broader **immune-reactive framework** rather than a purely mechanical or neuropathic one.

Suspected histamine intolerance

This is one of the strongest direct histamine-related items in the section because it reflects the patient's own recognition of a possible relationship between **food, skin symptoms, bladder**

symptoms, pelvic pain, and symptom fluctuation. It is not diagnostic by itself, but it becomes highly meaningful when the overall questionnaire profile is consistent.

Its value becomes especially high when it occurs together with **histamine-rich food sensitivity, fermented-food sensitivity, alcohol sensitivity, itching, flushing, hives, pressure-reactivity, and benefit from DAO, quercetin, hydroxyzine, cetirizine, bilastine, fexofenadine, or H2 blockers**. In that pattern, suspected histamine intolerance becomes more than a vague self-label; it becomes a plausible description of a **histamine-amplified pelvic pain phenotype**.

Skin reaction to cold

Skin reaction to cold suggests that temperature change may trigger **cutaneous or mast-cell-related reactivity**, not merely discomfort. In a pelvic pain context, this can be especially useful when the patient also reports that **cold exposure worsens pelvic symptoms**, because then cold may be acting through both **muscular/autonomic** and **immune-reactive** pathways.

This answer becomes especially meaningful when paired with **hypersensitivity to cold, itching, hives, flushing, or reaction to pressure**, because that combination suggests a more generalized **reactive sensory–immune phenotype** rather than simple temperature dislike.

Reaction to pressure (e.g., tight clothing)

This is one of the most sophisticated and clinically useful items in the entire immune section. Pressure reactivity can sometimes be purely mechanical, but in some patients it behaves as something more than simple compression: **pressure itself seems to trigger discomfort, skin reactivity, or pelvic pain disproportionate to the amount of contact**. This raises the possibility of a **pressure-sensitive mast-cell / sensory amplification phenotype**.

This answer becomes highly important when it occurs with **tight clothing worsening pain, dermographism, itching, perineal hypersensitivity, allodynia, and symptoms after pressure elsewhere in the questionnaire**. In that context, pressure is not just a posture issue; it may be acting at a **sensory–immune interface**.

Allergies to pollen, dust/mites, mold

These items strengthen the case for a broad **atopic background**. Their importance lies not in proving a pelvic diagnosis, but in showing that the patient's immune system may already be prone to reacting to **multiple environmental triggers**. In a patient with pelvic pain, this background may make histamine- or mast-cell–related interpretations more plausible when supported by the rest of the questionnaire.

The more environmental allergies cluster with **flushing, itching, hives, rhinitis, and food-triggered symptoms**, the more likely it becomes that pelvic symptoms may be occurring in an **allergy-prone physiological background** rather than in isolation.

Allergies to animal dander

Animal dander allergy serves a similar purpose. It is another signal of a **reactive immune phenotype**. On its own it does not diagnose a mast-cell disorder, but in combination with multiple other allergic and histamine-related items it strengthens the interpretation of **systemic immune reactivity**.

Allergies to perfumes/detergents/chemicals

This item is especially interesting because it suggests not only classical allergy but sometimes a broader pattern of **chemical sensitivity, sensory reactivity, or irritant-triggered mediator release**. In chronic pelvic pain, this may be relevant when the patient appears generally reactive to environmental exposures beyond food alone.

Its importance rises if paired with **flushing, itching, sensory hypersensitivity, headaches, autonomic symptoms, or strong pressure/clothing sensitivity**, because then the patient may have a broader **high-reactivity sensory–immune phenotype**.

Itching without rash

Itching without rash is a subtle but highly useful clue. It can suggest **mediator activity without obvious visible skin lesions**, which is especially relevant in histamine-related symptom patterns. In chronic pelvic pain patients, this may signal that the body is reactive even when classic allergy signs are incomplete.

This item becomes particularly meaningful when it occurs with **flushing, hives, dermographism, pressure reactions, and food-related worsening**, because then the patient's symptom profile becomes more consistent with a **mast-cell / mediator-release phenotype**.

Dermographism (skin reacts to touch)

Dermographism is one of the most concrete signs in the whole section that the skin and peripheral sensory-immune interface may be **abnormally reactive to touch or friction**. In a chronic pelvic pain context, it is highly informative because it parallels the idea that pelvic tissues, genital skin, or the perineal region may also be unusually pressure- or contact-sensitive.

This answer becomes especially important if it coexists with **reaction to pressure, tight clothing aggravation, itching, hives, or perineal/genital hypersensitivity**. In that pattern, the pelvic pain phenotype may be shaped not only by deep structures but also by a **reactive cutaneous-sensory-mast-cell layer**.

Summary of Point 18

Point 18 is the questionnaire's **background immune-reactivity and histamine-phenotype section**. Its purpose is to determine whether the patient's pelvic pain is occurring inside a broader pattern of **atopy, histamine sensitivity, cutaneous reactivity, pressure-reactivity, and mediator-type symptoms**. The strongest indicators of a histamine/MCAS-like background are **flushing, hives, suspected histamine intolerance, itching without rash, dermographism, and pressure reactivity**, especially when combined with allergic disease and food-triggered worsening. In professional interpretation, Point 18 is valuable because it identifies whether the patient's pelvic pain may be occurring in a body that is generally **immune-reactive and mediator-sensitive**, which makes later benefit from **antihistamines, hydroxyzine, H1/H2 blockers, low-histamine diet, quercetin, DAO, or even brief anti-inflammatory treatment** much more interpretable.

Point 19. Gastrointestinal Symptoms

(select all that apply)

Purpose of Point 19

Point 19 is designed to determine whether the patient's pelvic pain is linked to the **gut–pelvis axis**, including bowel function, food-triggered reactivity, fermentation, motility problems, reflux-related upper-GI symptoms, and the possibility that intestinal symptoms amplify pelvic pain. This section is extremely important because chronic pelvic pain does not exist in isolation from the bowel. The pelvis is mechanically, neurologically, and immunologically connected to the lower gastrointestinal tract. Current chronic pelvic pain frameworks increasingly recognize that bowel symptoms and pelvic symptoms frequently overlap.

This point matters for several reasons. First, bowel dysfunction can act as a **direct mechanical trigger** by increasing rectal pressure, abdominal pressure, pelvic floor guarding, and discomfort in the perineum or deep pelvis. Second, gut symptoms may indicate a **visceral hypersensitivity phenotype**, in which the gut and pelvis are cross-sensitized. Third, food-triggered bowel reactions may strengthen the importance of **diet, microbiota, fermentation, histamine handling, and immune activation** in the overall pain syndrome. Emerging research continues to explore gut microbiome differences in CP/CPPS, suggesting that the gut may be more than an incidental bystander in at least some men.

This section is also highly relevant when later interpreted alongside Point 17 and the treatment-response sections. For example, if the patient reports **bloating, IBS, food-triggered pelvic pain, gluten or dairy sensitivity, FODMAP sensitivity, and later improvement with low-FODMAP, probiotics, gluten-free, or low-histamine strategies**, then the gastrointestinal component of the phenotype becomes much stronger. If short steroid benefit is reported elsewhere, that may support inflammatory amplification in some patients, but again it should be treated as a **supportive clue**, not a standalone diagnosis.

Bloating

Bloating is one of the most clinically important GI items because it often reflects **fermentation, gas retention, altered motility, gut dysbiosis, or visceral hypersensitivity**. In the context of pelvic pain, bloating can also have a direct mechanical effect: a distended abdomen and bowel can increase pressure within the pelvis and aggravate pelvic floor tension, bladder discomfort, and deep pressure symptoms.

This answer becomes especially meaningful when paired with **constipation, IBS, sensitivity to FODMAP diet, food worsening pelvic pain, or pelvic pressure when the bladder is full**, because then the gastrointestinal system may be contributing both **mechanically and physiologically** to the pelvic pain syndrome. Recent literature continues to support gut–pelvis interactions and microbiome involvement in CP/CPPS, although these pathways are still being actively investigated rather than treated as universally established for every patient.

Constipation

Constipation is a major symptom in pelvic pain because it can increase **rectal pressure, straining, pelvic floor overactivity, outlet dysfunction, and deep pelvic discomfort**. In some men, constipation may be one of the main engines of recurrent pelvic flares. It is especially important because it can both reflect and worsen a **non-relaxing pelvic floor phenotype**.

This answer becomes much stronger when combined with **pain associated with constipation, difficulty passing stool, rectal fullness, lower abdominal pressure, or breath-holding under stress**. In that context, the bowel is not a separate system; it is actively participating in the same dysfunctional pressure-regulation network as the pelvic floor.

Diarrhea

Diarrhea suggests a different GI pattern from constipation. It may reflect **IBS, food intolerance, infection history, post-infectious bowel sensitivity, inflammatory irritability, or gut-triggered autonomic reactivity**. In pelvic pain, diarrhea can aggravate symptoms through repeated bowel urgency, rectal irritation, and broader visceral hypersensitivity.

This answer becomes more meaningful if it coexists with **nausea during flare-ups, food-triggered pelvic pain, IBS, reflux, or relief/worsening after bowel movement**, because then the bowel is clearly involved in shaping the flare pattern.

Irritable bowel syndrome (IBS)

IBS is one of the most important overlap syndromes in the questionnaire. Its presence strongly supports the idea that the patient's pelvic pain may be part of a broader **visceral hypersensitivity or gut–pelvis interaction phenotype**. IBS and chronic pelvic pain frequently coexist, and systematic review evidence supports a meaningful relationship between pelvic pain syndromes and IBS.

When IBS is present together with **bloating, food-triggered pelvic pain, bowel-movement-related changes, lower abdominal pain, and stress reactivity**, the case for a **gut–brain–pelvis phenotype** becomes much stronger. This does not mean that the bowel explains everything, but it means the bowel is likely a major part of the pain network.

Food worsens pelvic pain

This is one of the most directly interpretive GI items in the whole questionnaire. If food worsens pelvic pain, then the relationship between eating and symptoms is not incidental. It suggests that meals may trigger symptoms through **fermentation, histamine, bladder irritation, autonomic shifts, gut distension, or immune activation**.

This answer becomes particularly important when paired with **gluten sensitivity, dairy sensitivity, FODMAP sensitivity, histamine-food sensitivity, reflux, bloating, or nausea during flare-ups**, because then the pattern is much more consistent with a real **diet-sensitive pelvic pain phenotype** rather than with random food avoidance.

Gluten sensitivity

Gluten sensitivity is clinically relevant whether it reflects celiac disease, non-celiac gluten sensitivity, or a patient-specific gut symptom pattern. In chronic pelvic pain, it becomes meaningful when it contributes to **bloating, bowel disturbance, fatigue, abdominal discomfort, and worsening of pelvic symptoms after eating**.

Its importance rises when combined with **food intolerances, IBS, dairy sensitivity, FODMAP sensitivity, and positive celiac-related testing elsewhere in the survey**. In such a case, gluten may be part of a broader **gut-reactive phenotype** rather than an isolated nutritional preference.

Dairy sensitivity

Dairy sensitivity can indicate **lactose intolerance, broader food reactivity, fermentation problems, or gut-triggered symptom amplification**. In the pelvic pain setting, it becomes important if dairy ingestion is associated with **bloating, diarrhea, abdominal pain, or worsening pelvic symptoms**.

If paired with **food intolerances, positive lactose testing, IBS, and low-FODMAP benefit**, dairy sensitivity becomes a much stronger clue to a **fermentation- or intolerance-linked GI phenotype**.

Sensitivity to FODMAP diet

This item is especially informative because it directly points toward a **fermentation-sensitive gut phenotype**, often associated with IBS, bloating, gas, motility changes, and visceral hypersensitivity. In chronic pelvic pain, a positive answer here suggests that fermentable carbohydrates may be intensifying not only GI symptoms but also the pelvic pain state through **abdominal distension, gut–pelvis cross-sensitization, and increased pelvic floor load**.

This answer becomes especially important if later sections show **bloating, IBS, food worsening pelvic pain, relief with bowel management, or improvement on low-FODMAP diet**, because that combination strongly supports a **gut–pelvis fermentation-sensitive phenotype**.

Gastroesophageal reflux

Reflux is clinically relevant because it suggests that the GI phenotype is not confined to the lower bowel. It may reflect broader digestive dysregulation, upper-GI sensitivity, dietary reactivity, or autonomic imbalance. While reflux does not directly explain pelvic pain, it can indicate that the patient's digestive system as a whole is unusually reactive.

Its meaning increases when combined with **nausea during flare-ups, stomach pain after eating, hyperacidity, irregular sleep, or food-triggered pelvic pain**, because then the GI contribution appears more systemic rather than local.

Nausea during flare-ups

Nausea during flares suggests that the pain episodes may involve not only pelvic symptoms but also **broader autonomic or visceral activation**. It can indicate that flares are body-wide events, not merely local pain spikes.

This item becomes especially important if it appears with **food sensitivity, reflux, abdominal pain, diarrhea, stress reactivity, or pronounced flare cycles**, because it suggests that the GI and autonomic systems are part of the flare physiology.

Relief after bowel movement

Relief after bowel movement suggests that pelvic symptoms may be reduced when **rectal or abdominal pressure is released**. This strongly supports a **bowel–pelvic pressure interaction**. It may mean that stool burden, gas, or bowel distension is actively aggravating the pelvis and that evacuation temporarily decreases the load.

This answer becomes particularly meaningful with **constipation, bloating, rectal fullness, lower abdominal pain, and pelvic floor symptoms**, because then the bowel is functioning as a direct pressure modulator of pelvic pain.

Worsening after bowel movement

Worsening after bowel movement suggests a different mechanism from relief. It may indicate that the act of evacuation itself is painful because of **pelvic floor overactivity, anal sphincter spasm, rectal hypersensitivity, or bowel-triggered pelvic muscle recruitment**. This answer becomes especially relevant when paired with **pain associated with constipation, difficulty passing stool, anal pain, rectal fullness, or pain when stretching/contracting pelvic muscles**, because then defecation may be acting as a **trigger event** for an already sensitized pelvic floor.

Stomach pain after eating

Stomach pain after eating suggests that the patient has an **upper-GI meal-triggered pain response**, which may reflect gastritis, reflux, dyspepsia, food intolerance, or broader digestive hypersensitivity. In the chronic pelvic pain context, it becomes relevant because it suggests that meals are affecting the GI system strongly enough to potentially modulate the whole pelvic pain network.

This item becomes more important when combined with **reflux, hyperacidity, nausea during flare-ups, and food worsening pelvic pain**, because then the patient's flares may include an upper-GI component rather than only a lower bowel one.

Stomach hyperacidity

Hyperacidity supports an **upper-GI irritative phenotype**, which may coexist with reflux, gastritis-like symptoms, nausea, and food-triggered discomfort. On its own, it does not directly explain pelvic pain, but it shows that the patient's gastrointestinal tract may be chronically reactive and meal-sensitive.

Its relevance to pelvic pain becomes greater when it occurs with **food-triggered pelvic symptoms, nausea, reflux, and irregular sleep**, because those together suggest a broader **digestive-autonomic-pain phenotype**.

Too much bile in the stomach (gastritis)

This item reflects the patient's perception of a **gastritis-like or bile-related upper-GI irritation pattern**. In questionnaire interpretation, it is best treated as a marker of **digestive reactivity and upper-GI symptom burden**, rather than as a precise mechanistic diagnosis.

Its significance increases when paired with **stomach pain after eating, hyperacidity, nausea during flare-ups, reflux, and food-triggered pelvic pain**, because then the case for a broader **upper-GI–linked pain amplification pattern** becomes stronger.

Summary of Point 19

Point 19 is the questionnaire's **gastrointestinal and gut–pelvis axis section**. Its purpose is to determine whether the patient's chronic pelvic pain is linked to **bowel function, food-triggered reactivity, fermentation, motility problems, reflux, meal-related symptoms, and bowel-movement-related modulation of pain**. The strongest indicators of a gut–pelvis phenotype are **bloating, constipation, IBS, food worsening pelvic pain, FODMAP sensitivity, and clear symptom change after bowel movements**. Reflux, hyperacidity, stomach pain after eating, nausea, and gastritis-like symptoms broaden the interpretation toward a more **global digestive-reactive phenotype**. In professional use, Point 19 is essential because it identifies whether pelvic pain may be driven not only by pelvic floor or nerves, but also by a **gut–immune–autonomic–mechanical axis** that increases pelvic symptom burden. Emerging literature on CP/CPPS and the gut microbiome further supports taking this domain seriously, even though it remains an active field of research rather than a finalized explanatory model.

Purpose of Point 20

Point 20 is one of the most clinically valuable items in the entire questionnaire because it does something that the earlier symptom sections do not: it forces the patient to create a **hierarchy of real-life trigger dominance**. Earlier sections identify where the pain is, what it feels like, what

aggravates it, and what body systems seem involved. Point 20 asks a more decisive question: **when everything is taken together, which trigger actually matters most in daily life?** That makes this item far more than a simple summary. It is effectively a **priority map of the active phenotype**.

In chronic pelvic pain, patients often have more than one genuine mechanism. A man may have pelvic floor overactivity, bladder sensitivity, post-ejaculatory pain, histamine-related flares, poor sleep, and stress reactivity all at once. But not all mechanisms are equally active at the same moment in the same patient. One patient is mostly destabilized by sitting. Another by stress. Another by frequent ejaculation. Another by bowel and diet triggers. Another by sleep fragmentation. Point 20 is therefore crucial because it shifts interpretation from **"what is present?"** to **"what is clinically dominant?"**. That is fully consistent with modern phenotype-based chronic pelvic pain care.

This ranking question is also extremely important for treatment logic. If a patient's main trigger is stress, the case for psychophysiological and autonomic interventions becomes stronger. If the dominant trigger is sitting, then pressure unloading and pelvic floor mechanics become central. If diet ranks first, then gut/bladder/histamine pathways deserve much more attention. If sexual activity ranks first, then the clinician must carefully distinguish **sexual-overload, post-ejaculatory inflammatory or neuroimmune flares, poor recovery of deep ejaculatory structures, and congestion-sensitive phenotypes**. If infrequent sexual activity ranks first, that suggests a very different sexual phenotype from frequent-ejaculation worsening. Point 20 therefore helps move from descriptive medicine toward **mechanism-prioritized medicine**.

Another important function of this point is that it reduces interpretive confusion in patients with many symptoms. A patient may endorse ten or fifteen aggravators elsewhere in the survey, but that still leaves the question of what **actually destabilizes him first and most strongly**. Ranking obliges the patient to reveal which trigger has the greatest immediate influence on symptoms in real life. This is especially important in a syndrome like CPPS where **secondary amplifiers** often appear over time. For example, stress may increase attention to pain, but sitting may still be the primary trigger. Or sexual activity may provoke the strongest flare, but only when sleep is poor and stress is high. The ranking helps reveal that structure.

Susceptibility to stress

If stress is ranked first, that strongly suggests that the patient's pain is living inside a **highly stress-reactive neuro-autonomic system**. This does not mean the pain is "psychological instead of physical." It means that stress likely acts as a dominant **physiological amplifier** through pelvic floor tightening, breath holding, sympathetic activation, reduced parasympathetic recovery, bladder urgency, bowel tension, and heightened pain salience. Chronic pelvic pain guidelines explicitly recognize the importance of psychosocial and behavioral contributors and the need for multidisciplinary assessment.

This ranking becomes especially compelling if the patient also reports **job stress, chest-dominant breathing, breath holding during stress, pelvic floor clenching, worsening of symptoms during anxiety, body-focus worsening, poor sleep, palpitations, and difficulty relaxing even when time to rest is available**. In that pattern, the pelvis is functioning inside a broader **psychophysiological pain phenotype**. The dominant problem is not that stress "creates symptoms out of nothing," but that it repeatedly drives the same pelvic pain pathways through muscle guarding, autonomic arousal, and attention-related amplification.

If stress ranks first but sitting, ejaculation, and diet are also relevant, the interpretation is often that stress is the **master amplifier** of other triggers rather than the only cause. In such cases, even a real

local trigger may become far more symptomatic in the presence of high arousal. That distinction is clinically important.

Too long sitting

If prolonged sitting ranks first, the phenotype is strongly shifted toward a **mechanical-compressive pelvic pain model**. This suggests that the patient's pelvis is especially vulnerable to perineal pressure, pelvic floor shortening, static postural loading, pudendal-territory compression, and possibly venous pooling or posterior pelvic neural irritation. In men with chronic pelvic pain, sitting is one of the most clinically meaningful real-world triggers because it directly loads the exact region that many patients describe as painful.

This ranking becomes especially strong when paired with **perineal pain, anal pain, genital/pudendal pain, driving pain, cycling pain, leaning-forward worsening, tingling or numbness in the thighs or buttocks when sitting, and symptom improvement with cushions or offloading**. In such a case, sitting is not just "annoying"; it is likely the clearest expression of a **pelvic floor–pudendal–mechanical phenotype**. If the patient also reports that lying down improves symptoms, a vascular component may coexist, but sitting still remains the dominant daily trigger.

If stress is also high, sitting may still outrank it because the body can sometimes tolerate stress better than direct perineal compression. That is exactly why the ranking is useful.

Diet (e.g. unhealthy food, alcohol)

If diet ranks first, the patient likely has a **food-responsive bladder, gut, or immune-reactive phenotype**. This is one of the most important ranking outcomes because it suggests that meals, drinks, histamine load, acidity, fermentation, or metabolic-irritative factors directly destabilize pelvic symptoms. In current literature, CP/CPPS is increasingly recognized as potentially linked to gut microbiota, neuroimmune signaling, and the gut–prostate or gut–pelvis axis, although this remains an evolving field rather than a finalized causal model.

A diet-first ranking becomes especially meaningful when paired with **food intolerances, histamine-rich food sensitivity, alcohol worsening, fermented-food worsening, bloating, IBS, FODMAP sensitivity, reflux, nausea during flares, bladder pain, urgency, urethral burning, flushing, hives, or benefit from low-histamine diet, low-FODMAP diet, antihistamines, quercetin, DAO, luteolin, or vitamin C**. In that pattern, diet is not just a general "health factor"; it is likely one of the main ways that the patient's gut, bladder, or immune system repeatedly enters a flare-prone state.

If diet ranks first and the patient later reports benefit from **short steroid courses**, that may support an inflammatory or neuroimmune amplification layer during some flares. If he reports benefit from **hydroxyzine, H1 antihistamines, H2 blockers, or other antihistamines**, that adds weight to a histamine- or bladder-sensory phenotype, especially if allergic symptoms and histamine-food sensitivity are present. These treatment clues do not prove a single mechanism, but they become much more meaningful when diet ranks at the top.

Sexual activity (Ejaculations)

If sexual activity ranks first, then the questionnaire is identifying a patient whose pelvic system is especially destabilized by **arousal, orgasm, ejaculation, or post-ejaculatory recovery**. This is one of the most important ranking outcomes because it demands a very careful sexual-physiology

interpretation. In current AUA materials, pain with ejaculation is specifically highlighted as clinically important and often associated with pelvic floor myalgia or dysfunction.

This ranking becomes especially meaningful if the patient also reports **pain during ejaculation, pain immediately after ejaculation, pain several hours or the next day after ejaculation, urethral burning after ejaculation, seminal vesicle-region pain, reduced semen volume, weak ejaculation, pain during nocturnal emission, high libido, difficulty relieving sexual tension, and worsening with frequent daily ejaculation**. In that pattern, sexual activity is not simply "a trigger." It may be the main challenge that reveals poor recovery of the **pelvic floor–prostate–seminal vesicle–urethral system**.

This is also where the issue of **high sexual drive and frequent ejaculation** must be interpreted carefully. Current evidence does **not** justify saying that frequent ejaculation is universally harmful or that it inherently damages the prostate or seminal vesicles in all men. However, if sexual activity ranks first and the patient also has **high libido, repeated ejaculation over short intervals, deep seminal-vesicle/prostate-region pain, reduced semen volume, weak ejaculation, and post-ejaculatory flares**, then it is clinically reasonable to infer that his **deep ejaculatory structures may not be recovering comfortably between symptom-provoking events**. That is a **patient-specific load-recovery mismatch**, not a universal biological rule.

If the same patient later reports partial benefit from **brief steroids**, that may suggest a post-inflammatory or inflammatory amplification component in some flares. If he reports benefit from **antihistamines**, that may suggest that sexual activity is sometimes followed by a neuroimmune or histamine-amplified flare rather than only a mechanical one. Those later clues do not redefine the ranking, but they help explain **why sexual activity sits at the top**.

Physical activity (e.g. lifting weights, bicycle)

If physical activity ranks first, the dominant phenotype is likely **biomechanical, pressure-sensitive, or exercise-reactive**. This suggests that movement, load transfer, abdominal pressure, or muscle recruitment destabilizes symptoms more reliably than stress, diet, or ejaculation. In such cases the pelvis often behaves as part of a **larger lumbopelvic movement system** rather than as an isolated organ pain syndrome.

This ranking becomes especially compelling if paired with **heavy lifting as an aggravator, pain with increased abdominal pressure, bending, coughing, lower-back stiffness, hip tightness, hip pain, cycling pain, walking-related worsening, and pelvic floor pain during muscle contraction**. Then the likely phenotype is a **musculoskeletal–pelvic floor–pressure management problem**. If cycling is particularly prominent, pudendal or seat-pressure mechanisms become more likely. If weightlifting is dominant, pelvic floor over-recruitment and abdominal pressure dysregulation become more plausible.

If physical activity ranks first but the patient also responds well to **stretching, pelvic floor physiotherapy, myofascial release, trigger point therapy, dry needling, or relaxation-based approaches**, this strongly supports a functional musculoskeletal interpretation rather than primary inflammatory disease.

Sleep problems

If sleep problems rank first, the dominant issue may not be what initially caused the pelvic pain, but what now most strongly **controls flare threshold and recovery failure**. Sleep disruption is highly relevant in chronic pain because it increases pain sensitivity, worsens autonomic balance, reduces emotional regulation, intensifies body vigilance, and impairs tissue and nervous-system recovery.

This ranking becomes especially meaningful when paired with **difficulty sleeping, nocturia, nocturnal urgency, night pain, feeling mentally "on edge," racing thoughts, anxiety, irregular sleep aggravating symptoms, migraine, fibromyalgia, or pain that persists after the stimulus is removed**. In that context, the patient may have a **sleep-sensitive central amplification phenotype**, in which poor sleep makes every other trigger more potent.

If sleep problems rank first and the patient also reports diet and stress as highly ranked triggers, then sleep may be the **amplifier of amplifiers** rather than the original cause. That is an important distinction in interpretation.

Infrequent sexual activity

If infrequent sexual activity ranks first, this points toward a very different sexual phenotype from "frequent ejaculation worsens me." Here the dominant issue is likely **abstinence sensitivity, congestion sensitivity, deep pelvic fullness, or difficulty tolerating prolonged sexual inactivity**. This becomes especially important when the patient reports **relief after ejaculation, worsening after several days or weeks of abstinence, deep prostate/seminal-vesicle pressure, pelvic heaviness, or fullness in the pelvis**.

This ranking does **not** mean that abstinence is harmful for everyone or that all men need more ejaculation. It means that in this particular patient, the system appears unstable when sexual activity is absent for too long. In some cases, this may reflect a **congestion-sensitive phenotype** rather than a pelvic floor or inflammatory-overload phenotype. It may also overlap with vascular heaviness or deep ejaculatory-system pressure.

If later treatment-response items show that **increased ejaculation frequency** helps, that interpretation becomes much stronger. By contrast, if the patient is simply high-libido but also worsens with frequent ejaculation, then abstinence is less likely to be the dominant issue. The ranking helps separate those two clinically distinct sexual patterns.

Unknown / other factors

If unknown or other factors rank first, this usually suggests one of three possibilities. First, the patient may truly have a **multifactorial syndrome with no single dominant trigger**. Second, the main trigger may not be well captured by the listed categories — for example a very specific posture, bowel pattern, medication, environmental exposure, or life situation. Third, the syndrome may have become sufficiently chronic and self-sustaining that the patient no longer perceives one dominant driver.

This ranking is especially important because it warns the evaluator not to overfit the case to one simplistic explanation. In such patients, the diagnostic value of Point 21 and the treatment-response sections becomes even greater.

Summary of Point 20

Point 20 is the questionnaire's **dominant-trigger hierarchy section**. Its purpose is to identify which mechanism is **most immediately and most powerfully destabilizing the patient's symptoms in real life**. A top ranking for **stress** supports a psychophysiological/autonomic phenotype. A top ranking for **sitting** supports a mechanical-compressive / pudendal / pelvic floor phenotype. A top ranking for **diet** supports a gut–bladder–immune or histamine-reactive phenotype. A top ranking for **sexual activity** supports an ejaculatory-load / post-ejaculatory recovery phenotype, especially in men with high libido and frequent-ejaculation worsening. A top ranking for **physical activity** supports a biomechanical–pressure phenotype. A top ranking for **sleep**

problems supports a recovery-sensitive central amplification phenotype. A top ranking for **infrequent sexual activity** supports an abstinence/congestion-sensitive phenotype. In professional interpretation, this item is crucial because it converts a long symptom inventory into a **working clinical priority map**.

Point 21. DIAGNOSTIC TESTS in CPPS / Pelvic Pain

Purpose of Point 21

Point 21 is the questionnaire's **objective investigation and diagnostic-anchoring section**. Its purpose is to determine what has actually been examined in the patient and, by extension, which mechanisms have been **ruled out, supported, or made more plausible by testing**. This is critically important in chronic pelvic pain because CPPS is often a diagnosis reached after extensive evaluation, but it is not simply a diagnosis of "nothing found." Rather, modern guidance treats it as a complex syndrome in which pain may coexist with normal tests, subtle abnormalities, functional disorders, post-inflammatory change, pelvic floor dysfunction, neurologic findings, and overlap conditions.

This section is especially valuable because it allows the clinician or researcher to distinguish between **symptom reports** and **investigated biology**. For example, urinary burning with normal urine cultures points in a different direction from urinary burning with recurrent positive cultures. Deep prostate-region pain with normal imaging means something different from deep pain plus TRUS or MRI changes. Food-triggered flares plus positive celiac or DAO-related findings differ from similar symptoms without any testing. Point 21 therefore does not merely record whether the patient was "worked up." It maps **what kinds of mechanisms have already been explored**.

Another key function of Point 21 is to prevent false dichotomies. In chronic pelvic pain, **normal tests do not invalidate severe symptoms**, and abnormal tests do not automatically eliminate functional or nervous-system mechanisms. A patient may have real post-inflammatory or fibrotic changes on imaging and also have pelvic floor hypertonicity and central sensitization. Conversely, a patient may have completely negative imaging but a very clear pelvic floor and neuropathic phenotype. Modern chronic pelvic pain care is explicitly built around integrating symptom patterns with objective testing, rather than assuming that positive tests equal "real disease" and negative tests equal "functional symptoms."

Blood tests

The blood-test section is designed to identify whether systemic, hormonal, inflammatory, autoimmune, or histamine-related mechanisms have been formally evaluated. This includes PSA, sex hormones, inflammatory markers, cytokines, autoimmune markers, tryptase, plasma histamine, DAO activity, celiac-related markers, and related laboratory workups in the latest survey versions.

From an interpretive standpoint, blood tests help answer several different questions. PSA and hormone panels help determine whether the prostate/androgen axis may be relevant. CRP, ESR, cytokines, and autoimmune markers help identify whether there is evidence of **systemic inflammation or immune dysregulation**. Tryptase, plasma histamine, and DAO activity help explore whether a **histamine-sensitive or mast-cell–associated phenotype** has some biochemical support, recognizing that clinical histamine-related syndromes may still exist even with normal or borderline markers. Anti-transglutaminase antibodies and total IgA help determine whether gluten-related disease might contribute to a broader gut–immune phenotype.

Clinically, the value of these tests is not only in positive findings. A patient with very strong immune-type symptoms, food reactivity, itching, flushing, and antihistamine responsiveness may still have a largely normal routine blood panel. That does not erase the phenotype. It simply means that the immune-reactive layer is not captured by every routine laboratory test. This is why Point 21 is about the **scope and direction of evaluation**, not just about "abnormal versus normal."

Urine tests

The urine-test section is central because it helps separate **infectious, inflammatory, and noninfectious lower-tract pain patterns**. It typically includes urinalysis, urine culture, PCR tests for sexually transmitted organisms, and in newer versions also methylhistamine or related urine markers where applicable.

In chronic pelvic pain, one of the most important interpretive questions is whether urinary symptoms reflect **persistent infection, prior infection with ongoing post-infectious pain, bladder hypersensitivity, urethral irritation, pelvic floor dysfunction, or histamine-related lower-tract sensitivity**. Repeatedly negative urine cultures in a patient with urgency, burning, and bladder discomfort strongly support a **nonbacterial CPPS / bladder-sensory / pelvic floor phenotype** rather than ongoing infection. PCR testing becomes relevant when ordinary cultures are negative but atypical infectious pathways were still considered.

If the patient later reports benefit from **antihistamines** and also had bladder pain, urgency, pressure on filling, and negative cultures, that makes a bladder–sensory or immune-reactive interpretation more plausible. If short **steroid responsiveness** is present in some flares, it may support an inflammatory-amplification layer, but urine testing remains crucial to distinguish infection from noninfectious inflammation or hypersensitivity.

Stool examination

The stool section exists to determine whether **bowel inflammation, dysbiosis, digestion issues, parasites, or occult bleeding** have been explored. In the latest survey versions this includes calprotectin, general stool examination, microbiota testing, parasite testing, and fecal occult blood.

This is important because Point 19 may reveal a strong gut–pelvis phenotype, but stool testing helps show whether that phenotype has been investigated for **objective inflammatory or microbial clues**. Elevated calprotectin may point toward inflammatory bowel processes rather than simple IBS. Microbiota testing, though still an evolving area clinically, becomes relevant when bloating, IBS, food-triggered pelvic pain, and FODMAP sensitivity are prominent. Recent reviews continue to discuss the microbiota–CP/CPPS relationship as a plausible and important research direction.

Breath tests

Breath testing, especially for SIBO, lactose intolerance, and fructose intolerance, is highly relevant in patients with a **fermentation-sensitive or food-reactive phenotype**. This part of Point 21 asks whether gut symptoms were explored not just generally, but specifically in terms of **malabsorption and fermentation mechanisms**.

If a patient later ranks diet high, reports bloating, IBS, FODMAP sensitivity, relief with dietary change, and positive breath tests, then the gut contribution to pelvic pain becomes much more biologically plausible. It suggests that the pain may be amplified not only by pelvic tissues, but by **intestinal gas burden, visceral sensitivity, and gut–pelvis signaling**.

Saliva

Salivary testing, especially diurnal cortisol profiles, is relevant because it explores the **stress-regulation system** in a more dynamic way than a single morning blood test. This becomes especially important when stress ranks highly, sleep is poor, and symptoms clearly worsen during periods of psychological strain.

In chronic pelvic pain, abnormal diurnal cortisol patterns do not diagnose the syndrome, but they can support the idea that the body's **stress-response rhythm and recovery rhythm** are disrupted. That has special relevance in patients with strong psychophysiological and sleep-sensitive phenotypes.

Semen tests

The semen-testing block is one of the most important diagnostic subsections in men whose symptoms are strongly linked to ejaculation. The latest survey includes basic semen analysis, leukocytes, round cells, aggregates/clumps, tryptase if available, cytokines such as IL-6, semen culture, PCR for atypical organisms, and STI testing.

This section matters tremendously in patients with **pain during ejaculation, pain after ejaculation, deep seminal-vesicle/prostate-region pain, weak ejaculation, reduced semen volume, or high sexual drive with frequent-ejaculation worsening**. If inflammatory cells, cytokines, or other abnormalities are present in semen, they make it more plausible that the deep ejaculatory tract carries a real **inflammatory or post-inflammatory burden**. That does not exclude pelvic floor dysfunction or neural amplification, but it helps explain why some men feel as though the deeper sexual apparatus is truly "irritated" rather than merely over-monitored.

This is also one of the places where later response to **brief steroids** may have more meaning. Steroid responsiveness should never be treated as proof of one diagnosis, but if the patient has seminal-vesicle-region pain, post-ejaculatory delayed flares, and inflammatory semen findings, a steroid-responsive pattern may support an **inflammatory amplification layer** in selected cases. Likewise, if tryptase or cytokine-related semen findings are relevant and the patient later benefits from **antihistamines**, that may support a **neuroimmune or mast-cell–linked component** in some individuals. Those are phenotype clues, not absolute rules.

Endoscopy

Endoscopy, including gastroscopy and colonoscopy, is less about the pelvis directly and more about whether upper and lower GI pathology has been formally evaluated. This becomes highly relevant in men with **reflux, stomach pain after eating, hyperacidity, bowel symptoms, food-triggered pelvic pain, or possible inflammatory bowel disease features**.

If a patient has a strong GI phenotype but minimal work-up, Point 21 reveals a gap. If extensive endoscopy is normal despite strong GI symptoms, that may shift interpretation toward **functional gut–pelvis overlap** rather than overlooked structural GI disease.

Imaging tests (radiology)

Imaging is one of the most clinically sensitive components of the assessment because it addresses a central question for both patients and clinicians: **are there any visible structural or functional abnormalities within the pelvic region?** In the current framework, this includes TRUS, pelvic MRI, prostate MRI, lumbar and sacral MRI, dynamic pelvic floor ultrasound, hip imaging, Doppler ultrasound of pelvic veins, venous MRI assessment, CT/MR venography, and phlebography where applicable.

Imaging can support multiple pelvic pain phenotypes by identifying structural, vascular, or post-inflammatory changes. Prostate and pelvic MRI, particularly high-resolution imaging such as **3T MRI**, may reveal subtle but clinically relevant findings, including **post-inflammatory alterations, asymmetry, fibrosis-like changes, microcalcifications, and chronic tissue remodeling**. In some patients, especially those with deep pelvic pain or symptoms related to ejaculation, imaging of the prostate and seminal vesicles may also suggest **relative stasis of secretions, dilation of seminal vesicles, or signs consistent with impaired outflow**.

Importantly, advanced MRI imaging may in selected cases raise suspicion of **partial obstruction or functional impairment at the level of the ejaculatory ducts**, including areas where the outflow of prostatic or seminal vesicle fluid may be restricted. This can occur due to **chronic inflammation, fibrosis, calcifications, or structural narrowing**, potentially contributing to increased intraductal pressure, congestion, and irritation of surrounding tissues. Over time, such mechanisms may be associated with **chronic low-grade inflammation, recurrent irritation, or even stone (calcification) formation**, which can further disrupt normal drainage and perpetuate symptoms.

For example, long-standing changes within the seminal vesicles or prostate have been described in surgical and imaging contexts in patients with chronic pelvic pain, supporting the concept that deep pelvic structures can be involved in persistent symptom generation . However, it is essential to interpret such findings carefully and within the broader clinical context.

Beyond the prostate, lumbar and sacral imaging becomes highly relevant in patients presenting with neurological features such as numbness, tingling, radiating pain, or suspected nerve root involvement. Hip imaging is important in musculoskeletal phenotypes, particularly where movement-related pain or biomechanical dysfunction is suspected. Pelvic venous Doppler and venographic studies play a key role in identifying **vascular congestion syndromes**, especially in patients reporting pelvic heaviness, fullness, visible varicosities, or improvement in supine positions.

A critical interpretive principle must be emphasized:
positive imaging findings do not exclude functional, neuromuscular, or central pain mechanisms, and negative imaging does not invalidate the patient's symptoms. Many patients with significant pain have minimal or no visible structural abnormalities, while others may have imaging changes that are incidental or only partially explanatory.

Therefore, imaging should be understood as a tool that helps identify **possible structural, vascular, or post-inflammatory contributors**, but it does not replace a comprehensive phenotype-based interpretation that integrates muscular, neural, immune, and behavioral factors.

Urological examinations

This subsection includes DRE, cystoscopy, uroflowmetry, post-void residual urine, and urodynamic study. These tests help determine whether urinary symptoms reflect **structural outlet disease, functional voiding problems, bladder pain overlap, or largely sensory symptoms with little objective obstruction**.

For example, uroflowmetry and residual urine help distinguish true emptying impairment from a mainly sensory sensation of incomplete emptying. Cystoscopy may be relevant in selected bladder-pain phenotypes. DRE remains important for prostate tenderness and pelvic floor tone. If alpha-blockers later help, these tests help place that response in context.

Nervous system testing

The nervous-system subsection includes nerve conduction studies, sensory testing for allodynia and hypersensitivity, central sensitization questionnaires, QST, and in some versions even

acknowledgment of diagnosed ADHD or Asperger's syndrome. This section addresses whether the patient's pain has been objectively or semi-objectively explored from a **neuropathic or sensitization perspective**.

This is especially important in patients with **burning, electric shocks, allodynia, numbness, radiating pain, disproportionate pain, pain spread, migraine, fibromyalgia, or strong sensory sensitivity**. Modern chronic pelvic pain care increasingly recognizes central sensitization and neuropathic contributions, so this section helps document whether the patient's case has been assessed at that level.

Examination by a pelvic floor physiotherapist

This is one of the highest-value subsections in the entire diagnostic block. It includes digital rectal pelvic floor assessment, trigger point evaluation, functional ultrasound, pelvic floor EMG, biofeedback, breathing-pattern assessment, and posture/biomechanics evaluation.

In men with perineal tension, pelvic floor clenching, pain during ejaculation, rectal fullness, bowel dysfunction, sitting pain, or hip/lower-back stiffness, these evaluations can directly support a **pelvic floor myalgia / biomechanical phenotype**. This is especially important because many patients spend years with the label "prostatitis" before anyone directly examines the pelvic floor as a functional structure.

Allergy / immunology testing

The allergy/immunology subsection includes allergy testing, autoimmune testing, total IgE, and specific IgE in the latest versions. This section matters most in patients with **flushing, hives, dermographism, pressure reactivity, allergic disease, histamine-food sensitivity, or antihistamine responsiveness**.

Its value is not that it can diagnose every histamine-related or MCAS-like phenotype conclusively. Rather, it helps show whether the patient's **immune-reactive background** has been explored beyond symptom description alone.

Summary of Point 21

Point 21 is the questionnaire's **diagnostic evidence and investigation section**. Its purpose is to map **what has actually been tested** across hormonal, inflammatory, immune, urinary, gastrointestinal, sexual, structural, neurologic, musculoskeletal, and allergy-related domains. In professional interpretation, this point is essential because it shows not only whether the patient has been evaluated, but **which mechanisms were seriously considered**. Blood and immune tests clarify inflammatory, hormonal, autoimmune, and histamine-related possibilities. Urine and semen tests help distinguish persistent infection from post-infectious, inflammatory, or functional pain. Stool and breath tests strengthen gut–pelvis interpretations. Imaging clarifies structural, vascular, spinal, hip, and post-inflammatory possibilities. Urological studies assess bladder and outlet function. Nervous-system tests support neuropathic and sensitization phenotypes. Pelvic floor physiotherapy assessment identifies myofascial and functional pelvic floor drivers. Allergy/immunology testing supports immune-reactive backgrounds. Most importantly, Point 21 reinforces a core principle of modern chronic pelvic pain medicine: **normal tests do not invalidate severe symptoms, and abnormal tests do not eliminate functional, muscular, neural, or central amplification mechanisms**. The section's real purpose is therefore not simply to collect investigations, but to anchor the patient's phenotype in the strongest available objective context.

Point 22. Non-pharmacological treatments - for some it helps, for others it does not. *(check all that have helped you)*

Purpose of Point 22

Point 22 is one of the most clinically valuable sections in the whole questionnaire because it treats **response to non-pharmacological treatment as diagnostic information**. In chronic pelvic pain, what helps is not random. Improvement after a particular intervention often reveals **which biological system is currently dominant**: mechanical compression, pelvic floor overactivity, deep myofascial tension, bladder or bowel reactivity, autonomic overactivation, vascular loading, neuropathic pain, or histamine-related symptom amplification. In that sense, this section functions almost like a **natural experiment**. It asks: when the patient changes the way he sits, breathes, moves, relaxes, eats, or unloads the pelvis, does the pain change in a meaningful way? If yes, that itself becomes powerful phenotype evidence.

This section is especially important because current guidelines do not support a one-size-fits-all model for CPPS. Men with chronic pelvic pain are heterogeneous, and non-drug interventions often reveal that heterogeneity more clearly than labels do. For example, if the patient improves mainly with pelvic floor physiotherapy, trigger point work, and diaphragmatic breathing, that suggests a different dominant mechanism than improvement mainly with low-histamine diet, antihistamines, and bladder-directed measures. Likewise, improvement with offloading cushions and reducing sitting strongly supports a different phenotype from improvement with increased ejaculation frequency. Point 22 therefore helps convert a long symptom survey into a **functionally testable map of mechanism**.

A further strength of this section is that it helps distinguish between what **aggravates** symptoms and what genuinely **modulates** them. A patient may know that sitting worsens pain, but Point 22 asks whether reducing sitting actually helps. A patient may know that ejaculation can trigger symptoms, but Point 22 asks whether **less** ejaculation helps, whether **more** ejaculation helps, or whether the pattern is mixed. This is especially important in the context of high sexual drive and frequent ejaculation, where current medical knowledge does **not** support a universal rule, but does support a phenotype-specific interpretation: in some men, repeated ejaculation seems to outpace the comfortable recovery of the **pelvic floor–prostate–seminal vesicle system**, while in others infrequent ejaculation worsens congestion-type symptoms.

Reducing sitting time

If reducing sitting time helps, that strongly supports a **mechanical-compressive / pelvic floor / pudendal-sensitive phenotype**. Sitting is one of the most consistent real-world aggravators in chronic pelvic pain because it loads the perineum, shortens the hips, reduces movement variability, and may increase pelvic floor guarding. Improvement after reducing sitting suggests that the pain is at least partly driven by **chronic perineal load rather than purely internal organ dysfunction**. This becomes especially strong if the patient also has perineal pain, anal pain, driving pain, cycling sensitivity, or tingling and numbness when sitting.

Reduced frequency of ejaculation

If reduced ejaculation frequency helps, the likely interpretation is a **sexual-overload phenotype**. This does not mean ejaculation is inherently harmful. It means that, in this patient, repeated sexual activation appears to exceed the comfortable recovery capacity of the deep pelvic system. This is

especially important when accompanied by **pain during ejaculation, pain immediately or hours after ejaculation, pain the next day, seminal vesicle-region pain, reduced semen volume, weak ejaculation, high libido, or worsening with frequent daily ejaculation**. In such cases, reduced frequency may help because the pelvic floor, peri-prostatic tissues, and deep ejaculatory structures are given more time to settle between events. That is a patient-specific **load–recovery mismatch**, not a universal biological rule.

Increased frequency of ejaculation

If increased ejaculation frequency helps, that supports a different sexual phenotype: a more **congestion-sensitive or abstinence-sensitive pattern**. In such men, the dominant issue may be deep pressure, fullness, pelvic heaviness, or worsening after several days or weeks of abstinence rather than sexual overuse. Improvement with more frequent ejaculation becomes especially meaningful if the patient also reports **relief after ejaculation, worsening with abstinence, deep prostate/seminal-vesicle pressure, or a pelvic fullness/heaviness pattern**. This is one of the clearest examples of why the questionnaire separates sexual activity into more than one mechanistic model.

Regular physical activity

Improvement with regular physical activity usually supports a phenotype in which the body benefits from **circulatory normalization, reduced static compression, improved movement variability, and lower muscular guarding**. This may indicate that the patient's system is harmed more by immobility and prolonged static postures than by movement itself. It can also support a psychophysiological model in which movement reduces threat-focus and improves autonomic regulation. However, the interpretation depends on the rest of the survey: if "intense physical activity" worsens symptoms elsewhere, then the useful dose may be **moderate, regular, non-overloading movement** rather than strenuous exercise.

Loose underwear / clothing

If loose clothing helps, this suggests a **pressure-sensitive phenotype**. That may reflect local perineal or genital hypersensitivity, allodynia, a pudendal-territory pain pattern, or in some patients a more **mast-cell/sensory-reactive phenotype** in which tight contact itself amplifies symptoms. This answer becomes particularly meaningful when paired with **tight clothing worsening pain, reaction to pressure, dermographism, penile or perineal hypersensitivity, and light touch causing pain**.

U-shaped cushion (perineal relief cushion)

This is one of the clearest phenotype-marking items in the survey. If a perineal relief cushion helps, that strongly supports a **seat-pressure / pudendal / pelvic floor compression phenotype**. The cushion does not treat infection or endocrine disease; it reduces pressure on the same anatomical region that often hurts in sitting-sensitive pelvic pain. Improvement here strongly suggests that the pain is being shaped by **mechanical offloading**.

Sitting slightly sideways (offloading one side)

If sitting off to one side helps, that again strongly supports a **mechanical pressure distribution phenotype**. It suggests that small changes in load placement alter symptoms, which is highly consistent with **perineal compression, pelvic floor asymmetry, pudendal irritation, or unilateral deep tissue sensitivity**. The importance of this item lies in its specificity: improvement from a positional unloading strategy is rarely random.

Meditation / yoga

Improvement with meditation or yoga supports a **psychophysiological / autonomic / pelvic floor down-regulation phenotype**. These interventions often help not only through "stress reduction" in a general sense, but by changing breathing pattern, muscle tone, autonomic balance, and the patient's relationship to pain signals. This becomes especially meaningful in men who report **stress as a major trigger, difficulty relaxing, breath holding, pelvic floor clenching, or symptom worsening when focusing on the body**.

Breathing exercises (diaphragmatic breathing)

If diaphragmatic breathing helps, this strongly supports a **breathing–pelvic floor pressure regulation phenotype**. The diaphragm, abdominal wall, and pelvic floor act as a linked pressure-management system, so poor breathing can maintain pelvic floor tension. Improvement from diaphragmatic breathing suggests that part of the pain is being maintained by **abdominal bracing, upper-chest breathing, breath holding, and impaired down-regulation of the pelvic floor**. This is particularly strong when the patient also endorses chest-dominant breathing, difficulty breathing into the abdomen, or breath holding during stress.

Stress reduction techniques

If general stress-reduction strategies help, that supports a **stress-reactive pain phenotype**. It suggests that symptom generation is being substantially amplified by autonomic activation, pelvic floor recruitment, poor recovery, and attention-related pain intensification. This is especially relevant when stress ranks highly in Point 20 or when the patient reports racing thoughts, anxiety, poor sleep, and stress-linked worsening.

Psychotherapy

Improvement with psychotherapy does **not** imply that the pain was "psychological instead of physical." Rather, it suggests that cognition, fear, trauma, symptom monitoring, stress reactivity, or avoidance patterns were playing a meaningful role in maintaining the syndrome. In chronic pelvic pain, psychotherapy may help reduce catastrophizing, fear of flares, body vigilance, compulsive symptom checking, and autonomic overactivation. If psychotherapy helps, the case for a **psychophysiological maintenance layer** becomes stronger.

Pelvic floor physiotherapy

This is one of the highest-value items in the entire non-pharmacological section. If pelvic floor physiotherapy helps, that is powerful evidence for a **pelvic floor myalgia / myofascial / biomechanical phenotype**. Current AUA and EAU frameworks both strongly emphasize pelvic floor dysfunction in chronic pelvic pain, and the EAU guideline specifically recommends specialized physiotherapy for pelvic floor overactivity and myofascial trigger points. Improvement here suggests the pelvic floor is not just reacting to pain but is likely part of the **active engine** of the pain.

Pelvic floor muscle relaxation training

If specific pelvic floor relaxation training helps, that strengthens the interpretation of a **non-relaxing pelvic floor phenotype**. The EAU guideline explicitly notes that learning pelvic floor relaxation can help interrupt the **pain–spasm–pain cycle**. This item becomes especially meaningful

when the patient reports perineal tension, involuntary clenching, upward pulling, painful contraction, rectal fullness, and constipation-related pelvic pain.

Trigger point therapy

Improvement with trigger point therapy strongly supports a **myofascial trigger-point phenotype**. It suggests that localized hyperirritable points in the pelvic floor or related lumbopelvic muscles are contributing to the pain map, potentially causing both local and referred symptoms. This becomes especially relevant in men with perineal pain, rectal fullness, pain during ejaculation, or hip/groin/lower-back overlap.

Myofascial release

If myofascial release helps, that again supports a **deep soft-tissue restriction / myofascial tension phenotype**. It implies that fascial stiffness, tissue shortening, or chronic holding patterns may be part of what maintains pain. Improvement here is often especially meaningful when sitting, posture, hip tightness, and abdominal-pressure triggers are prominent.

Dry needling

Improvement with dry needling suggests that the syndrome includes a **trigger-point or neuromuscular hyperirritability component**. It is especially consistent with a muscular phenotype in which deep tissue stimulation temporarily resets or reduces abnormal recruitment. This becomes more convincing when paired with pelvic floor spasm, painful contraction, or widespread lumbopelvic tension.

Wise-Anderson protocol

If the Wise-Anderson protocol helps, that strongly supports a **psychophysiological pelvic pain phenotype with pelvic floor overactivity and autonomic amplification**. The protocol is specifically associated with relaxation, paradoxical relaxation, and reduction of chronic pelvic holding. Improvement here is particularly relevant in men with stress sensitivity, symptom monitoring, difficulty relaxing, and pelvic floor tension.

Heat therapy

If heat helps, this often supports a **muscular, spasm-dominant, or autonomic guarding phenotype**. Heat commonly reduces muscular guarding and may improve comfort in tight or overactive soft tissues. Improvement is especially meaningful when cold aggravates symptoms, when pelvic floor spasm is present, or when stress-linked tension dominates.

Cold therapy (cryotherapy)

If cold helps, the interpretation is different. This may suggest a more **acute inflammatory, pressure-sensitive, or flare-based phenotype**, or a patient who responds better to dampening of local sensitivity than to muscle relaxation. Cold helping is less typical of chronic pelvic floor spasm than heat helping, so when present it may point toward a subgroup with **active flare biology** rather than purely chronic muscle holding.

Warm baths

Warm baths helping often supports a **whole-pelvis relaxation phenotype**, especially when the patient's symptoms respond to warmth, reduced guarding, and environmental safety. They may help muscular, autonomic, and pelvic floor components simultaneously, and their effect becomes especially meaningful when sitting, stress, and pelvic floor spasm are prominent.

Sauna

If sauna helps, that may suggest benefit from **whole-body relaxation, increased circulation, autonomic down-regulation, or muscular decompression**. However, interpretation should remain individualized, because some patients with vascular heaviness or heat sensitivity worsen with heat. If sauna helps while heat exposure worsens symptoms elsewhere, the patient may have a more complex mixed phenotype.

Stretching

Improvement with stretching supports a **shortened muscle / myofascial restriction phenotype**, especially if the patient also reports pain on stretching pelvic muscles, hip tightness, lower-back stiffness, and movement-related triggers. The EAU guideline specifically notes that when muscles are shortened, **relaxation alone is not enough and stretching is needed to regain length and function**.

Ergonomic chair and desk

If ergonomics help, the likely interpretation is a **postural and load-distribution phenotype**. This suggests that the pelvis responds not only to total sitting time but to the way sitting and work posture are mechanically organized. Improvement here supports a **sitting-mechanics and lumbopelvic alignment component**.

Nerve blocks

If nerve blocks help, that strongly supports a **neural or mixed neuromechanical phenotype**. Relief after a nerve-targeted intervention suggests that peripheral neural input is an important contributor to symptom generation or maintenance. This becomes especially meaningful when the patient has burning, electric-shock sensations, radiating pain, allodynia, or pudendal-territory pain.

Pelvic floor botox injections

If pelvic floor botox helps, that is strong evidence for a **pelvic floor overactivity / spasm-driven phenotype**. Botox is not a general calming treatment; it specifically reduces muscular overactivity. Therefore, improvement suggests that excessive pelvic floor contraction or non-relaxation is a major part of the syndrome.

Shockwave therapy

If shockwave therapy helps, that may support a **myofascial, chronic pain modulation, or tissue-irritability phenotype**. Contemporary AUA commentary notes that low-intensity extracorporeal shockwave therapy has been reviewed in the male chronic pelvic pain guideline context, reflecting growing interest in it as part of multimodal care.

TENS (electrical stimulation)

Improvement with TENS suggests that the pain may be modifiable by **peripheral neuromodulation**, which can support a **neuropathic, myofascial, or mixed pain-processing**

phenotype. It suggests that changing afferent signaling changes symptoms, which is clinically important in chronic pain phenotyping.

Low-histamine diet

If a low-histamine diet helps, this is one of the strongest clues to a **histamine-reactive / immune-amplified pelvic phenotype**. This becomes especially important when paired with alcohol sensitivity, fermented-food sensitivity, histamine-rich food sensitivity, flushing, hives, itching, dermographism, pressure reactivity, or antihistamine benefit. In such cases, pelvic pain may be occurring within a broader **bladder–pelvic floor–immune interface**.

Gluten-free diet

If a gluten-free diet helps, that supports a **gut-reactive phenotype**. This does not prove celiac disease, but it suggests that gluten-related intestinal or immune effects may be amplifying pelvic symptoms. It becomes especially meaningful with bloating, IBS, food intolerances, positive celiac-related testing, or food-triggered pelvic pain.

Learning to relax pelvic floor muscles

This is closely related to pelvic floor relaxation training, but its wording emphasizes the patient's **acquired self-regulation skill**. If this helps, then the syndrome is likely highly dependent on the patient's ability to consciously reduce pelvic guarding. That strongly supports a **self-amplifying pelvic floor tension phenotype** rather than a pain condition entirely outside voluntary influence.

Prostate massage

If prostate massage helps, that may support a **deep pressure/congestion or peri-prostatic phenotype**. Interpretation must remain cautious, because it is not a universally recommended or universally beneficial intervention. But in some patients it may suggest that decompression or drainage of deep pelvic structures changes symptoms. If it helps alongside relief after ejaculation and abstinence worsening, the case for a **congestion-sensitive deep pelvic phenotype** becomes more plausible.

CBD (cannabidiol)

If CBD helps, that may support a more **neuro-regulatory, sensory-amplification, or stress-reactive phenotype**, particularly if the patient also has sleep problems, anxiety, autonomic overactivation, or widespread sensory intensity. The interpretation should remain cautious and symptom-based rather than categorical, since evidence varies and clinical responses are heterogeneous.

Low-FODMAP diet

If low-FODMAP helps, that strongly supports a **fermentation-sensitive gut–pelvis phenotype**. It becomes especially meaningful in patients with bloating, IBS, food-triggered pelvic pain, constipation/diarrhea shifts, and sensitivity to fermentable carbohydrates. In such cases the gut may be contributing mechanically, immunologically, and viscerally to pelvic symptoms.

Low-Level Laser Therapy (LLLT)

Low-level laser therapy (LLLT), also known as photobiomodulation, is a non-invasive physiotherapeutic modality that uses low-intensity light, typically in the red or near-infrared spectrum, to modulate cellular activity and support tissue recovery. It is applied in selected patients with chronic prostatitis/chronic pelvic pain syndrome (CP/CPPS), particularly as part of a multimodal treatment strategy targeting both peripheral and central mechanisms of pain.

The biological effects of LLLT are multifactorial. At the local level, it may reduce inflammation through modulation of cytokine signaling and improvement of microcirculation, leading to enhanced tissue oxygenation and metabolic exchange. In addition, photobiomodulation influences neuronal activity by reducing peripheral nerve sensitization and nociceptive signaling. This is particularly relevant in the context of neurogenic inflammation, where activated nerve endings release mediators such as substance P and calcitonin gene-related peptide (CGRP), contributing to persistent pain, hypersensitivity, and amplification of local inflammatory responses.

From a neuroimmune perspective, LLLT may also interact with pathways involved in mast cell activation and histamine-mediated responses. Although clinical evidence in this specific domain is still emerging, experimental and translational data suggest that photobiomodulation may reduce the release of pro-inflammatory mediators and contribute to stabilization of local immune activity. In patients presenting with an MCAS-like or histamine-reactive phenotype, characterized by burning pain, urgency, pressure, and sensitivity to triggers such as stress, diet, or mechanical stimuli, LLLT may provide indirect therapeutic benefit by modulating the interaction between the nervous system and immune mediators.

Within the context of this questionnaire, a positive response to LLLT may therefore indicate the presence of local inflammatory or post-inflammatory processes, neurogenic sensitization, microcirculatory dysfunction, and in some cases neuroimmune or mast cell–related mechanisms. This modality may be particularly relevant in patients presenting with localized pelvic or perineal pain, myofascial tension, burning or hypersensitive pain patterns, or features suggesting impaired tissue recovery.

As with other therapeutic interventions in CP/CPPS, the clinical response to LLLT is variable and should be interpreted within the broader clinical context as part of a phenotype-based, multimodal approach, rather than as a standalone diagnostic indicator.

Summary of Point 22

Point 22 is the questionnaire's **non-pharmacological response-as-diagnosis section**. Its purpose is to identify **which non-drug interventions actually change symptoms**, and therefore which mechanisms are most likely active. Improvement with **reducing sitting, offloading cushions, and side-sitting** supports a mechanical-compressive / pudendal phenotype. Improvement with **pelvic floor physiotherapy, relaxation training, trigger point work, myofascial release, dry needling, stretching, and botox** supports a pelvic floor myofascial phenotype. Improvement with **meditation, breathing exercises, stress reduction, and psychotherapy** supports a psychophysiological/autonomic phenotype. Improvement with **low-histamine diet** supports a histamine-reactive immune phenotype. Improvement with **low-FODMAP or gluten-free diet** supports a gut-reactive phenotype. Improvement with **reduced ejaculation frequency** supports a sexual-overload / poor-recovery phenotype, whereas improvement with **increased ejaculation frequency** supports an abstinence/congestion-sensitive phenotype. In professional interpretation, Point 22 is indispensable because it shows **what the organism actually responds to**, not just what the patient reports feeling.

Point 23. Pharmacological treatments - for some it helps, for others it does not.

(check all that have helped you)

Purpose of Point 23

Point 23 is one of the strongest phenotype-defining sections in the entire questionnaire because it treats **medication response as a biological challenge test**. In chronic pelvic pain, the question is not only what symptoms the patient has, but **which biological pathway becomes quieter when a particular drug class is used**. Relief from an alpha-blocker, a neuromodulator, a muscle relaxant, a steroid, a PDE5 inhibitor, an antihistamine, or a bladder-directed drug does not automatically diagnose one disease, but it often provides one of the clearest clues to the **dominant active mechanism**.

This section is especially important because modern chronic pelvic pain care is **multimodal and mechanism-oriented**. The 2025 AUA male chronic pelvic pain guideline and the EAU chronic pelvic pain guideline both support broad phenotyping rather than reflexively assuming one organ-based cause. Medication response fits directly into that logic. For example, if a patient improves with alpha-blockers and has weak stream and hesitancy, the smooth-muscle / outlet phenotype becomes more plausible. If he improves with pregabalin or gabapentin and also has burning, allodynia, and radiating pain, a neuropathic phenotype becomes more likely. If he improves with hydroxyzine, H1 blockers, or H2 blockers and also has histamine-rich food sensitivity, bladder pain, and flushing, a **histamine-amplified bladder/pelvic phenotype** becomes more plausible.

This section is also the place where the newer information in your document about **steroids and antihistamines** matters most. Short-term steroid benefit can be clinically meaningful because it may suggest that, at least in some flares, symptoms include an **inflammatory or post-inflammatory amplification layer**. That does not prove a single inflammatory diagnosis, but it becomes more persuasive when steroid benefit occurs alongside **deep prostate/seminal-vesicle pain, post-ejaculatory delayed flares, inflammatory semen findings, or broader immune-type symptom clusters**. Likewise, antihistamine benefit becomes much more meaningful when it occurs in patients who already have **bladder pain, urgency, histamine-food sensitivity, flushing, hives, dermographism, or pressure-reactive symptoms**. Current EAU and bladder pain guidance recognizes that mast cells and histamine may be relevant in a subset of bladder-pain patients, although evidence for specific antihistamines is mixed and should not be overstated.

Tamsulosin

If tamsulosin helps, that supports a **bladder outlet / smooth-muscle / voiding dysfunction phenotype**. Its benefit becomes especially meaningful when the patient also reports **weak stream, hesitancy, intermittent stream, incomplete emptying, or post-void dribbling**. Relief here suggests that outlet resistance or functional smooth-muscle tension is contributing meaningfully to symptoms. AUA commentary specifically notes that alpha-blockers are most useful in men with pain plus **obstructive voiding symptoms**.

Doxazosin

Doxazosin carries a similar interpretation: improvement supports a **voiding/outlet phenotype** in which alpha-adrenergic tone contributes to symptoms. If doxazosin helps in the setting of urinary hesitancy, weak stream, and pelvic pressure, the phenotype may include an important **smooth-**

muscle/outlet component rather than purely pain amplification.

Terazosin

Terazosin helping also supports an **alpha-blocker-responsive outlet phenotype**. In current evidence, alpha-blockers are not a universal solution for all CPPS patients, but they remain clinically relevant in the subgroup with obstructive or outlet-type symptoms. Therefore, a positive answer has strong interpretive value when matched with the urinary pattern.

Alfuzosin – strongly relaxes smooth muscles

If alfuzosin helps, the likely interpretation is again **smooth-muscle / outlet / pelvic outlet tension involvement**. Because the questionnaire explicitly frames it as strongly relaxing smooth muscle, a positive response is particularly meaningful in men who feel deep pelvic or bladder-neck pressure together with urinary flow symptoms. Relief here can also sometimes overlap with reduction of **deep post-void pelvic pressure**.

Silodosin – most strongly relaxes smooth muscles

If silodosin helps, that again supports a **voiding/smooth-muscle phenotype**, often perhaps more strongly in men whose symptoms cluster tightly around urinary hesitancy, weak stream, and incomplete emptying. In a patient with pain plus clear outlet symptoms, response to silodosin can be one of the most useful pharmacologic clues that the lower urinary tract is a major active contributor.

Pregabalin

If pregabalin helps, that strongly supports a **neuropathic or sensitization phenotype**. Pregabalin is most informative in patients with **burning, electric-shock sensations, tingling, radiating pain, allodynia, hypersensitivity, or pain that persists after triggers end**. Improvement suggests that abnormal neural excitability or amplified sensory processing is contributing substantially to symptoms.

Gabapentin

Gabapentin has a similar interpretation to pregabalin. Relief supports a **neuropathic-pain or central/peripheral sensitization phenotype**, especially in patients with abnormal sensory symptoms rather than only deep pressure or urinary complaints. If both pregabalin and gabapentin help, the neural phenotype becomes especially persuasive.

Amitriptyline

If amitriptyline helps, it often supports a **sensory-amplification, neuropathic, bladder-pain, or sleep-linked chronic pain phenotype**. Amitriptyline has long been used in chronic pain and bladder pain contexts, and the AUA bladder pain guideline lists it among oral medication options. Relief becomes particularly meaningful when burning, urgency, bladder pain, poor sleep, or widespread pain amplification are also present.

Diazepam (oral)

If oral diazepam helps, this often supports a **muscle guarding / anxiety-linked autonomic / generalized relaxation phenotype**. It may reduce pelvic floor recruitment indirectly through

broader anxiolytic and muscle-relaxing effects. Improvement becomes especially relevant when stress, difficulty relaxing, pelvic clenching, and post-trigger spasm are prominent.

Diazepam (rectal suppositories)

If rectal diazepam helps, that gives even stronger support to a **pelvic floor spasm / local muscle overactivity phenotype**, because the route is much more specifically targeted to the pelvic floor region. Relief here is highly consistent with men whose dominant mechanisms include pelvic floor hypertonicity, painful contraction, rectal fullness, and pelvic spasm.

Baclofen

If baclofen helps, that also supports a **muscle-spasm / overactivity phenotype**, especially in men with pelvic floor contraction pain, spasm episodes, or deep muscle-related post-trigger flares. It may also have relevance in patients whose pain is strongly linked to pressure spikes, exertion, or sexual activity that provokes muscular over-recruitment.

NSAIDs

If NSAIDs help, that suggests at least some component of **inflammatory or mechanical-irritative pain**, although the effect is often nonspecific. Relief becomes more meaningful when flares follow exertion, sexual activity, or episodes that plausibly involve inflammatory amplification. However, NSAID response alone is not enough to define a purely inflammatory phenotype.

Steroids (short-term use only)

This is one of the most clinically interesting items in the whole medication section. If short-term steroids help, that suggests that at least some of the patient's symptoms may be **amplified by an inflammatory, post-inflammatory, or neuroimmune component**. Current evidence does not justify treating steroid response as proof of one diagnosis, but it can be a highly valuable clue when interpreted in context. Relief is especially meaningful in patients with **deep prostate/seminal-vesicle pain, pain the day after ejaculation, delayed post-ejaculatory flares, inflammatory semen findings, immune-reactive symptoms, or broader histamine-like symptom clusters**. In such men, steroid response may indicate that symptoms are not only mechanical or neural, but that **inflammatory amplification is biologically relevant during at least some flare states**.

The questionnaire's wording "short-term use only" is clinically appropriate, because steroids are not a general long-term solution for chronic pelvic pain. Their main interpretive value here is **phenotyping**, not universal treatment recommendation.

Tadalafil

If tadalafil helps, the likely interpretation is broader than erectile function alone. It may support a phenotype involving **pelvic smooth muscle relaxation, vascular unloading, better deep pelvic perfusion, or improved tolerance of bladder/outlet symptoms**. Relief becomes especially meaningful in patients with **pelvic heaviness, pressure/fullness, urinary symptoms, erectile dysfunction, or deep post-ejaculatory discomfort**. In some men, tadalafil may fit a **vascular–smooth muscle–deep pelvic pressure phenotype**.

Antihistamines

If “antihistamines” in general help, this is one of the strongest clues to a **histamine-reactive, bladder-sensory, or mast-cell–linked phenotype**. This becomes especially important if the patient also reports **alcohol sensitivity, histamine-rich food sensitivity, fermented-food sensitivity, bladder pain, urgency, urethral burning, flushing, hives, itching, dermographism, or pressure-triggered symptoms**. Current EAU guidance discusses mast cells and histamine receptor antagonists in bladder pain syndrome, while emphasizing that evidence is variable. That makes antihistamine benefit a **clinically valuable phenotype clue**, not definitive proof.

Hydroxyzine

If hydroxyzine helps, the interpretation is similar but more specific. Hydroxyzine is especially relevant in **bladder-pain / histamine-reactive / nighttime symptom / anxiety-overlap phenotypes**. Current bladder pain guidance includes hydroxyzine among oral medication options, while evidence remains mixed and not uniformly positive in trials. Therefore, benefit in an individual patient is clinically meaningful when the rest of the questionnaire supports a histamine-reactive phenotype.

H1 antihistamines: Bilastine / Cetirizine / Fexofenadine

If any of the specific H1 blockers help, that strongly supports a **histamine-mediated symptom component**, particularly when pelvic pain overlaps with bladder pain, urgency, skin symptoms, and histamine-food triggers. The fact that the survey separates specific H1 agents is clinically useful, because it allows recognition that some patients respond to one H1 blocker better than another. In a patient with a consistent histamine pattern, relief from these drugs strengthens the interpretation of a **bladder–pelvic floor–immune phenotype**.

However, it should be remembered that long-term use of these drugs may cause the regenerated nerves to more easily feel post-inflammatory changes in the surrounding tissues, which may intensify pain symptoms.

Intravenous or topical ketamine

If ketamine preparations help, that strongly supports a **pain-modulation / sensitization / neuropathic phenotype**. Ketamine is especially interpretable in patients with burning, allodynia, pain spread, disproportionate pain, and persistent post-trigger pain. Relief suggests that the pain is heavily shaped by abnormal **central or peripheral sensitization mechanisms** rather than only structural irritation.

Intravenous or topical Lidocaine medications

If lidocaine rectal preparations help, the likely interpretation is a **local peripheral neural / sensory / pelvic floor phenotype**. Relief suggests that peripheral afferent input from the pelvic outlet or pelvic floor is an important contributor to the pain. This becomes particularly meaningful in men with rectal fullness, anorectal pain, perineal burning, or pelvic floor spasm.

Pentosan polysulfate (Elmiron)

If pentosan polysulfate helps, that supports a **bladder-pain / urothelial barrier / bladder-sensory phenotype**. This becomes especially meaningful when the patient has pain on bladder filling, urgency, suprapubic pain, relief after voiding, or an interstitial cystitis/bladder pain diagnosis. Pentosan polysulfate remains one of the classic oral options discussed in bladder pain guidance.

DMSO rectal medications

If DMSO-type rectal therapies help, that may support a **local anti-inflammatory / sensory-modulating pelvic phenotype**, though the exact interpretation depends on the broader symptom pattern. It becomes more meaningful when deep pelvic, anorectal, or bladder-adjacent symptoms are prominent.

LDN

If low-dose naltrexone helps, that can support a **neuroimmune / pain-regulation / central-amplification phenotype**. While not specific to one CPPS mechanism, LDN relief becomes particularly interesting in patients with mixed **immune-reactive symptoms, widespread reactivity, sensory amplification, poor recovery after triggers, and cyclic flare patterns**. In that setting it may suggest that the pain system is being maintained partly through **neuroimmune amplification** rather than only structural irritation.

H2 blockers

If H2 blockers help, that strongly strengthens a **histamine-related interpretation**, particularly when combined with H1 benefit, histamine-food sensitivity, bladder pain, flushing, or GI overlap. Current bladder pain literature notes that H2 receptor antagonists such as cimetidine can improve symptoms in some bladder-pain patients, while evidence remains limited and context-dependent. Therefore, H2 benefit in this survey has significant phenotype value.

Summary of Point 23

Point 23 is the questionnaire's **medication-response-as-biological-test section**. Its purpose is to determine **which pharmacologic pathway, when modulated, actually reduces pain**, and therefore which mechanism is most likely active. Relief from **alpha-blockers** supports a smooth-muscle / bladder outlet phenotype. Relief from **pregabalin, gabapentin, ketamine, lidocaine, and sometimes amitriptyline** supports a neuropathic or sensitization phenotype. Relief from **diazepam and baclofen** supports a pelvic floor spasm / muscle overactivity phenotype. Relief from **NSAIDs and especially short-course steroids** supports at least some inflammatory or post-inflammatory amplification, especially when deep ejaculatory or immune-reactive symptoms are present. Relief from **tadalafil** supports a vascular–smooth muscle–deep pelvic pressure phenotype. Relief from **antihistamines, hydroxyzine, specific H1 blockers, and H2 blockers** strongly supports a histamine-reactive bladder/pelvic phenotype, especially when paired with food, skin, and pressure-triggered symptoms. Relief from **Elmiron** supports a bladder-pain phenotype. Relief from **LDN** supports a neuroimmune pain-regulation phenotype. In professional interpretation, Point 23 is one of the most powerful parts of the questionnaire because it shows **how the organism responds when a specific biological axis is pharmacologically challenged**.

Point 24. SUPPLEMENTS for some it helps, for others it does not.

check all that have helped you.

Purpose of Point 24

Point 24 is designed to determine whether **non-prescription biological modifiers** change the patient's symptoms in a way that reveals the dominant active phenotype. In chronic pelvic pain, supplement response is not merely anecdotal noise. It can provide clinically useful clues about whether symptoms are being driven more strongly by **inflammation, neuroimmune amplification,**

histamine sensitivity, pelvic floor tension, bladder reactivity, gut dysbiosis, oxidative stress, or neuropathic pain mechanisms. The AUA and EAU frameworks both support a phenotype-based approach, and supplement response fits naturally into that logic when interpreted cautiously and in context.

This section is especially important because the supplements listed are **not functionally interchangeable**. Improvement after quercetin does not mean the same thing as improvement after magnesium, DAO, probiotics, or L-arginine. Each positive response shifts the interpretation toward a somewhat different biological layer. The key rule is that supplement benefit should never be treated as a stand-alone diagnosis. Rather, it becomes meaningful when it matches the rest of the questionnaire. For example, benefit from quercetin, luteolin, DAO, vitamin C, and low-histamine diet is much more persuasive in a patient who also has **flushing, hives, dermographism, bladder pain, alcohol sensitivity, and histamine-rich food triggers**. Likewise, benefit from PEA or acetyl-L-carnitine becomes more meaningful in a patient with **burning, electric-shock sensations, allodynia, or pain spread**.

This section is also the place where the newer medical knowledge must be handled carefully. Some supplements have better support than others. For example, current evidence suggests that **quercetin has meaningful supportive evidence in CP/CPPS**, while **saw palmetto has not shown clear significant benefit in systematic review-level evidence for CP/CPPS**, and NCCIH states there is not enough evidence for most uses beyond BPH, with a 2022 review not finding a significant benefit for CP/CPPS. Pollen extract has supportive trial data, including newer 2025 clinical data, but evidence is still not uniform enough to justify treating it as universally effective. PEA has growing evidence in pain modulation and some encouraging CP/CPPS-related data, but again should be interpreted as a phenotype clue rather than a guaranteed treatment.

Saw palmetto

If saw palmetto helps, the most likely interpretation is that the patient may have a **prostate-/lower urinary tract–adjacent phenotype**, especially one involving mild voiding symptoms, deep pelvic pressure, or a symptom picture the patient perceives as “prostate-related.” However, this interpretation must be cautious. NCCIH states there is **not enough evidence** to determine usefulness of saw palmetto for most health purposes beyond BPH, and notes that a 2022 review in CP/CPPS did **not** find significant benefit. That means a positive individual response may still be real, but it should be interpreted as **patient-specific**, not as strong proof of a classic saw-palmetto-responsive disease model.

If a patient reports benefit from saw palmetto together with **weak stream, hesitancy, incomplete emptying, deep pelvic pressure, and alpha-blocker responsiveness**, then the supplement may be marking a **prostate/outlet-adjacent phenotype**. But if the broader questionnaire is dominated by neuropathic burning, histamine symptoms, or severe sitting intolerance, saw palmetto benefit would be less central to interpretation.

African Plum

African plum is generally used in a phytotherapeutic context for **urinary/prostate-type symptoms**, so if it helps, the interpretation is broadly similar to saw palmetto: a possible **deep pelvic / urinary / prostate-adjacent phenotype**. Its meaning is strongest if the patient also reports urinary flow symptoms, pelvic heaviness, deep pressure behind the testicles, or post-void discomfort. Because evidence in CP/CPPS is more limited and less standardized than for better-studied interventions, benefit should be treated as **suggestive rather than definitive**.

Quercetin

Quercetin is one of the most clinically interesting items in the supplement section because it sits at the intersection of **anti-inflammatory, antioxidant, and mast-cell / histamine-modulating interpretations**. A recent review and meta-analytic summary reported significant improvement in NIH-CPSI scores compared with placebo, suggesting that quercetin has some of the strongest supplement-level support among nutraceuticals discussed in CP/CPPS. If quercetin helps, it may indicate that the patient has a relevant **inflammatory, neuroimmune, or mast-cell–amplified layer**.

This becomes especially meaningful when quercetin benefit appears alongside **histamine-rich food sensitivity, alcohol flares, flushing, hives, dermographism, bladder hypersensitivity, urgency, or benefit from antihistamines or hydroxyzine**. In such a patient, quercetin may be functioning as a marker of a **histamine-reactive / neuroimmune pelvic phenotype** rather than just a generic antioxidant response. It can also be relevant in patients with post-ejaculatory delayed flares or deep pelvic inflammatory-feeling discomfort, especially if short steroid benefit elsewhere suggests an inflammatory amplification layer.

PEA (palmitoylethanolamide)

PEA is especially important because it often points toward a **neuroimmune, neuropathic, or chronic pain-modulation phenotype**. Recent reviews indicate that PEA has potential in chronic and neuropathic pain, and CP/CPPS-related studies suggest possible improvement in pain and urinary symptom domains in some patients. If PEA helps, the patient may have a symptom pattern shaped by **microglial/neuroimmune amplification, peripheral nerve irritability, or mixed inflammatory-neuropathic signaling**.

This becomes particularly meaningful when the rest of the survey shows **burning pain, electric-shock sensations, allodynia, pain spread, delayed post-trigger flares, or a poor-recovery pain pattern**. If PEA helps in a patient who also reports benefit from LDN, ketamine/lidocaine preparations, or steroids, that can further support a **neuroimmune-amplified phenotype**, though not any single diagnosis.

Pollen Extract (Cernilton / Graminex)

Bee pollen extract is one of the better-known phytotherapy options in chronic prostatitis/chronic pelvic pain literature. Clinical studies, including newer 2025 data, suggest that standardized pollen extract can improve symptoms in some patients, sometimes alone and sometimes with an alpha-blocker. If pollen extract helps, it may indicate a **prostate pain / deep pelvic inflammatory-feeling / urinary overlap phenotype**, especially in men with perineal pain, urinary symptoms, or post-ejaculatory discomfort.

This item becomes more persuasive when paired with **deep pelvic ache, seminal-vesicle or prostate-region discomfort, urinary symptoms, or partial anti-inflammatory responsiveness**. It does not prove infection or classic prostatitis, but it may support a **deep pelvic phytotherapy-responsive phenotype**.

Omega-3

If omega-3 helps, the interpretation often leans toward a more general **anti-inflammatory or systemic regulatory phenotype**. Omega-3 response is usually less specific than quercetin or DAO response. It may suggest that low-grade inflammatory amplification, systemic stress physiology, or tissue irritability contributes to symptoms. Benefit is often more convincing when the patient also reports inflammatory-feeling flare patterns, broader body pain, or immune-reactive features.

Curcumin

Curcumin helping may indicate an **anti-inflammatory-responsive phenotype**, especially where symptoms have a flare-based, inflammatory, or deep aching component rather than purely neuropathic burning. A 2025 report involving curcumin/quercetin-based combination therapy in chronic prostate pain/PPPS reflects continuing interest in this pathway, although combination designs limit certainty about curcumin alone. Benefit becomes more meaningful if the patient also shows food or immune-reactive aggravation, delayed post-ejaculatory pain, or partial steroid responsiveness.

Zinc

If zinc helps, interpretation is usually more cautious. Zinc may be relevant in men who view their symptoms through a **prostate or seminal-fluid health lens**, but by itself it is not a strongly defining CP/CPPS phenotype marker. A positive response is most meaningful if it appears together with **reproductive/ejaculatory symptoms** or broader supplement response in a deep pelvic phenotype

Vitamin C

Vitamin C becomes especially interesting in patients with a possible **histamine-reactive or mast-cell–amplified phenotype**, because it is sometimes used in that context. If it helps, and the patient also reports **flushing, hives, itching, histamine-food sensitivity, antihistamine benefit, or bladder-sensory symptoms**, then vitamin C may be marking an **immune-mediator-sensitive pelvic pain profile**. Otherwise, it remains a more general supportive antioxidant clue.

Magnesium

If magnesium helps, the strongest interpretation is usually a **muscle-tension, spasm, or autonomic-overactivation phenotype**. Magnesium benefit is especially meaningful in patients with **pelvic floor tension, involuntary clenching, muscle spasm episodes, breath holding, sleep difficulty, and stress-linked worsening**. In such cases, magnesium may be acting as a marker of a **pelvic floor hypertonicity / poor down-regulation phenotype** rather than a gland-centered one.

Ashwagandha

If ashwagandha helps, the interpretation often points toward a **stress-reactive, autonomic, sleep-sensitive phenotype**. This becomes especially relevant when the patient ranks stress highly, has racing thoughts, poor relaxation, sleep problems, and symptom worsening during psychological strain. Improvement here suggests that lowering general arousal may reduce symptom amplification.

L-theanine

L-theanine response has a similar interpretive logic to ashwagandha, but perhaps with more emphasis on **anxiety reduction, mental quieting, and autonomic settling**. If it helps, this supports a phenotype in which **stress physiology and mental overactivation** are materially contributing to pain intensity or flare susceptibility.

Probiotics

If probiotics help, that strongly supports a **gut–pelvis axis phenotype**, especially if the patient also reports **bloating, IBS, bowel-movement-related symptom shifts, food-triggered pelvic pain, or a history of antibiotic-related worsening**. Emerging research continues to support investigation of microbiome changes in CP/CPPS, even though the field is still evolving and not every probiotic response maps neatly onto one diagnosis.

DAO supplements

DAO supplementation helping is one of the strongest clues to a **histamine-sensitive phenotype**. It becomes especially meaningful when paired with **suspected histamine intolerance, fermented-food sensitivity, alcohol worsening, flushing, hives, itching, dermographism, or antihistamine benefit**. In that context, DAO response strongly supports the idea that symptom flares are partly linked to **histamine load and poor histamine handling**, with pelvic symptoms possibly mediated through bladder, urethral, bowel, and sensory pathways.

Pumpkin and black seed oil

If this combination helps, the interpretation is usually more **supportive and nonspecific**, but it may lean toward a **deep urinary/prostate-comfort or low-grade inflammatory phenotype** in some patients. It becomes more meaningful if improvement is modest but reproducible and occurs in a broader prostate/urinary symptom cluster.

NAC

NAC helping may suggest a phenotype involving **oxidative stress, inflammatory amplification, mucus/secretory regulation, or neuroimmune modulation**, but interpretation should remain cautious. It becomes more meaningful when paired with immune-reactive symptoms, delayed flare patterns, or broader inflammatory-feeling symptom episodes.

Luteolin

Luteolin is particularly interesting in the context of **mast-cell or mediator-related hypotheses**. If it helps, especially alongside quercetin, DAO, antihistamines, and low-histamine diet, that strengthens the case for a **histamine-amplified or neuroimmune-reactive phenotype**.

Boswellia serrata

If Boswellia helps, the likely interpretation is an **anti-inflammatory-responsive phenotype**, particularly in patients with deep aching, flare-based pain, or tissue-irritability symptoms. Boswellia has been studied in pelvic pain-related contexts, though evidence remains limited and not sufficiently specific to define one CP/CPPS subtype on its own. It becomes more meaningful when the patient also reports partial NSAID or steroid responsiveness.

L-arginine

If L-arginine helps, the interpretation often shifts toward a **vascular or perfusion-related phenotype**, especially if the patient also has **pelvic heaviness, fullness, deep pressure, erectile dysfunction, or benefit from tadalafil**. In such cases, improved vascular tone or perfusion may be clinically relevant.

Epilobium parviflorum

If Epilobium helps, the interpretation often leans toward a **prostate-/deep pelvic anti-inflammatory phytotherapy phenotype**. Its meaning becomes stronger when paired with deep pelvic pressure, ejaculatory discomfort, or other prostate-perceived symptoms, though robust CP/CPPS-specific evidence remains limited. A 2024 observational study involving a PEA/Epilobium/Calendula combination suggests interest in this space, but combination designs limit certainty about single-agent effects.

Acetyl L-carnitine

If acetyl L-carnitine helps, that may suggest a **neuropathic or nerve-support phenotype**, especially in patients with burning, tingling, radiating pain, or a sensory-predominant pattern. It becomes more meaningful when paired with PEA, pregabalin/gabapentin benefit, or other evidence of nerve-related pain.

Palmitoylethanolamide, Epilobium and Calendula Suppositories

This combination therapy, administered as rectal suppositories, includes Palmitoylethanolamide (PEA), Epilobium (willowherb), and Calendula (marigold). It is designed to act locally within the pelvic region and is used in some patients with chronic prostatitis/chronic pelvic pain syndrome (CP/CPPS), particularly when symptoms are persistent and non-bacterial in origin. Each component targets a different mechanism: Palmitoylethanolamide (PEA) modulates neuroinflammation and may reduce nerve sensitization and chronic pain signaling; Epilobium exhibits anti-inflammatory effects and is traditionally associated with relief of prostate-related symptoms; and Calendula supports tissue repair while providing local anti-inflammatory and regenerative properties. The rectal route allows for direct delivery of active compounds in close proximity to the prostate, seminal vesicles, and pelvic floor structures, which may enhance local therapeutic effects.

In the context of this questionnaire, improvement with this therapy may suggest a combined phenotype involving neurogenic inflammation, deep pelvic (prostate and seminal vesicle) involvement, and local inflammatory or tissue-irritative processes. This pattern may be particularly relevant in patients reporting deep pelvic or prostate-region pain, post-ejaculatory discomfort, persistent symptoms despite negative microbiological findings, or features indicating chronic irritation and impaired tissue recovery. As with all interventions in CP/CPPS, the clinical response is variable and should be interpreted within the broader context of a multimodal, phenotype-based approach rather than as a standalone diagnostic indicator.

Summary of Point 24

Point 24 is the questionnaire's **supplement-response phenotype section**. Its purpose is to identify whether symptom improvement occurs when the patient modulates **inflammatory, neuroimmune, histamine-related, musculoskeletal, gut-related, vascular, or neuropathic pathways** through supplements. Benefit from **quercetin, luteolin, DAO, vitamin C, and low-histamine strategies** strongly supports a histamine-reactive / neuroimmune phenotype. Benefit from **PEA and acetyl L-carnitine** supports a neuroimmune or neuropathic phenotype. Benefit from **bee pollen extract** supports a deep pelvic/prostate-adjacent phytotherapy-responsive phenotype. Benefit from **probiotics or low-FODMAP-compatible supplements** supports a gut–pelvis phenotype. Benefit from **magnesium, ashwagandha, and L-theanine** supports a tension/autonomic/stress-reactive phenotype. Benefit from **L-arginine** supports a vascular or perfusion-related layer. In professional interpretation, Point 24 is valuable because it reveals **which non-prescription biological pathway seems to matter most for that specific patient**, while still requiring cautious, phenotype-based interpretation rather than overclaiming efficacy.

Point 25. Other medications used by CPPS patients for other diagnosed diseases (e.g., cholesterol - statins).

Mark if you also feel worse after using them

Purpose of Point 25

Point 25 is one of the most diagnostically powerful sections in the entire questionnaire because it uses **negative pharmacologic response as a phenotype clue**. In chronic pelvic pain, what worsens symptoms is often just as informative as what relieves them. If a specific drug class consistently aggravates symptoms, that can reveal which biological pathway is **fragile, overreactive, or poorly tolerated** in that patient. This is entirely consistent with modern phenotype-based chronic pelvic pain care, which treats the syndrome as a network of interacting systems rather than a single organ disorder.

This section is especially important because the "worsening" items do not all point in the same direction. Some suggest **outlet/smooth-muscle vulnerability**, others **serotonergic or autonomic sensitivity**, others **hormonal/prostate-axis reactivity**, others **histamine release or bladder irritation**, and others **gut-related or metabolic irritative effects**. A patient who worsens on pseudoephedrine and anticholinergics has a very different clinical picture from a patient who worsens on testosterone, finasteride, or acidic-urine–inducing agents. The section therefore helps define not only what the patient has, but what his system appears to **dislike biologically**.

This section is also where the newer document-based information about medications is particularly relevant. For example, if the patient worsens on agents that can provoke **histamine release**, and also benefits from antihistamines and reacts to high-histamine foods, that strongly supports a histamine-reactive phenotype. If he worsens on testosterone or anabolic precursors and has deep ejaculatory/prostate-region pain, then a hormone-sensitive deep pelvic phenotype becomes more plausible. If he worsens on serotonergic antidepressants and also has strong sensory amplification, autonomic symptoms, and pelvic floor guarding, that may suggest a **sensitive serotonergic-autonomic pain-regulation profile**, even though these drugs help some other pain patients. Point 25 is therefore about **individual reactivity**, not universal drug rules.

Decongestants (e.g., pseudoephedrine)

If decongestants worsen pain, that strongly supports an **outlet/smooth-muscle/urinary phenotype**. Pseudoephedrine can increase smooth-muscle tone and worsen voiding in susceptible people, so aggravation becomes especially meaningful when the patient also has **hesitancy, weak stream, incomplete emptying, retention episodes, or bladder-neck/outlet sensitivity**. In such a case, worsening does not just mean the patient "felt bad on the drug"; it suggests that increasing adrenergic tone destabilizes the pelvic/urinary system.

Anticholinergic drugs

If anticholinergic drugs worsen symptoms, the interpretation may again point toward **emptying difficulty, outlet dysfunction, retention vulnerability, or bladder–pelvic discoordination**. This is especially important when paired with urinary hesitancy, incomplete emptying, and pelvic

pressure. In some patients, anticholinergics may worsen the sensation of not emptying or increase post-void discomfort.

SSRI antidepressants (e.g., sertraline)

If SSRIs worsen pain, the interpretation is more complex. This may suggest a **serotonergic sensitivity in pain regulation, autonomic reactivity, sexual side-effect vulnerability, or worsening of pelvic awareness/tension** in that specific patient. It does **not** mean SSRIs are generally harmful in pelvic pain. Rather, it means that for this person, serotonergic modulation may be interacting badly with sexual function, autonomic tone, muscle tension, or sensory amplification. This becomes more meaningful if the patient also has **increased anxiety, sleep disturbance, genital sensitivity, or worsening of sexual symptoms**.

SNRI antidepressants

If SNRIs worsen symptoms, a similar logic applies, but with additional possible relevance to **noradrenergic/autonomic activation**. In a patient with palpitations, tension, sweating changes, or a strong psychophysiological pelvic phenotype, worsening on SNRIs may suggest that the system is particularly sensitive to increased arousal or altered pain-regulation tone. Again, this is an individualized clue, not a universal rule.

Long-term Amitriptyline (in some cases)

This item is especially nuanced because amitriptyline can help some chronic pain and bladder-pain patients, yet worsen or complicate symptoms in others, especially over time. If long-term amitriptyline worsens pain or overall function, the interpretation may involve **anticholinergic burden, sexual side effects, cognitive dulling, poor tolerability, or changing pain response over time**. The EAU guideline notes amitriptyline can be beneficial in some bladder-pain contexts, but this item reminds us that benefit is not universal.

If worsening occurs in a patient who already has emptying difficulty, low libido, poor erection quality, or strong sensitivity to medication effects, this becomes especially meaningful.

Testosterone therapy

If testosterone therapy worsens pain, this can suggest a **hormone-sensitive deep pelvic phenotype**, especially where symptoms are experienced in the prostate/seminal-vesicle region, after ejaculation, or as deep pressure/fullness. This item is particularly relevant in men with high libido, sexual-triggered symptoms, or symptom onset/worsening after hormonal shifts. Worsening here does not prove a single androgen-mediated mechanism, but it supports the possibility that endocrine stimulation changes the way the deep pelvic system behaves.

Anabolic steroids / hormone precursors

If anabolic steroids or hormone precursors worsen pain, the interpretation again points toward a **hormone-sensitive pelvic phenotype**, potentially with deeper relevance to prostate-region, seminal-vesicle, or sexual-load symptoms. This becomes especially important if the patient also has **high sexual drive, frequent ejaculation worsening, deep post-ejaculatory pain, or fluid/pressure-type symptoms**. In such patients, hormone-driven increases in drive or deep tissue responsiveness may intensify a preexisting **load–recovery mismatch**.

Finasteride

If finasteride worsens symptoms, this may reflect a **hormonal/neurosexual sensitivity phenotype**, especially in men whose pelvic symptoms intertwine with libido change, erectile dysfunction, orgasmic difficulty, or genital sensory change. The interpretation should remain cautious and individualized, but worsening suggests that altering androgen signaling destabilizes the system rather than calming it.

Proton pump inhibitors (e.g., omeprazole)

If PPIs worsen symptoms, the interpretation may point toward a **gut-sensitive, microbiome-sensitive, or medication-reactive phenotype** rather than a purely pelvic-organ process. In some patients, PPI-related worsening may reflect changes in digestion, microbiota, upper-GI state, or broader systemic tolerability. This becomes especially meaningful in men with strong GI overlap, food-triggered flares, or microbiome-sensitive symptom patterns.

Statins

If statins worsen symptoms, a possible interpretation is a **muscle-sensitive or systemic medication-reactive phenotype**, especially if the patient also has lower-back stiffness, hip pain, generalized aching, or exertion-related worsening. Statin aggravation would fit less well with a purely bladder/histamine phenotype and more with a broader pain-sensitivity or muscular irritability picture.

Beta-blockers

If beta-blockers worsen symptoms, interpretation may involve **circulatory, autonomic, sexual, or fatigue-related mechanisms**. In some patients they may worsen cold extremities, erectile symptoms, fatigue, or general bodily tone in ways that indirectly aggravate pelvic pain. This becomes particularly relevant in patients who already endorse **cold hands/feet, dizziness on standing, erectile dysfunction, or autonomic instability**.

Medications triggering histamine release (individual reaction)

This is one of the strongest items in the whole “worsening” section for identifying a **histamine-reactive phenotype**. If histamine-releasing medications worsen symptoms, and the patient also reports **high-histamine food sensitivity, alcohol flares, flushing, hives, itching, dermographism, bladder pain, urgency, or antihistamine benefit**, then the pelvic pain is much more plausibly embedded in a **mediator-sensitive / immune-reactive biological context**. This item is particularly valuable because it tests histamine sensitivity from the opposite direction: not what helps, but what provokes.

Certain supplements or medications causing acidic urine

If acidic-urine–inducing agents worsen symptoms, this strongly supports a **bladder–urethral irritative phenotype**. It suggests that the lower urinary tract is reacting to urine chemistry in a clinically meaningful way. This becomes particularly important if the patient also has **burning urination, glans or urethral burning, bladder filling pain, urgency, citrus/tomato sensitivity, and relief with bladder-calming approaches**. In such patients, pelvic pain may be strongly shaped by **urothelial or urethral sensory irritability**.

Duloxetine

Duloxetine is listed separately from general SNRI antidepressants, which is clinically useful. If duloxetine worsens symptoms, the interpretation may overlap with the SNRI logic but becomes more specific to the patient's actual experience. Worsening may indicate **serotonergic/noradrenergic sensitivity, autonomic overactivation, increased genital awareness, sexual side effects, or poor tolerability in a sensitized pain system**. If the patient also has strong sexual symptoms, bladder sensitivity, or pelvic floor guarding, the worsening may reflect how the drug interacts with that particular pattern rather than a general rule about duloxetine.

Summary of Point 25

Point 25 is the questionnaire's **negative medication-response phenotype section**. Its purpose is to identify which drugs or drug classes make symptoms worse, thereby revealing which physiological systems may be especially **fragile, overreactive, or poorly tolerant** in that patient. Worsening with **decongestants and anticholinergics** supports an outlet/emptying-sensitive phenotype. Worsening with **SSRIs, SNRIs, or duloxetine** supports a serotonergic-autonomic or sexual-function-sensitive phenotype in that patient. Worsening with **testosterone, anabolic precursors, or finasteride** supports a hormone-sensitive deep pelvic/sexual phenotype. Worsening with **histamine-releasing medications** strongly supports an immune-reactive/histamine-sensitive phenotype. Worsening with **acidic-urine–inducing agents** supports a bladder–urethral irritative phenotype. Worsening with **PPIs, statins, or beta-blockers** may reveal broader gut, muscle, circulatory, or autonomic vulnerability. In professional interpretation, Point 25 is extremely important because it shows that chronic pelvic pain is not only about what calms the system, but also about **what predictably destabilizes it pharmacologically**.

Final Overall Synthesis of the Questionnaire

1. What the questionnaire does as a whole

Taken as a whole, the questionnaire builds a **layered clinical map** of chronic pelvic pain. It begins with the patient's age and duration of symptoms, then reconstructs the likely background of the disorder, its onset pattern, anatomical pain distribution, temporal pattern, urinary and sexual involvement, mechanical and positional triggers, vascular clues, breathing and pelvic floor dysfunction, neurologic and autonomic features, psychological and neurodevelopmental traits, immune and histamine-related aggravators, gastrointestinal overlap, trigger hierarchy, objective diagnostic testing, and finally response to both non-pharmacological and pharmacological interventions.

Its purpose is not to force every patient into one diagnosis. Its purpose is to identify the **dominant active mechanisms** that shape symptoms in that specific patient. In clinical reality, most chronic pelvic pain patients are **mixed**, not pure. One man may have a primarily pelvic floor phenotype with a secondary histamine-sensitive bladder component. Another may have a primarily post-ejaculatory deep pelvic phenotype with superimposed central sensitization. Another may have a sitting-dependent pudendal phenotype with bowel overlap and anxiety-driven amplification. The questionnaire is designed to capture precisely that complexity. (auanet.org)

In other words, the questionnaire does not ask, "What single disease does this patient have?" It asks, "**Which systems are involved, which triggers dominate, which mechanisms are likely primary,**

which are secondary, and what profile of chronic pelvic pain is emerging from the full pattern?”

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2. The logic of the full instrument

The first group of questions establishes the **clinical frame**. Age and duration of symptoms do not diagnose anything by themselves, but they shape interpretation. Younger patients more often fit functional, neuromuscular, autonomic, and sensory-amplified patterns, while older patients require broader consideration of outlet, vascular, hormonal, and structural overlap. Longer symptom duration increases the probability that the original trigger is no longer acting alone and that **secondary chronicity mechanisms** such as pelvic floor guarding, central sensitization, sleep disruption, sexual fear, and behavioural adaptation have developed.

The next group of items identifies the **context and onset pathway**. These questions reveal whether the syndrome likely began through infection, sexual overloading, prolonged abstinence, severe stress, prolonged sitting, pelvic injury, spinal dysfunction, bowel disturbance, hormonal exposure, intense exercise, cold exposure, or other destabilizing events. This is clinically important because the initial trigger often points toward the **first system that failed**, even if the current condition is now much broader.

The pain-mapping sections then define the **phenomenology of the syndrome**: where pain is located, when it occurs, what it feels like, whether it is linked to ejaculation or independent of it, whether it follows a pudendal-territory pattern, and whether it is more pressure-like, neuropathic, muscular, vascular, or mixed. These questions are among the most important in the entire questionnaire because they convert vague “pelvic pain” into a much more precise functional picture.

The urinary, sexual, mechanical, vascular, breathing, pelvic floor, neurologic, autonomic, psychological, immune, and gastrointestinal sections then identify **overlap systems**. These are the domains that usually explain why chronic pelvic pain becomes so persistent and variable. Current guideline-based care explicitly recognizes these overlap domains as clinically meaningful. (auanet.org) (uroweb.org)

The final sections on trigger ranking, diagnostic testing, treatment response, supplement response, and medication worsening then transform the questionnaire from descriptive to **mechanistically interpretive**. They reveal what the patient’s body actually reacts to, what has already been investigated, and which biological pathways appear modifiable or vulnerable.

The major patient profiles the questionnaire can generate

A. The Pelvic Floor Myalgia / Hypertonic Pelvic Floor Profile

This is one of the most common and most clinically important profiles the questionnaire can generate. It emerges when the patient shows combinations such as: perineal tension, involuntary pelvic floor clenching, upward-pulling sensations, difficulty relaxing the pelvic floor, pain with contraction or stretching, rectal fullness, constipation-associated pain, bowel-emptying difficulty, pain during ejaculation, sitting-related worsening, pressure sensitivity, and relief from pelvic floor

physiotherapy, trigger point therapy, diaphragmatic breathing, relaxation training, or pelvic floor botox.

In this profile, the pelvic floor is not simply reacting to pain. It is often one of the **main engines of the pain**. It may generate local pain, referred pain into the penis, rectum, perineum, bladder region, or prostate region, and may also create urinary hesitancy, bowel dysfunction, and post-ejaculatory flares. This is fully consistent with current guidance, which recognizes pelvic floor myalgia/dysfunction as a major component of male chronic pelvic pain. (auanews.net) (uroweb.org)

B. The Mechanical / Pudendal / Sitting-Dependent Profile

This profile emerges when pain is concentrated in the perineum, genitals, anus, or pudendal territory and is clearly worsened by **sitting, driving, cycling, leaning forward, seat pressure, or tight clothing**, especially when accompanied by tingling, numbness, electric-shock sensations, or relief with offloading cushions and positional unloading.

This type of patient often describes pain as pressure-sensitive, seat-sensitive, and day-accumulative. The syndrome behaves like a **mechanical compression disorder of a sensitized pelvic system**, often with overlap between pelvic floor tension and pudendal neural irritation. It may coexist with sacral or lumbopelvic features. (auanet.org)

C. The Sexual-Overload / Post-Ejaculatory Recovery Failure Profile

This is one of the most distinctive profiles in the questionnaire. It emerges when the patient reports **pain during ejaculation, pain immediately after, pain hours later, pain the next day, worsening with frequent daily ejaculation, high sexual drive, difficulty relieving sexual tension, seminal vesicle-region pain, reduced semen volume, weak ejaculation, and deep pain behind the testicles/prostate region**.

The medically careful interpretation of this profile is not that ejaculation is inherently harmful in general. It is that, in this patient, **sexual load appears to exceed comfortable recovery capacity** of the pelvic floor–prostate–seminal vesicle–urethral system. In some men this may be mainly a muscular/pelvic-floor phenomenon. In others it may include a deeper inflammatory or post-inflammatory layer, especially if there are **delayed flares, semen inflammatory findings, or partial benefit from short steroid courses**. (auanet.org) (d56bochluxqnz.cloudfront.net)

This is also the profile in which **high libido and frequent ejaculation** become clinically meaningful. The key issue is not morality or generalized advice. It is whether the patient's actual pelvic system appears unable to recover fully between repeated activations.

D. The Abstinence / Congestion-Sensitive Sexual Profile

This is the mirror-image sexual profile. It appears when the patient reports **worsening after several days or weeks of abstinence, relief after ejaculation, pelvic heaviness or fullness, deep pressure in the seminal-vesicle or prostate region, and possibly vascular/heaviness symptoms**.

This pattern suggests that, for this patient, symptoms may be aggravated by **stasis, pressure build-up, unresolved sexual tension, or deep pelvic congestion**, rather than by excessive ejaculation. This is why the questionnaire wisely includes both "reduced ejaculation helps" and "increased ejaculation helps": they identify two **very different sexual phenotypes**.

E. The Bladder Pain / Bladder Hypersensitivity Profile

This profile emerges when the urinary domain is dominant: **frequency, urgency, nocturia, bladder pain, pain on filling, relief after urination, bladder hypersensitivity, suprapubic pain,**

pelvic pressure when the bladder is full, diagnosis of painful bladder syndrome/interstitial cystitis, and food/acid-related bladder irritation.

This profile becomes especially strong when combined with **histamine-rich food sensitivity, alcohol worsening, citrus/tomato sensitivity, antihistamine benefit, hydroxyzine response, H1/H2 blocker benefit, or Elmiron response**. Current bladder pain guidance and EAU chronic pelvic pain guidance recognize that bladder pain can overlap substantially with chronic pelvic pain and may include **mast-cell or histamine-related contributions** in some patients. (auajournals.org) (uroweb.org)

F. The Outlet / Smooth-Muscle / Voiding Dysfunction Profile

This profile appears when urinary symptoms are dominated by **weak stream, intermittent stream, hesitancy, incomplete emptying, post-void dribbling, retention episodes**, and when alpha-blockers such as tamsulosin, alfuzosin, silodosin, or similar agents help. Worsening with decongestants or anticholinergics also strengthens this profile.

This phenotype suggests that the lower urinary tract has a major **functional outlet or smooth-muscle component**, whether through bladder neck tension, pelvic floor discoordination, or true outlet-related symptoms. It often overlaps with pelvic floor dysfunction.

G. The Neuropathic / Sensory-Amplified Profile

This profile emerges when the patient reports **burning pain, electric-shock sensations, tingling, numbness, hypersensitivity of the perineum or penis, pain radiating along nerves, allodynia, pain spread, pain persisting after the stimulus ends, and disproportionate pain relative to findings**.

The profile becomes even stronger when the patient improves with **pregabalin, gabapentin, ketamine, lidocaine, nerve blocks, PEA, or acetyl-L-carnitine**. This suggests that abnormal sensory processing, peripheral nerve irritability, or central/peripheral sensitization is a major part of the syndrome. Current chronic pelvic pain guidance explicitly supports attention to neuropathic and central pain mechanisms. (auanet.org) (uroweb.org)

H. The Central Sensitization / Widespread Amplification Profile

This profile is related to the neuropathic one but broader. It emerges when the patient shows **pain disproportionate to physical findings, spread beyond the original location, allodynia, persistence after stimulus removal, poor sleep, migraine, fibromyalgia, strong symptom monitoring, fear of worsening, catastrophizing, and multi-domain overlap**.

This does not mean the pain is not biologically grounded. It means the nervous system has become a major **amplifier and maintainer** of the syndrome. Such patients often have mixed peripheral and central features, and their symptom severity may exceed what any one local finding would predict. (pubmed.ncbi.nlm.nih.gov)

I. The Psychophysiological / Autonomic Dysregulation Profile

This profile emerges when stress is highly ranked and the patient reports **feeling on edge, difficulty relaxing, chest-dominant breathing, breath holding during stress, inability to fully relax breathing, palpitations, cold hands/feet, sweating abnormalities, dizziness on standing, sleep problems, and symptoms worsening with attention to the body**.

This profile often overlaps strongly with pelvic floor dysfunction because chronic autonomic arousal is one of the most powerful drivers of **pelvic floor recruitment and incomplete down-**

regulation. Improvement with meditation, yoga, diaphragmatic breathing, stress reduction, psychotherapy, and similar interventions strengthens this interpretation. (uroweb.org) (ptsd.va.gov)

J. The Neurodivergent / Sensory-Regulation Vulnerability Profile

This profile emerges when the patient endorses many Point 16 features such as **distractibility, attention difficulty, sensory hypersensitivity, overanalysis of bodily sensations, hyperfocus on symptoms, compulsive or high-drive sexual behavior, social overwhelm, repetitive thoughts, and strong internal awareness of body signals**.

This does not define the pain source, but it defines the **regulatory terrain** in which the pain exists. Such patients may be more vulnerable to symptom amplification, prolonged vigilance, sensory overload, and dysregulated sexual or stress patterns. Recent evidence supports greater atypical sensory processing in ADHD populations, which is relevant to this interpretive layer. (sciencedirect.com)

K. The Histamine-Reactive / MCAS-like / Immune-Reactive Profile

This is one of the most distinctive profiles the questionnaire can identify. It emerges when the patient reports **worsening after alcohol, fermented foods, histamine-rich foods, pressure, irregular sleep, flushing, hives, itching without rash, dermographism, allergic rhinitis, hay fever, asthma, and possible benefit from antihistamines, hydroxyzine, H1 blockers, H2 blockers, low-histamine diet, quercetin, DAO, luteolin, or vitamin C**.

This profile becomes especially important when pelvic symptoms include **bladder pain, urgency, urethral burning, pressure-reactive symptoms, and food-triggered flares**. Current EAU guidance supports considering histamine/mast-cell-related contributions particularly in bladder pain phenotypes, while evidence remains mixed and phenotype-dependent rather than universal. (uroweb.org)

If such a patient also reports **short steroid benefit**, that may strengthen the idea that some flares involve inflammatory or immune amplification, but steroids remain a supportive clue rather than a definitive diagnostic test. (d56bochluxqnz.cloudfront.net)

L. The Gut–Pelvis / IBS / Fermentation-Sensitive Profile

This profile appears when the gastrointestinal domain is strong: **bloating, constipation, diarrhea, IBS, food worsening pelvic pain, gluten sensitivity, dairy sensitivity, FODMAP sensitivity, reflux, nausea during flare-ups, and symptoms changing after bowel movements**.

This profile suggests that the bowel may be influencing pelvic pain through **pressure mechanics, visceral hypersensitivity, fermentation, microbiome-related effects, and gut–pelvis cross-sensitization**. It becomes especially strong when the patient improves with **probiotics, low-FODMAP diet, gluten-free diet, bowel management, or other gut-directed approaches**. Emerging research continues to support possible microbiome links in CP/CPPS. (frontiersin.org)

M. The Vascular / Pelvic Congestion Profile

This profile appears when the patient reports **pelvic heaviness, fullness, pressure, standing-related worsening, evening accumulation, improvement when lying down, visible scrotal varicosities, and sometimes benefit from tadalafil or positional unloading**.

This suggests that the pelvis may be behaving partly like a **venous-loading compartment**, not just a muscular or neurologic system. In some men, this may overlap with deep prostate/seminal-vesicle pressure or with abstinence-sensitive sexual symptoms. The vascular literature increasingly

recognizes male pelvic venous disorders as under-recognized contributors to chronic pelvic pain. (pubmed.ncbi.nlm.nih.gov)

N. The Inflammatory / Post-Inflammatory Deep Pelvic Profile

This is one of the more subtle but important profiles. It emerges when there is evidence of **deep prostate/seminal-vesicle pain, post-ejaculatory delayed flares, semen inflammatory findings, imaging evidence of chronic/post-inflammatory change, and partial benefit from NSAIDs, curcumin, quercetin, Boswellia, or especially brief steroid courses**.

This profile is important because it prevents the false conclusion that all chronic pelvic pain is either "infection" or "purely functional." Some patients may have meaningful **post-inflammatory tissue burden** that coexists with pelvic floor overactivity, neural amplification, or immune reactivity. The questionnaire is strong precisely because it allows these combinations rather than forcing one explanation.

The most important mixed profiles

In real use, the questionnaire will probably identify **mixed phenotypes** more often than pure ones. Some of the most clinically plausible combined profiles include:

The **pelvic floor + psychophysiological profile**, where stress, breath holding, poor relaxation, and pelvic floor clenching all reinforce each other.

The **pelvic floor + pudendal/mechanical profile**, where sitting, driving, cycling, and trigger-point tenderness all point to a compression-sensitive pelvic floor syndrome.

The **sexual-overload + deep inflammatory/post-inflammatory profile**, where high sexual drive, frequent ejaculation, seminal vesicle-region pain, delayed flares, reduced semen volume, and steroid responsiveness suggest that repeated sexual loading is acting on a biologically vulnerable deep ejaculatory system.

The **bladder pain + histamine-reactive profile**, where urgency, bladder pain, food sensitivity, flushing, antihistamine benefit, and pressure sensitivity all fit a bladder–immune–sensory subtype.

The **gut–histamine–pelvic floor profile**, where bloating, IBS, food-triggered pelvic pain, histamine reactivity, and pelvic floor spasm all coexist.

The **neuropathic + central sensitization profile**, where burning, electric pain, allodynia, pain spread, poor sleep, migraine, fibromyalgia, and body vigilance define a high-gain nervous system phenotype.

The **vascular + deep pressure + sexual congestion profile**, where heaviness, fullness, improvement lying down, abstinence worsening, and deep prostate/seminal-vesicle pressure suggest a venous/deep pelvic loading state.

Why the last sections are so important

The final sections of the questionnaire — ranking, diagnostics, treatments, supplements, and medications that worsen symptoms — are what turn the instrument from a descriptive symptom survey into a **clinical phenotype engine**.

The ranking section reveals **which trigger dominates**.

The diagnostics section shows **what has been objectively explored** and prevents simplistic conclusions from either normal or abnormal tests.

The non-pharmacological treatment section reveals what changes when the patient modifies **mechanics, breathing, sitting, stress, sexual pattern, diet, and pelvic floor behavior**.

The medication section reveals which **pharmacologic axis**, when challenged, improves symptoms.

The supplement section adds further biological clues at a lower-intensity or broader-modulation level.

The worsening-medications section reveals which systems are **especially fragile or reactive**.

This is a major strength of the questionnaire. It does not only ask “What hurts?” It asks:
What started it? What keeps it going? What body systems are involved? What triggers are strongest? What improves it? What worsens it? What has already been tested?

That is exactly the kind of structure needed for modern phenotype-based chronic pelvic pain assessment.

Final conclusion

The full 25-point questionnaire creates not one diagnosis, but a set of **structured patient profiles**. These profiles can include:

a pelvic floor overactivity profile,
a mechanical sitting-dependent/pudendal profile,
a sexual-overload post-ejaculatory profile,
an abstinence/congestion-sensitive sexual profile,
a bladder pain/hypersensitivity profile,
a voiding/outlet dysfunction profile,
a neuropathic profile,
a central sensitization profile,
a psychophysiological/autonomic profile,
a neurodivergent sensory-regulation vulnerability profile,
a histamine-reactive/MCAS-like immune profile,
a gut–pelvis/IBS/fermentation-sensitive profile,
a vascular/congestion profile,
and a post-inflammatory deep pelvic profile.

Most importantly, the questionnaire is designed to show **how these profiles overlap**. That is its greatest value. Male chronic pelvic pain is often not one thing. It is usually a **stack of mechanisms**, with one or two dominant and several secondary. This questionnaire is unusually strong because it allows the clinician or researcher to identify that hierarchy rather than forcing a false single-cause answer.

In professional use, the questionnaire can therefore serve as:
a **phenotyping instrument**,
a **clinical interview scaffold**,
a **research stratification tool**,
and an **educational map for patients and clinicians** trying to understand why chronic pelvic pain is so persistent, so variable, and so often misunderstood.

An example of a stepped, multidisciplinary diagnostic pathway for chronic pelvic pain syndrome (CPPS)

In patients presenting with pelvic pain persisting for more than six months, with repeatedly negative microbiological findings and limited response to standard urological management, the diagnostic focus should shift from a purely infectious paradigm toward a **multidimensional, phenotype-based evaluation**. Contemporary understanding of CP/CPPS recognizes it as a complex disorder involving interactions between **neuromuscular, neural, immune, vascular, gastrointestinal, and psychophysiological systems**.

Step 1: Re-evaluation by Urology (Advanced Assessment Phase)

Following unsuccessful initial management, a second-stage urological evaluation should be performed, focusing on exclusion of overlooked structural or functional pathology. This includes:

- Detailed digital rectal examination (including pelvic floor tone assessment)
- Uroflowmetry and post-void residual urine
- Consideration of cystoscopy (if hematuria, severe LUTS, or atypical symptoms)
- Semen analysis with inflammatory markers (leukocytes, cytokines if available)

At this stage, the goal is no longer to repeatedly confirm absence of infection, but to identify **functional or structural contributors**, particularly related to the prostate, bladder outlet, or ejaculatory system.

Step 2: High-Resolution Imaging (Structural and Deep Pelvic Evaluation)

If symptoms persist, **advanced imaging should be performed**, preferably:

- **Multiparametric MRI of the prostate and seminal vesicles (3T MRI)**
- Pelvic MRI (including surrounding soft tissues)

Imaging may reveal:

- Post-inflammatory changes or fibrosis
- Asymmetry of prostate or seminal vesicles
- Microcalcifications or calcified deposits
- Signs of **seminal vesicle dilation or secretion stasis**
- Possible **functional or partial obstruction at the level of ejaculatory ducts**

These findings, while not always definitive, may support a **deep pelvic phenotype involving impaired outflow, increased intraductal pressure, and chronic irritation**.

If neurological symptoms are present (e.g., radiating pain, numbness), additional imaging should include:

- **Lumbar and sacral spine MRI**

If vascular features are suspected (pelvic heaviness, improvement when lying down):

- Doppler ultrasound of pelvic veins
- MR/CT venography

Step 3: Pelvic Floor Physiotherapy Assessment (Neuromuscular Evaluation)

At this stage, referral to a **specialized pelvic floor physiotherapist** is essential. This evaluation should include:

- Digital rectal assessment of pelvic floor tone
- Identification of trigger points
- Assessment of relaxation capacity (not only strength)
- Breathing pattern analysis
- Postural and biomechanical evaluation

In a significant proportion of CPPS patients, this step reveals **pelvic floor hypertonicity, non-relaxing patterns, or myofascial dysfunction**, which may act as a primary or secondary driver of symptoms.

Step 4: Neurological and Pain System Evaluation

If symptoms suggest neuropathic involvement (burning pain, allodynia, radiating patterns), the patient should be referred to a:

- **Neurologist or pain specialist**

Assessment may include:

- Clinical evaluation for peripheral neuropathy or radiculopathy
- Quantitative sensory testing (if available)
- Evaluation for central sensitization syndromes

This step aims to identify **neurogenic amplification mechanisms**, which are common in chronic pelvic pain and often coexist with musculoskeletal dysfunction.

Step 5: Gastrointestinal and Microbiome Assessment

If the clinical picture includes bloating, bowel irregularities, or diet-related symptom fluctuation, referral to a:

- **Gastroenterologist**

is recommended, with evaluation including:

- Stool testing (calprotectin, microbiota where relevant)
- Breath tests for SIBO, lactose, or fructose intolerance

- Assessment for functional bowel disorders (e.g., IBS)

This step addresses the **gut–pelvis axis**, which plays a significant role in a subset of patients through visceral cross-sensitization and inflammatory signaling.

Step 6: Immunological / Histamine-Related Evaluation

In patients reporting:

- Food sensitivity
- Alcohol intolerance
- Flushing, urticaria, dermographism
- Bladder irritation without infection

referral to an:

- **Allergist / immunologist**

should be considered. Investigations may include:

- Total and specific IgE
- Tryptase (baseline)
- Assessment for histamine intolerance (clinical or laboratory-supported)

This step explores **immune and mast-cell–related amplification**, which may contribute to a histamine-reactive pelvic pain phenotype.

Step 7: Endocrine and Sexual Function Assessment

If symptoms include low libido, erectile dysfunction, fatigue, or hormonal fluctuations, referral to an:

- **Endocrinologist or andrology specialist**

is indicated, with evaluation of:

- Testosterone, SHBG, prolactin
- Thyroid function

This step helps identify **hormonal modulation of pelvic symptoms**, particularly relevant in patients with altered sexual function or recovery patterns.

Step 8: Psychophysiological and Behavioral Assessment

Given the strong interaction between chronic pain and the central nervous system, evaluation by a:

- **Psychologist, psychiatrist, or pain specialist**

should be considered, particularly in patients with:

- Stress-related symptom exacerbation

- Anxiety or depressive symptoms
- Sleep disturbances
- Fear-avoidance behavior

This step addresses **autonomic dysregulation, central sensitization, and learned pain responses**, which frequently coexist with peripheral mechanisms.

Final Integration

After completion of the above steps, findings should be integrated into a **multidimensional phenotype**, rather than reduced to a single diagnosis. In most patients, CPPS represents a combination of:

- Pelvic floor dysfunction
- Neural sensitization
- Local inflammatory or post-inflammatory changes
- Vascular or outflow-related factors
- Immune or histamine-related amplification
- Psychophysiological modulation

The goal of this structured pathway is not only to exclude serious pathology, but to identify the **dominant and contributing mechanisms**, allowing for a targeted and individualized therapeutic strategy.

Phenotype-Based Interpretation of Medications, Supplements, and Physiotherapy in CPPS

Below is the expanded description version, including all medication groups, supplements, and physiotherapeutic/non-pharmacological interventions listed in the questionnaire, organized by CPPS phenotype. This is not medical advice and should be used as an educational framework for discussing treatment with a qualified clinician. The questionnaire itself lists the relevant medication, supplement, diagnostic, and non-pharmacological response categories

1. Outlet / Urinary Phenotype

This phenotype is suspected when the dominant symptoms are weak urinary stream, intermittent stream, difficulty initiating urination, post-void dribbling, sensation of incomplete bladder emptying, urinary retention episodes, bladder pressure, urgency, nocturia, or worsening after drugs such as pseudoephedrine or anticholinergics. Mechanistically, this is often not a fixed anatomical obstruction, but a functional “closed valve” state: increased smooth muscle tone in the bladder neck, prostatic urethra, and prostate region. It may be driven by sympathetic overactivity, stress, pelvic floor guarding, inflammation, or prostate-related irritation.

Alpha-blockers — tamsulosin, doxazosin, terazosin, alfuzosin, silodosin.
These medications reduce alpha-adrenergic smooth muscle tone in the bladder neck, prostate, and prostatic urethra. If they help, they usually improve urinary flow, reduce hesitancy, reduce post-void pressure, and may indirectly reduce deep pelvic pain caused by high outlet resistance. A positive response suggests an **outlet / smooth muscle / urinary phenotype**. Silodosin is often the strongest and most prostate/outlet-selective option, but it is also more likely to cause ejaculatory side effects, such as reduced or absent ejaculation. Alfuzosin is usually more balanced and may be better suited to mixed CPPS patients with moderate urinary symptoms and pain, especially when preservation of ejaculation is important.

Tadalafil.
Tadalafil may help by improving nitric oxide–mediated smooth muscle relaxation, pelvic blood flow, erectile function, and possibly bladder/prostate perfusion. If tadalafil improves urinary symptoms, pelvic heaviness, erection quality, and deep pelvic pressure, this suggests a mixed **outlet + vascular/congestion phenotype**.

Relevant supplements.
Pollen Extract/Cernilton/Graminex, Epilobium parviflorum, African Plum, Saw palmetto, pumpkin and black seed oil, and zinc may fit this phenotype when symptoms feel prostate-related, pressure-like, or urinary. Pollen extract and Epilobium are more relevant to deep pelvic/prostate discomfort; saw palmetto and African Plum are more general prostate-comfort supplements and should be interpreted cautiously rather than as strong diagnostic markers.

Helpful non-pharmacological actions.
Reducing sitting time, using an ergonomic chair, warm baths, regular physical activity, pelvic floor relaxation, diaphragmatic breathing, and physiotherapy may reduce sympathetic/pelvic floor contribution to outlet tension. Uroflowmetry and post-void residual urine testing are especially useful to support this phenotype.

What may worsen it.
Decongestants such as pseudoephedrine may increase smooth muscle tone and worsen urinary retention or pelvic pressure. Anticholinergic drugs may worsen incomplete emptying or retention. If these drugs aggravate symptoms, it strongly supports an outlet-sensitive phenotype.

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2. Pelvic Floor / Myofascial Phenotype

This phenotype is suspected when the patient reports perineal tension, involuntary pelvic floor clenching, a sensation of muscles pulling upward, difficulty relaxing the pelvic floor, pain with contraction or stretching, rectal fullness, constipation-related pain, difficulty passing stool, pelvic muscle spasms, hip tightness, lower back stiffness, or pain with hip movement. Mechanistically, this is a chronic overactivity state of the pelvic floor muscles. The muscles behave like a fist that cannot fully open. This can compress nerves, restrict blood flow, create trigger points, and generate referred pain into the penis, testes, anus, groin, bladder, or prostate region.

Pelvic floor physiotherapy.
This is the core intervention for this phenotype. A specialized pelvic floor physiotherapist can assess pelvic floor tone, trigger points, ability to relax, breathing mechanics, posture, hip mobility, and lumbopelvic biomechanics. If pelvic floor physiotherapy helps, it strongly confirms that muscle tone and myofascial dysfunction are important pain drivers.

Pelvic floor muscle relaxation training.
This is different from strengthening. In CPPS, many patients do not need more contraction; they need better relaxation. If relaxation training reduces pain, urinary symptoms, rectal fullness, or post-ejaculatory discomfort, the patient likely has a non-relaxing pelvic floor component.

Trigger point therapy and myofascial release.
These target hyperirritable points and fascial restrictions in pelvic floor, hip, abdominal, gluteal, and adductor tissues. Improvement suggests a myofascial trigger-point phenotype.

Dry needling.
Dry needling may help when deep muscular trigger points or neuromuscular irritability contribute to symptoms. A positive response suggests a muscular or mixed neuromuscular pain generator.

Wise-Anderson protocol.
This fits patients whose pelvic pain is maintained by chronic pelvic tension, stress physiology, and poor relaxation. Improvement suggests a psychophysiological pelvic floor phenotype.

Breathing exercises / diaphragmatic breathing.
The diaphragm and pelvic floor work as a pressure-regulation system. Chest-dominant breathing, breath holding, and abdominal bracing may maintain pelvic floor tension. If diaphragmatic breathing helps, it supports a breathing–pelvic floor–autonomic phenotype.

Heat therapy, warm baths, sauna.
Heat and warm baths may reduce muscle guarding and improve comfort in spasm-dominant patients. Sauna may help some through whole-body relaxation and circulation, but it may worsen patients with heat sensitivity, vascular congestion, or histamine-reactive symptoms.

Cold therapy / cryotherapy.
Cold may help acute inflammatory flares or local hypersensitivity in some patients, but may worsen muscle spasm, cold-sensitive neuropathic pain, or autonomic guarding.

Stretching.
Stretching may help if hip tightness, shortened pelvic muscles, or myofascial restriction contribute to symptoms. It must be gradual; aggressive stretching may worsen sensitized tissues.

Ergonomic chair, U-shaped cushion, sitting sideways/offloading.
If these help, the pain is strongly pressure-sensitive. This suggests perineal compression, pudendal irritation, pelvic floor overload, or vascular congestion.

Medications fitting this phenotype.
Diazepam, especially rectal diazepam suppositories, may reduce local pelvic floor spasm. Oral diazepam may reduce both muscle tone and anxiety-driven guarding but carries risks of sedation, tolerance, and dependence. Baclofen may reduce muscle spasm and overactivity. These should be used only under medical supervision.

Supplements fitting this phenotype.
Magnesium may reduce neuromuscular excitability. Ashwagandha and L-theanine may help when stress and autonomic arousal maintain muscle tension. PEA may help if myofascial pain overlaps with neuroinflammation.

What may worsen it.
Heavy lifting, abdominal bracing, intense strength training, prolonged sitting, stress, breath holding, constipation, and aggressive Kegel exercises may worsen this phenotype.

3. Neuropathic / Neurogenic / Central Sensitization Phenotype

This phenotype is suspected when symptoms include burning, electric-shock sensations, tingling, prickling, numbness, allodynia, hypersensitivity of the penis or perineum, pain radiating along nerves, suspected pudendal neuralgia, suspected sacral nerve irritation, pain spreading beyond the

original location, pain persisting after the stimulus is removed, migraine, fibromyalgia, palpitations, cold hands/feet, sweating changes, dizziness on standing, or heat/cold intolerance.

Mechanistically, the problem is abnormal nerve excitability and pain amplification. Peripheral nerves may release inflammatory mediators such as substance P and CGRP, creating neurogenic inflammation. Over time, the spinal cord and brain may amplify pain signals, creating central sensitization.

Pregabalin and gabapentin.
These reduce neuronal excitability and are most relevant when pain is burning, electric, tingling, radiating, or hypersensitive. If they reduce pain, this strongly supports a neuropathic component.

Amitriptyline.
Amitriptyline can reduce central pain processing, improve sleep, and calm visceral hypersensitivity. It may help neuropathic pain, bladder pain overlap, and central sensitization. Long-term use can worsen some patients through anticholinergic effects, sedation, sexual dysfunction, or poor tolerability.

Ketamine medications.
Ketamine may reduce NMDA-mediated sensitization and is most relevant in severe central sensitization, allodynia, persistent post-trigger pain, or burning hypersensitive pain.

Lidocaine medications.
Lidocaine suggests a more local peripheral sensory generator. If rectal lidocaine helps, local pelvic floor, anorectal, pudendal, or peripheral afferent input may be a major driver.

LDN — low-dose naltrexone.
LDN is interpreted as a neuroimmune/pain-modulation option. If it helps, it may suggest microglial activation, neuroimmune amplification, central sensitization, or chronic pain regulation dysfunction.

PEA — palmitoylethanolamide.
PEA fits especially well in this phenotype because it may modulate neuroinflammation, mast-cell activity, and nerve sensitization. Improvement with PEA suggests a neuroimmune or neuropathic component.

Acetyl L-carnitine.
This may support nerve metabolism and is most relevant when symptoms are burning, tingling, radiating, or nerve-like.

TENS.
TENS may help by neuromodulating peripheral afferent signals. Improvement suggests that pain is responsive to peripheral nerve modulation.

Nerve blocks.
If nerve blocks help, this strongly suggests peripheral neural contribution, such as pudendal, sacral, or local pelvic nerve involvement.

Supplements fitting this phenotype.
PEA, acetyl L-carnitine, omega-3, NAC, curcumin, boswellia, quercetin, and luteolin may help when neuropathic pain overlaps with neuroinflammation or immune activation.

What may worsen it.
Poor sleep, stress, prolonged sitting, aggressive manual therapy, repeated trigger exposure, catastrophizing, and constant symptom monitoring can amplify nervous-system sensitivity. Some

patients may worsen on SSRIs, SNRIs, duloxetine, or long-term amitriptyline due to individual autonomic, sexual, or serotonergic sensitivity.

4. Deep Pelvic / Prostate–Seminal Vesicle / Ejaculatory Phenotype

This phenotype is suspected when the patient reports pain during ejaculation, immediately after ejaculation, several hours after ejaculation, the day after ejaculation, pain after ejaculation combined with sitting, pain in the seminal vesicle region, reduced semen volume, weak ejaculation, urethral burning after ejaculation, testicular pain after ejaculation, pain during nocturnal emission, worsening with frequent daily ejaculation, or worsening after abstinence.

Mechanistically, this phenotype involves the prostate, seminal vesicles, ejaculatory ducts, pelvic floor, urethra, and autonomic nerves. Some patients have a **load–recovery imbalance**, where frequent sexual activation and ejaculation exceed the recovery capacity of the deep pelvic system. Others have a **congestion/abstinence-sensitive pattern**, where infrequent ejaculation increases fullness, pressure, or stagnation.

Reduced frequency of ejaculation.
If reducing ejaculation frequency helps, this suggests sexual overload, post-ejaculatory inflammatory flare, pelvic floor spasm after ejaculation, or insufficient recovery of prostate/seminal vesicle tissues.

Increased frequency of ejaculation.
If increasing ejaculation helps, this suggests congestion, stasis, or abstinence-sensitive deep pelvic pressure.

Alpha-blockers.
They may help if ejaculation-related symptoms are linked to smooth muscle spasm, outlet pressure, or impaired outflow through the prostate/urethral system.

Tadalafil.
May help if the deep pelvic phenotype overlaps with vascular congestion, erectile dysfunction, smooth muscle tension, or pelvic blood flow problems.

NSAIDs.
NSAIDs may reduce inflammatory flares, especially when pain appears after ejaculation, exertion, or tissue irritation. A positive response suggests an inflammatory component but is not specific.

Steroids — short-term use only.
Steroids are important interpretively but must be handled carefully. If a short course produces clear improvement, this may suggest an inflammatory, post-inflammatory, autoimmune-like, or neuroimmune amplification component. This is most meaningful when the patient has deep prostate/seminal vesicle pain, delayed post-ejaculatory pain, inflammatory semen findings, imaging signs of chronic inflammation, or immune/histamine symptoms. However, steroid response is not a definitive diagnosis and steroids are not a long-term CPPS solution. They should only be considered under physician supervision.

Pollen Extract / Cernilton / Graminex.
This fits a deep pelvic/prostate-related phenotype. If it helps, it may suggest prostate-region inflammation, edema, or deep pelvic irritation.

Palmitoylethanolamide, Epilobium and Calendula suppositories.
This combination fits a mixed phenotype involving neuroinflammation, prostate/seminal vesicle

irritation, local tissue inflammation, and impaired recovery. Rectal delivery may be relevant because of proximity to the prostate, seminal vesicles, and pelvic floor.

Quercetin, curcumin, omega-3, boswellia, NAC.
These are most relevant when the deep pelvic phenotype has inflammatory or oxidative-stress features.

Prostate massage.
If prostate massage helps, it may suggest congestion, drainage-related pressure, or deep pelvic stasis. If it worsens pain, this may suggest tissue irritation, inflammation, pelvic floor spasm, or nerve hypersensitivity.

What may worsen it.
Frequent ejaculation without recovery, edging, delayed ejaculation, interrupted intercourse, prolonged sitting after ejaculation, long abstinence in congestion-sensitive patients, testosterone/anabolic stimulation in susceptible men, and untreated pelvic floor tension may all worsen this phenotype.

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5. Histamine / Immune / MCAS-Like Phenotype

This phenotype is suspected when symptoms worsen after alcohol, coffee, spicy foods, fermented foods, high-histamine foods, sour foods, artificial sweeteners, energy drinks, citrus fruits, tomatoes, intense physical activity, irregular sleep, pressure, cold, or when the patient has allergic rhinitis, hay fever, asthma, flushing, hives, itching without rash, dermographism, food intolerance, suspected histamine intolerance, or reactions to perfumes, detergents, chemicals, pollen, dust, mold, or animal dander.

Mechanistically, mast cells and histamine-related pathways may amplify pelvic pain, bladder urgency, burning, pressure, urethral symptoms, bowel symptoms, and nerve hypersensitivity. Substance P and mast cells can reinforce each other: nerves activate immune mediators, and immune mediators increase nerve sensitivity.

Antihistamines.
If antihistamines help, this supports a histamine-reactive phenotype. This is especially meaningful when food triggers, skin symptoms, bladder pain, urgency, burning, or pressure sensitivity are present.

Hydroxyzine.
Hydroxyzine may help through H1 blockade, sedation, sleep improvement, anxiety reduction, and bladder pain modulation. If it helps, it may indicate histamine involvement, bladder-pain overlap, or neuroimmune arousal.

H1 antihistamines — bilastine, cetirizine, fexofenadine.
If one of these helps, it suggests H1-mediated histamine symptoms. The response may differ between agents, so individual response matters.

H2 blockers.
If H2 blockers help, this may suggest histamine involvement in gut, bladder, reflux-like symptoms, or systemic histamine reactivity. H2 benefit is especially meaningful when combined with H1 benefit.

Steroids — short-term use only.
If short-term steroids help in this phenotype, it may indicate inflammatory or immune amplification. This is most relevant in flares with histamine-like symptoms, bladder irritation, deep pelvic inflammation, or post-ejaculatory inflammatory pain. Long-term steroid use is not appropriate as a general CPPS strategy.

Quercetin and luteolin.
These are mast-cell-stabilizing and anti-inflammatory supplements. If they help, especially with histamine food triggers, flushing, itching, or antihistamine response, this strongly supports a histamine/MCAS-like component.

DAO supplements.
DAO response suggests food-histamine handling problems. This is most relevant when high-histamine foods, fermented foods, alcohol, or aged foods trigger pelvic symptoms.

Vitamin C.
May support histamine degradation and oxidative stress control. It can help some histamine-sensitive patients but may worsen bladder/urethral burning in patients sensitive to acidic urine or acidic supplements.

PEA.
PEA fits histamine/immune and neurogenic phenotypes because of its neuroimmune and mast-cell-related activity.

Low-histamine diet.
Improvement on a low-histamine diet is one of the strongest non-drug indicators of this phenotype.

What may worsen it.
Alcohol, fermented foods, histamine-rich foods, irregular sleep, stress, heat or cold exposure in sensitive patients, pressure/tight clothing, and medications triggering histamine release. If histamine-releasing medications worsen symptoms, the immune/histamine phenotype becomes very likely.

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6. Bladder Pain / Urethral Sensory / IC-Like Phenotype

This phenotype is suspected when symptoms include bladder pain, bladder pressure, pain when the bladder fills, relief after urination, worsening after urination, urgency attacks, nocturnal urgency, frequent urination, urethral burning, bladder hypersensitivity, pelvic pressure when the bladder is full, or diagnosis of painful bladder syndrome/interstitial cystitis.

Mechanistically, the bladder and urethra behave as hypersensitive organs. The cause may involve urothelial irritation, mast-cell activity, urine chemistry, pelvic floor guarding, central sensitization, or overlap with bowel and immune triggers.

Amitriptyline.
May reduce bladder pain, urgency, visceral hypersensitivity, and sleep-related pain amplification.

Hydroxyzine, H1 antihistamines, H2 blockers.
Most relevant when bladder symptoms coexist with allergies, histamine foods, flushing, hives, or pressure sensitivity.

Pentosan polysulfate / Elmiron.
Most relevant for bladder pain syndrome/IC-like symptoms, especially pain with bladder filling, urgency, and relief after urination. If it helps, it suggests a bladder/urothelial barrier or bladder sensory phenotype.

DMSO rectal medications.
If helpful, this may suggest local anti-inflammatory or pelvic sensory modulation effects, though interpretation depends on formulation and clinical context.

Lidocaine and ketamine medications.
These fit bladder/pelvic sensory hypersensitivity, peripheral neural irritation, and central sensitization.

Tadalafil.
May help if bladder symptoms overlap with pelvic smooth muscle tension, vascular congestion, or LUTS.

Supplements.
Quercetin, luteolin, PEA, DAO, probiotics, omega-3, curcumin, and NAC may help if bladder symptoms are linked to histamine, gut, or inflammation.

Dietary actions.
Low-histamine diet, avoiding acidic foods, avoiding citrus/tomatoes, avoiding coffee/alcohol/energy drinks, and monitoring acidic urine triggers may be useful.

What may worsen it.
Acidic urine, vitamin C in sensitive individuals, citrus, tomatoes, coffee, alcohol, artificial sweeteners, energy drinks, spicy foods, and some supplements or medications that irritate the bladder.

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7. Vascular / Pelvic Congestion Phenotype

This phenotype is suspected when symptoms include pelvic heaviness, pressure, fullness, visible scrotal varicose veins, worsening with standing, improvement when lying down, deep pelvic pressure, or end-of-day worsening.

Mechanistically, the problem may involve impaired venous drainage, venous pooling, pressure overload, or pelvic congestion. It can coexist with prostate/seminal vesicle symptoms and pelvic floor guarding.

Tadalafil.
This is the most relevant medication in the questionnaire for this phenotype. It may improve pelvic blood flow, smooth muscle relaxation, erectile function, and deep pelvic pressure.

L-arginine.
May support nitric oxide pathways and vascular tone. If it helps with heaviness, erection quality, or pelvic pressure, it may suggest a vascular component.

Regular physical activity.
Movement improves venous return and reduces static pelvic pressure. If activity helps while standing still worsens, this supports congestion physiology.

Reducing sitting time, offloading, ergonomic chair, U-shaped cushion.
These reduce pelvic pressure and may help both vascular and pudendal/mechanical phenotypes.

Heat vs cold.
Heat may help muscular pain but may worsen vascular congestion in some patients. Cold may reduce acute vascular/inflammatory discomfort but may worsen muscle spasm or neuropathic cold sensitivity.

Supplements.
Omega-3, curcumin, boswellia, NAC, and L-arginine may be supportive if vascular congestion overlaps with inflammation or oxidative stress.

What may worsen it.
Prolonged standing, prolonged sitting, heat exposure in heat-sensitive patients, lack of movement, and possibly high pelvic pressure from constipation or heavy lifting.

8. Gut / IBS / Microbiome / Food-Related Phenotype

This phenotype is suspected when symptoms include bloating, constipation, diarrhea, IBS, food worsening pelvic pain, gluten sensitivity, dairy sensitivity, FODMAP sensitivity, reflux, nausea during flare-ups, relief after bowel movement, worsening after bowel movement, stomach pain after eating, hyperacidity, or gastritis-like symptoms.

Mechanistically, the bowel can influence pelvic pain through shared nerves, pelvic floor pressure, immune activation, visceral hypersensitivity, microbiome changes, and mechanical distension. Constipation can overload the pelvic floor; diarrhea and IBS can increase visceral sensitivity; food reactions can trigger immune, histamine, bladder, and pelvic floor symptoms.

Low-FODMAP diet.
If it helps, this suggests fermentation-sensitive IBS/gut–pelvis interaction.

Gluten-free diet.
If it helps, this suggests gluten-related sensitivity or gut immune involvement. Celiac testing should be considered before long-term gluten elimination if clinically relevant.

Probiotics.
If they help, this suggests microbiome-related contribution, especially with bloating, IBS, antibiotic history, or food-triggered symptoms.

DAO supplements and low-histamine diet.
If food triggers are histamine-rich, DAO and low-histamine diet may point toward gut–histamine overlap.

H2 blockers.
May help histamine/reflux/upper-GI overlap.

Amitriptyline.
May help visceral hypersensitivity, IBS-like pain, and sleep-related amplification.

PEA, quercetin, luteolin, NAC, curcumin, omega-3.
These may help if gut symptoms overlap with neuroimmune or inflammatory sensitivity.

What may worsen it.
Proton pump inhibitors such as omeprazole may worsen symptoms in some sensitive patients, possibly through digestion or microbiome changes, though they help others with reflux. Fermented foods, alcohol, FODMAP foods, dairy, gluten, acidic foods, stress, and poor sleep may worsen this phenotype.

9. Psychophysiological / Autonomic / Attention-Pain Loop Phenotype

This phenotype is suspected when symptoms worsen with stress or anxiety, focusing on the body, symptom monitoring, catastrophizing, fear of worsening, poor sleep, racing thoughts, difficulty relaxing, breath holding, chest-dominant breathing, palpitations, cold hands/feet, sweating abnormalities, dizziness on standing, or when symptoms improve with distraction or engagement.

Mechanistically, this is not “imaginary pain.” It means the autonomic nervous system, attention networks, breathing, pelvic floor tone, and threat perception amplify real pelvic signals. The body stays in a defensive state.

Psychotherapy.
If psychotherapy helps, it suggests that fear, avoidance, trauma, stress physiology, symptom monitoring, or catastrophizing contribute to maintenance of pain.

Meditation / yoga.
May reduce autonomic arousal, improve body awareness without fear, and lower pelvic guarding.

Stress reduction techniques.
If these help, stress is a major amplifier.

Breathing exercises.
If diaphragmatic breathing helps, the diaphragm–abdomen–pelvic floor pressure system is important.

Wise-Anderson protocol.
Fits patients with chronic pelvic tension, anxiety physiology, and learned pelvic guarding.

Amitriptyline.
May help sleep, pain processing, and central sensitization.

Pregabalin / gabapentin.
May help if anxiety-like arousal overlaps with neuropathic pain.

Diazepam.
May help acutely with severe guarding, panic-like pelvic tension, or spasm, but is not a long-term solution due to dependence risk.

LDN.
May fit chronic neuroimmune pain with central sensitization.

Supplements.
Magnesium, ashwagandha, L-theanine, PEA, omega-3, and NAC may support nervous-system downregulation, sleep, and stress tolerance.

What may worsen it.
Poor sleep, caffeine/energy drinks, stimulants, psychoactive substances, constant symptom checking, fear avoidance, and in some patients SSRIs, SNRIs, duloxetine, or long-term amitriptyline due to individual sensitivity, sexual side effects, or autonomic activation.

10. Hormonal / Sexual Regulation Phenotype

This phenotype is suspected when symptoms involve high or low libido, erectile dysfunction, difficulty achieving orgasm, weak ejaculation, reduced semen volume, increased libido, decreased libido, compulsive sexual behavior, pornography-driven overstimulation, worsening after hormonal therapy, testosterone therapy, anabolic steroids, hormone precursors, or finasteride.

Mechanistically, hormones influence sexual drive, ejaculation frequency, pelvic autonomic tone, prostate/seminal vesicle activity, erectile function, and recovery capacity. The issue is not that libido is "bad," but that sexual activation may become mismatched with pelvic recovery.

Tadalafil.
May help erectile function, vascular tone, smooth muscle relaxation, and pelvic congestion.

Testosterone therapy / anabolic steroids / hormone precursors.
If these worsen symptoms, they may increase libido, sexual frequency, autonomic activation, prostate/seminal vesicle reactivity, or pelvic load before recovery is complete. Worsening suggests a hormone-amplified sexual/pelvic phenotype.

Finasteride.
If finasteride worsens symptoms, it may suggest sensitivity to androgen pathway modulation, sexual dysfunction, genital sensory change, libido disturbance, or mood/autonomic effects.

SSRIs / SNRIs / duloxetine.
These may help some patients with anxiety or central pain, but may worsen sexual function, orgasm, ejaculation, libido, or genital sensory symptoms in others. If they worsen pelvic pain through sexual dysfunction or autonomic changes, that reaction becomes diagnostically meaningful.

Supplements.
Zinc, L-arginine, Pollen Extract, Epilobium, PEA, and acetyl L-carnitine may be relevant depending on whether the dominant issue is prostate/seminal vesicle, vascular, neuropathic, or sexual-performance related.

Behavioral interventions.
Reduced ejaculation frequency supports overload phenotype. Increased ejaculation frequency supports abstinence/congestion phenotype. Avoiding edging, prolonged arousal, interrupted intercourse, and repeated ejaculation without recovery may be important in overload-type patients.

11. Inflammatory / Post-Inflammatory / Fibrosis-Calcification Phenotype

This phenotype is suspected when symptoms began after infection, STI, urinary catheterization, surgery, antibiotic treatment, gastrointestinal illness, pelvic injury, or when imaging shows post-inflammatory prostate or seminal vesicle changes, fibrosis-like remodeling, calcifications, stones, asymmetry, impaired drainage, seminal vesicle dilation, or possible ejaculatory duct outflow impairment.

Mechanistically, tissue may remain irritated after inflammation. Fibrosis, calcifications, microstones, ductal narrowing, and secretion stasis may increase pressure, reduce drainage, irritate nerves, and maintain low-grade inflammation. Imaging such as TRUS, prostate MRI, pelvic MRI, and 3T MRI may help identify visible structural contributors, although normal imaging does not exclude pain.

NSAIDs.
If NSAIDs help, this suggests inflammatory or mechanical-irritative pain, but the response is nonspecific.

Steroids — short-term use only.
Steroids deserve special attention. A clear response to a short steroid course may indicate active inflammatory amplification, post-inflammatory edema, immune activation, autoimmune-like mechanisms, or neuroimmune flare biology. This is most meaningful when combined with

inflammatory semen markers, cytokines, imaging abnormalities, histamine/immune symptoms, or delayed post-ejaculatory pain. However, steroid response is not proof of one diagnosis and steroids can carry significant risks, so they should be used only under medical supervision and not as a chronic strategy.

Pollen Extract / Cernilton / Graminex.
May support prostate-region anti-inflammatory, anti-edema, or symptom-reducing effects.

PEA, Epilobium and Calendula suppositories.
May fit mixed local inflammation, neuroinflammation, tissue irritation, and deep pelvic/prostate-seminal involvement.

Quercetin.
Fits inflammatory, antioxidant, and mast-cell-related mechanisms.

Curcumin, omega-3, boswellia, NAC, luteolin, vitamin C.
These may support anti-inflammatory or antioxidant pathways. Vitamin C should be used cautiously in bladder-sensitive patients because acidic urine may worsen urethral/bladder burning.

What may worsen it.
Repeated sexual overload, poor sleep, alcohol, histamine triggers, untreated outlet dysfunction, pelvic floor spasm, heavy lifting, and prolonged sitting may perpetuate tissue irritation.

How to Identify Your Type or Types

The most important principle is that CPPS is usually mixed. A patient rarely has only one phenotype. The aim is to identify the **dominant driver** and the **secondary amplifiers**.

If alpha-blockers improve urinary flow and reduce pelvic pressure, the patient likely has an **outlet / urinary phenotype**.
If pelvic floor physiotherapy, relaxation training, trigger point therapy, warm baths, or breathing work help, the patient likely has a **pelvic floor / myofascial phenotype**.
If pregabalin, gabapentin, amitriptyline, ketamine, lidocaine, PEA, acetyl L-carnitine, TENS, or nerve blocks help, the patient likely has a **neuropathic / sensitization phenotype**.
If reduced ejaculation helps, the patient likely has a **sexual overload / poor recovery phenotype**.
If increased ejaculation helps, the patient may have an **abstinence / congestion phenotype**.
If antihistamines, hydroxyzine, H1/H2 blockers, DAO, quercetin, luteolin, or low-histamine diet help, the patient likely has a **histamine / immune / MCAS-like phenotype**.
If tadalafil, L-arginine, lying down, movement, or venous offloading help, the patient may have a **vascular / congestion phenotype**.
If low-FODMAP diet, probiotics, bowel treatment, gluten-free diet, or DAO help, the patient likely has a **gut–pelvis phenotype**.
If psychotherapy, meditation, stress reduction, breathing, sleep improvement, or Wise-Anderson work helps, the patient likely has a **psychophysiological / autonomic phenotype**.
If NSAIDs, short-term steroids, quercetin, curcumin, boswellia, PEA, or imaging-supported tissue findings align with symptoms, the patient may have an **inflammatory / post-inflammatory phenotype**.

The best interpretation comes from combining: symptoms, triggers, diagnostic tests, what helps, what worsens, and what type of response appears repeatedly.

This questionnaire is based on the latest available medical knowledge and current multidisciplinary understanding of chronic pelvic pain syndrome (CPPS). However, it is intended solely as an educational and supportive tool and does not replace consultation with a

qualified healthcare professional. The results obtained from this questionnaire should not be considered a medical diagnosis, and no clinical decisions should be made without appropriate medical evaluation. The creators of this questionnaire do not assume responsibility for any interpretations or actions taken based on its results.

Chronic pelvic pain can be a complex and often frustrating condition, especially when initial tests and treatments do not provide clear answers. However, clinical experience and numerous patient-reported outcomes consistently show that a step-by-step, phenotype-based approach—identifying and addressing underlying mechanisms one by one—can lead to meaningful and lasting improvement. Although this process may take time, many patients have achieved significant symptom relief by gradually understanding their individual triggers and responses. Real-world patient experiences, including those shared on platforms such as ucpps.men and similar communities, highlight that recovery is often not immediate, but progressive and achievable with a structured, personalized strategy. This reinforces an important message: even in long-standing cases, improvement is possible when the condition is approached comprehensively and systematically.

Main references

1. American Urological Association. Male Chronic Pelvic Pain Guideline. 2025. Available from: https://www.auanet.org/guidelines-and-quality/guidelines/male-chronic-pelvic-pain
2. European Association of Urology. Guidelines on Chronic Pelvic Pain. 2025. Available from: https://uroweb.org/guidelines/chronic-pelvic-pain
3. Rees J, Abrahams M, Doble A, Cooper A. Diagnosis and treatment of chronic bacterial prostatitis and chronic prostatitis/chronic pelvic pain syndrome. BMJ. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6040620/
4. Krieger JN, Nyberg L Jr, Nickel JC. NIH consensus definition and classification of prostatitis. JAMA. 1999;282(3):236–7.
5. StatPearls Publishing. Chronic Prostatitis and Chronic Pelvic Pain Syndrome (CPPS). Available from: https://www.ncbi.nlm.nih.gov/books/NBK599550/
6. European Association of Urology. Management of Chronic Pelvic Pain – Chapter. Available from: https://uroweb.org/guidelines/chronic-pelvic-pain/chapter/management
7. Ji RR, Nackley A, Huh Y, Terrando N, Maixner W. Neuroinflammation and central sensitization in chronic pain. Nat Rev Neurosci. 2018;19(7):455–70. Available from: https://pubmed.ncbi.nlm.nih.gov/25659506/
8. Frontiers in Pain Research. Central Sensitization in Chronic Pain Disorders. 2026. Available from: https://www.frontiersin.org/articles/10.3389/fpain.2026.1738762/full
9. Frontiers in Microbiology. Microbiota and Chronic Pelvic Pain. 2025. Available from: https://www.frontiersin.org/articles/10.3389/fmicb.2025.1488732/full
10. Frontiers in Immunology. Immune and Mast Cell Mechanisms in Chronic Pelvic Pain. 2026. Available from: https://www.frontiersin.org/articles/10.3389/fimmu.2026.1793156/full
11. Theoharides TC, et al. Mast cells and inflammation in chronic pelvic pain syndromes. Available from: https://pubmed.ncbi.nlm.nih.gov/26827846/
12. Theoharides TC. Neurogenic inflammation, mast cells, and chronic pain. Available from: https://pubmed.ncbi.nlm.nih.gov/28321127/
13. Dimitrakov J, et al. Chronic prostatitis/chronic pelvic pain syndrome: a possible role for mast cells. Available from: https://pubmed.ncbi.nlm.nih.gov/16952676/
14. Offiah I, McMahon SB, O'Reilly BA. Mast cells and their role in urological chronic pelvic pain syndromes. Available from: https://pubmed.ncbi.nlm.nih.gov/24774531/
15. Shoskes DA, Nickel JC, Dolinga R, Prots D. Clinical phenotyping of patients with chronic prostatitis/chronic pelvic pain syndrome and correlation with symptom severity. J Urol. 2009;182(1):123–9. Available from: https://pubmed.ncbi.nlm.nih.gov/19118880/
16. Shoskes DA, Nickel JC, Kattan MW. Phenotypically directed multimodal therapy for CP/CPPS: a prospective study using UPOINT. Available from: https://pubmed.ncbi.nlm.nih.gov/20363491/
17. Nickel JC. Phenotypic approach to the management of chronic prostatitis/chronic pelvic pain syndrome. Available from: https://pubmed.ncbi.nlm.nih.gov/19570493/
18. Bryk DJ, Shoskes DA. Using the UPOINT system to manage men with CP/CPPS. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC8451687/
19. Anderson RU, Wise D, Sawyer T, Chan CA. Integration of myofascial trigger point release and paradoxical relaxation training for CPPS. Available from: https://pubmed.ncbi.nlm.nih.gov/16952676/
20. FitzGerald MP, Payne CK, Lukacz ES, et al. Randomized trial of pelvic floor physical therapy for urological chronic pelvic pain syndromes. Available from: https://pubmed.ncbi.nlm.nih.gov/21613621/
21. Shoskes DA, et al. The role of the microbiome in chronic prostatitis. Available from: https://pubmed.ncbi.nlm.nih.gov/28676230/

22. Mullins C, et al. The gut microbiome and chronic pelvic pain. Available from: https://pubmed.ncbi.nlm.nih.gov/30017649/
23. Beard RW, et al. Pelvic congestion syndrome. Available from: https://pubmed.ncbi.nlm.nih.gov/10775844/
24. Gavrilov SG, et al. Pelvic venous disorders and chronic pelvic pain. Available from: https://pubmed.ncbi.nlm.nih.gov/31525770/
25. Mayer EA. The neurobiology of stress and pain. Available from: https://pubmed.ncbi.nlm.nih.gov/24726462/
26. Lee KS. Chronic prostatitis: approaches for best management. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC3285711/
27. Franco JVA, Turk T, Jung JH, et al. Pharmacological interventions for treating CP/CPPS. Cochrane Database. Available from: https://www.cochrane.org/evidence/CD012552
28. Qin Z, Wu J, Zhou J, et al. Oral pharmacological treatments for CP/CPPS: systematic review and network meta-analysis. Lancet eClinicalMedicine. Available from: https://www.thelancet.com/journals/eclinm/article/PIIS2589-5370%2822%2900187-0/fulltext
29. Pontari MA, et al. Pregabalin for CP/CPPS: randomized controlled trial. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC3767281/
30. Benelli A, et al. Once-daily tadalafil for CP/CPPS. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC6295787/
31. Tawfik AM, et al. Tadalafil monotherapy in CP/CPPS. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC9512753/
32. Wagenlehner FME, et al. Pollen extract in CP/CPPS: randomized trial. Available from: https://pubmed.ncbi.nlm.nih.gov/19524353/
33. Cai T, et al. Flower pollen extract in CP/CPPS: systematic review. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC5401347/
34. Magri V, et al. Nutraceuticals in CP/CPPS. Available from: https://www.mdpi.com/2673-4397/6/1/4
35. Palmitoylethanolamide (PEA) in chronic pain and inflammation. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC12358156/
36. PubMed. Phytotherapy and combination treatments in chronic pelvic pain. Available from: https://pubmed.ncbi.nlm.nih.gov/38934521/
37. NCCIH. Saw Palmetto: Usefulness and Evidence. Available from: https://www.nccih.nih.gov/health/saw-palmetto
38. PubMed. Bee pollen extract in CPPS. Available from: https://pubmed.ncbi.nlm.nih.gov/40981184/
39. American Urological Association. Diagnosis and treatment of interstitial cystitis/bladder pain syndrome. Available from: https://www.auajournals.org/doi/10.1097/JU.0000000000002756
40. Traish AM. Post-finasteride syndrome. Available from: https://pubmed.ncbi.nlm.nih.gov/28289563/
41. Corona G, et al. Testosterone and lower urinary tract symptoms. Available from: https://pubmed.ncbi.nlm.nih.gov/26654610/

Introduction

Chronic pelvic pain in men (CP/CPPS – Chronic Prostatitis / Chronic Pelvic Pain Syndrome) is one of the most complex pain syndromes in urology. In some patients, the dominant symptom is **pain occurring after ejaculation**, often with a delay ranging from several hours to even one day.

An increasing number of studies suggest that in some cases the source of pain is not limited to the prostate alone, but involves the **seminal tract – particularly the seminal vesicles and ejaculatory ducts**.

1. Seminal Vesicles as a Potential Source of Pain

The seminal vesicles are among the **most densely innervated structures within the male pelvis**. Their innervation originates mainly from:

- the inferior hypogastric plexus
- pelvic splanchnic nerves
- autonomic fibers from S2–S4
- sympathetic fibers from the pelvic plexus

Because of this rich innervation, they can respond rapidly to **mechanical, inflammatory, and hormonal stimuli**.

For this reason, even **small structural or inflammatory changes** may produce significant pain symptoms. Such changes may include:

- chronic inflammatory alterations
- microcalcifications
- mild tissue fibrosis
- stagnation of seminal secretions
- mild dilation of the ejaculatory ducts

Imaging studies (TRUS and MRI) often demonstrate these subtle abnormalities in patients with chronic pelvic pain.

Due to the extensive innervation of the seminal vesicles, even minor disturbances in their function may lead to **significant pain symptoms**.

2. Neurogenic Inflammation

One of the most important hypotheses explaining post-ejaculatory pain is the phenomenon of **neurogenic inflammation**.

Nerve endings in pelvic tissues may release mediators such as:

- Substance P
- CGRP (calcitonin gene-related peptide)
- neurokinins

These substances cause:

- vasodilation
- mast cell activation
- release of inflammatory mediators

As a result, a **neuro-immune loop** may develop:

nerves → mast cells → inflammatory mediators → increased nerve sensitivity.

This mechanism may explain symptoms such as:

- pain after ejaculation
- pain during prolonged sitting
- a sensation of tension in the perineum
- burning in the urethra.

3. Why Pain Appears Several Hours After Ejaculation

In many patients, pain appears only **several hours after ejaculation**.

One hypothesis proposes the following mechanism:

1. ejaculation causes strong contractions of the seminal vesicles

2. inflammatory mediators are released from seminal fluid and surrounding tissues
3. mast cells become activated
4. local neurogenic inflammation develops
5. pain appears with a delay of several hours.

4. The Role of Mast Cells and Histamine

Histological studies have demonstrated an increased number of **mast cells in prostate and pelvic tissues** in some patients with CPPS.

Mast cells release substances such as:

- histamine
- prostaglandins
- tryptase
- inflammatory cytokines.

These mediators increase the sensitivity of pain receptors and may amplify pain signal transmission within pelvic nerves.

5. Medications Used in Treatment

Steroids

Steroids (for example prednisone) reduce:

- production of inflammatory cytokines
- mast cell activity
- tissue edema.

They may therefore temporarily reduce pain associated with inflammation.

Antihistamines

Antihistamines may reduce the effect of histamine on nerve endings.

Commonly used agents include:

- hydroxyzine
- cetirizine
- fexofenadine.

In some patients, a reduction of pain symptoms has been observed.

Quercetin

Quercetin is a natural flavonoid with:

- anti-inflammatory properties
- antioxidant effects
- mast cell stabilizing activity.

Clinical studies have shown that it may reduce pain symptoms in patients with CPPS.

6. Modern Approach to CPPS

Currently, chronic pelvic pain is considered a **multifactorial syndrome** involving:

- inflammatory processes
- neurological mechanisms
- pelvic floor muscle dysfunction
- immunological factors.

For this reason, treatment often requires a **multidisciplinary approach**.

Reliable Sources

AUA Male Chronic Pelvic Pain Guideline https://www.auanet.org/guidelines-and-quality/guidelines/male-chronic-pelvic-pain
Mast cells in chronic prostatitis https://pubmed.ncbi.nlm.nih.gov/37442295/
Neurogenic inflammation and pelvic pain https://pubmed.ncbi.nlm.nih.gov/37118962/
Quercetin therapy in CPPS https://pubmed.ncbi.nlm.nih.gov/10604689/

Biomarkers of inflammation in seminal plasma https://pubmed.ncbi.nlm.nih.gov/40914239/