Description of the Chronic Pelvic Pain Syndrome (CPPS) Survey

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
6. been excluded
7. you have consulted several urologists
8. 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, and 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://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.

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 pro- cesses—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 & 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 UCPPS 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**.