Fertility for Every Rehab Professional: Your Role in the Journey

Dr. Yeni Abraham, PT, DPT, PRFS

medbridge

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Learning Outcomes

- Recognize the basic physiology of conception and the primary female and male factors that contribute to infertility
- Identify modifiable lifestyle factors, including physical activity, body composition, metabolic health, and stress, that influence fertility across genders
- Recognize common clinical presentations, such as polyendocrine metabolic ovarian syndrome (PMOS) / polycystic ovary syndrome (PCOS) and metabolic-related anovulation, that a generalist rehabilitation clinician may encounter
- Recognize how appropriately dosed exercise supports reproductive health and how excessive training load or low energy availability can impair it
- Summarize the relationship between male metabolic and lifestyle factors and semen quality and fertility outcomes
- Identify the rehabilitation clinician's scope of practice and appropriate referral pathways within the fertility care team

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Chapter 1

Why Fertility Belongs in the Rehab Conversation

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The Endocrine Movement Interface



Endocrine Signaling

- Tissue healing
- Muscle mass
- Pain processing

Bidirectional Feedback

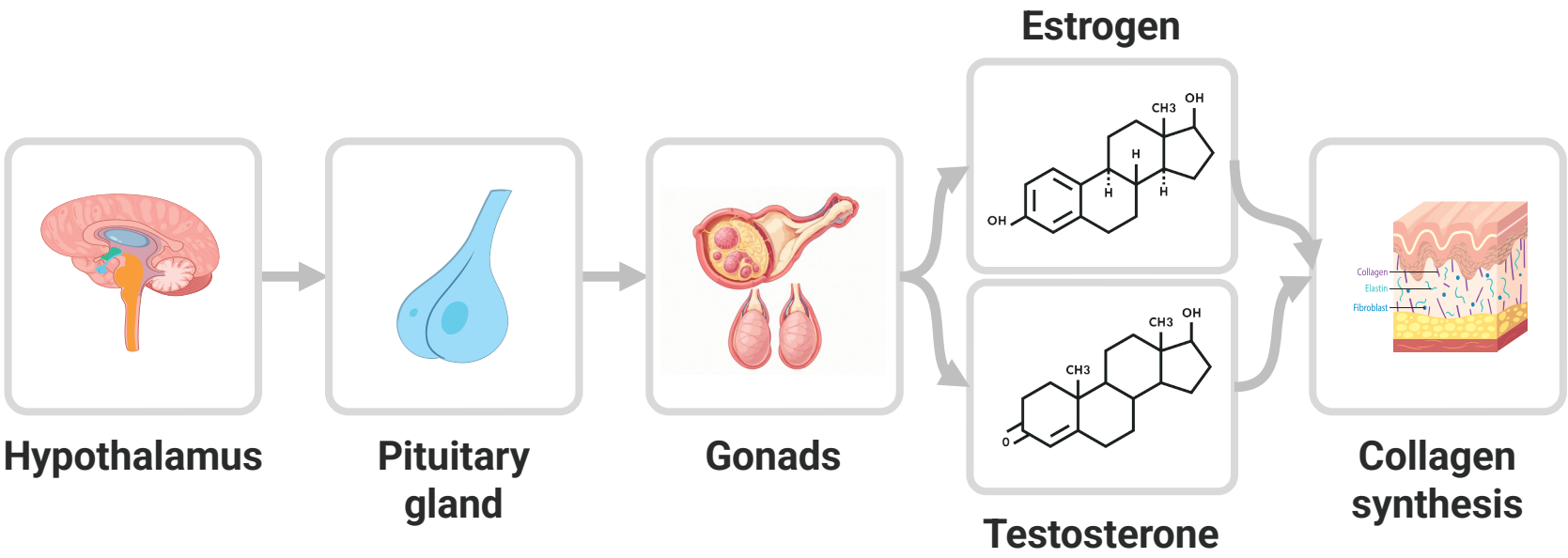


Movement System Loading

Physical activity regulates systemic hormone sensitivity

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HPG Axis and Connective Tissue Dynamics



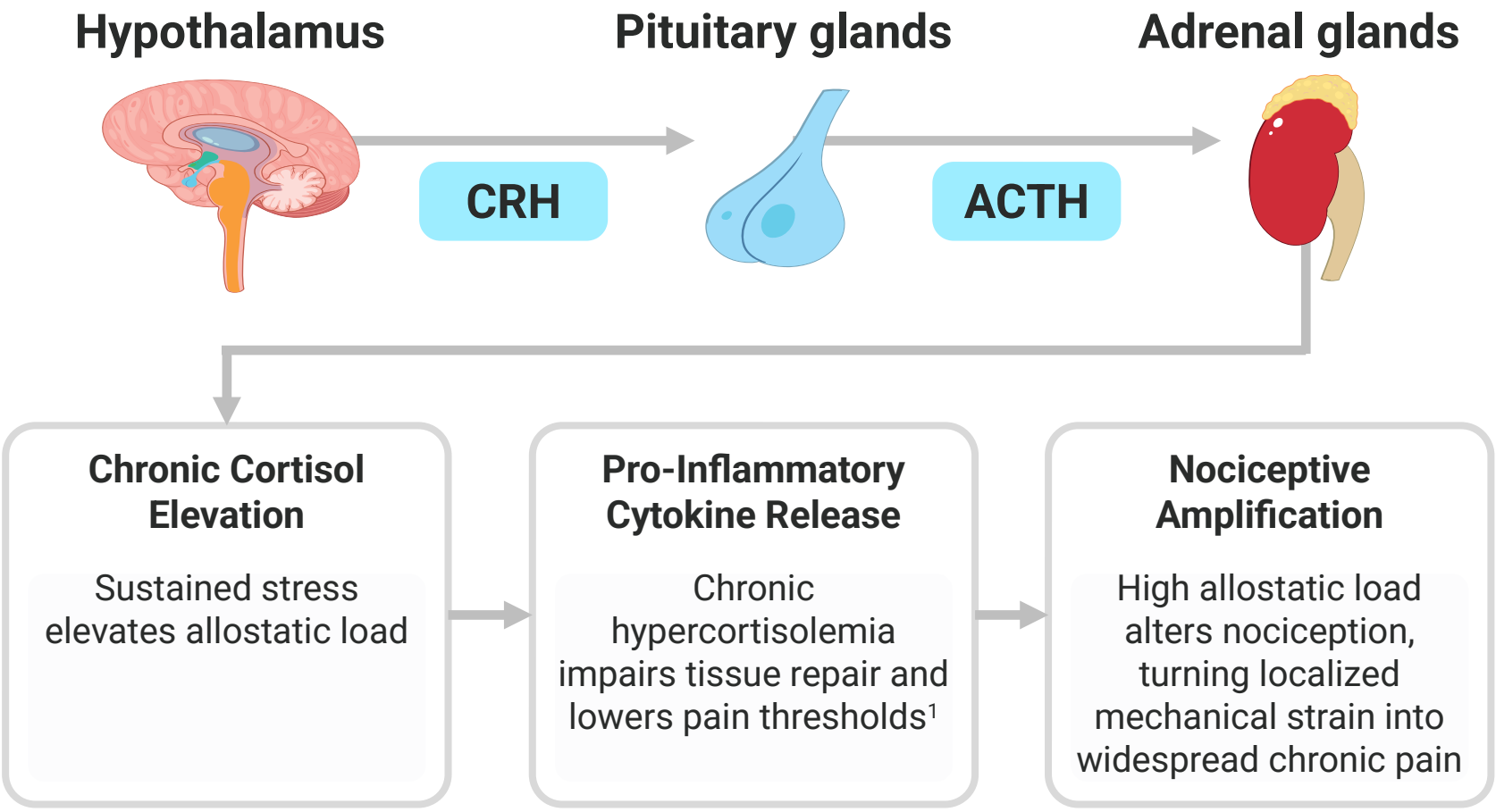
Estrogen modulates type I collagen and ligamentous laxity; androgen signaling supports muscle protein synthesis and bone density[1]

1. Hansen & Kjaer, 2014

2. Bhasin et al., 2018

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Cortisol, Allostatic Load, and Central Sensitization



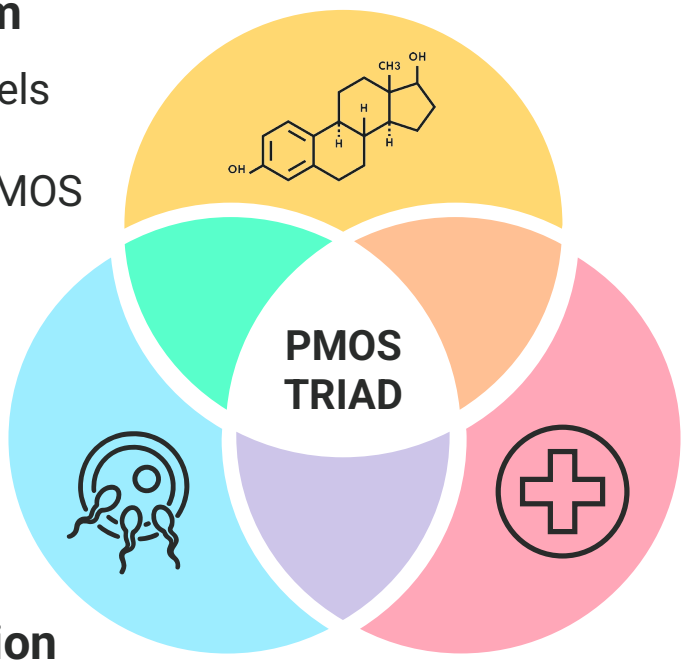
1. Hannibal & Bishop, 2014

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PMOS Pathophysiology and Systemic Markers

Hyperandrogenism

Elevated androgen levels lead to clinical and metabolic features of PMOS



Insulin Resistance

Reduced insulin sensitivity drives hyperinsulinemia and metabolic changes

Insulin resistance drives low-grade systemic inflammation and metabolic dysfunction[1]

Ovulatory Dysfunction

Disrupted follicle maturation and ovulation lead to irregular cycles and infertility

1. Teede et al., 2023

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Chapter Summary

- Chronic stress and cortisol dysregulate the HPG axis, fueling systemic inflammation and weakening connective tissue integrity
- Polyendocrine metabolic ovarian syndrome (PMOS) is driven by an interconnected loop of hyperandrogenism, insulin resistance, and low-grade systemic inflammation
- Systemic and metabolic shifts translate directly into clinical presentations, such as lumbar-pelvic instability, tendinopathy risks, and altered muscle function
- Conditions like endometriosis induce pelvic floor hypertonicity and viscerosomatic convergence, often manifesting as persistent lower back and pelvic pain

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Chapter 2

Movement, Metabolism, and the Modifiable Factors

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Exercise Interventions in PMOS: Evidence Summary

Outcome	High-Intensity Interval Training	Progressive Resistance Training
Intervention example	Short bursts of high-intensity exercise (e.g., 30–60 sec) with recovery intervals	Progressive increase in load (e.g., 2–3 sets, 8–12 reps, 2–3 times/week)
Effect on insulin sensitivity	**Significant improvement** Increased GLUT4 translocation and glucose uptake	**Significant improvement** Increased GLUT4 translocation and glucose uptake
Effect on ovulatory rate	**Moderate–significant increase** More regular menstrual cycles and higher ovulation rate	**Moderate–significant increase** Improves ovulatory function and menstrual regularity

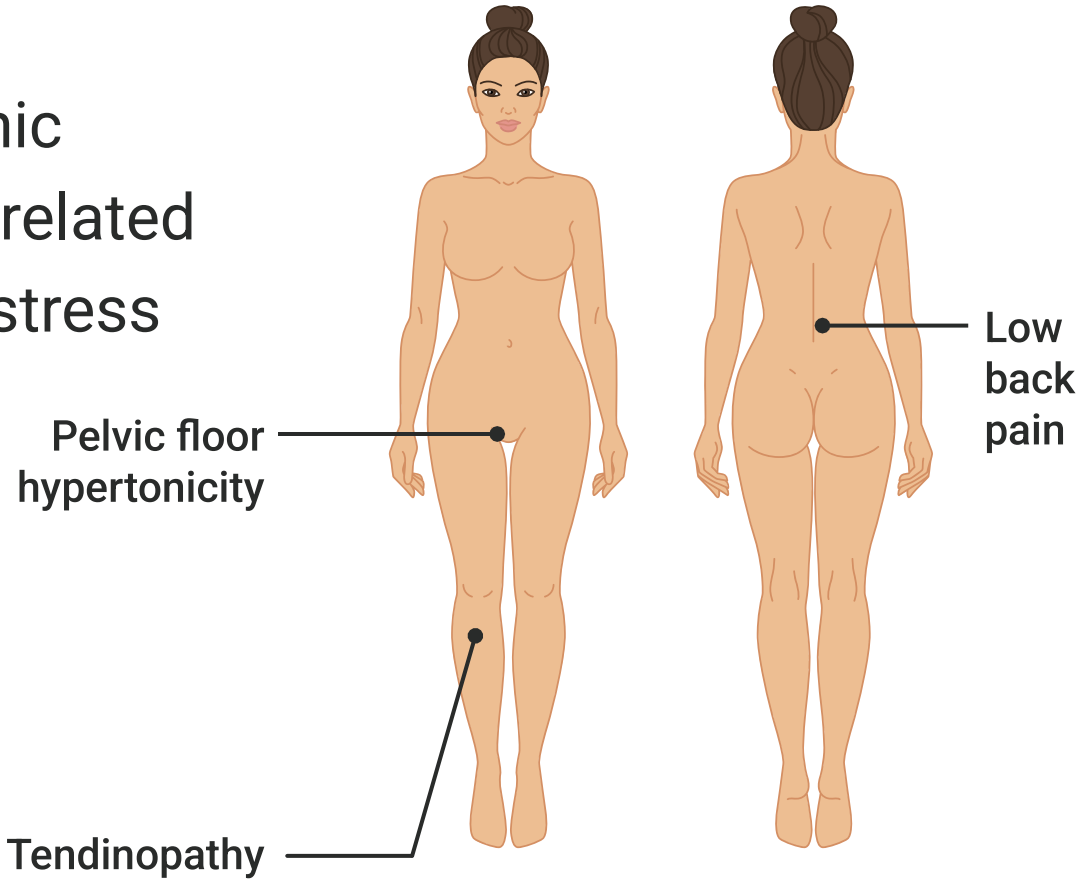
Exercise restores GLUT4 translocation and lowers circulating free androgens.[1]

1. Teede et al., 2023

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PMOS Musculoskeletal Manifestations

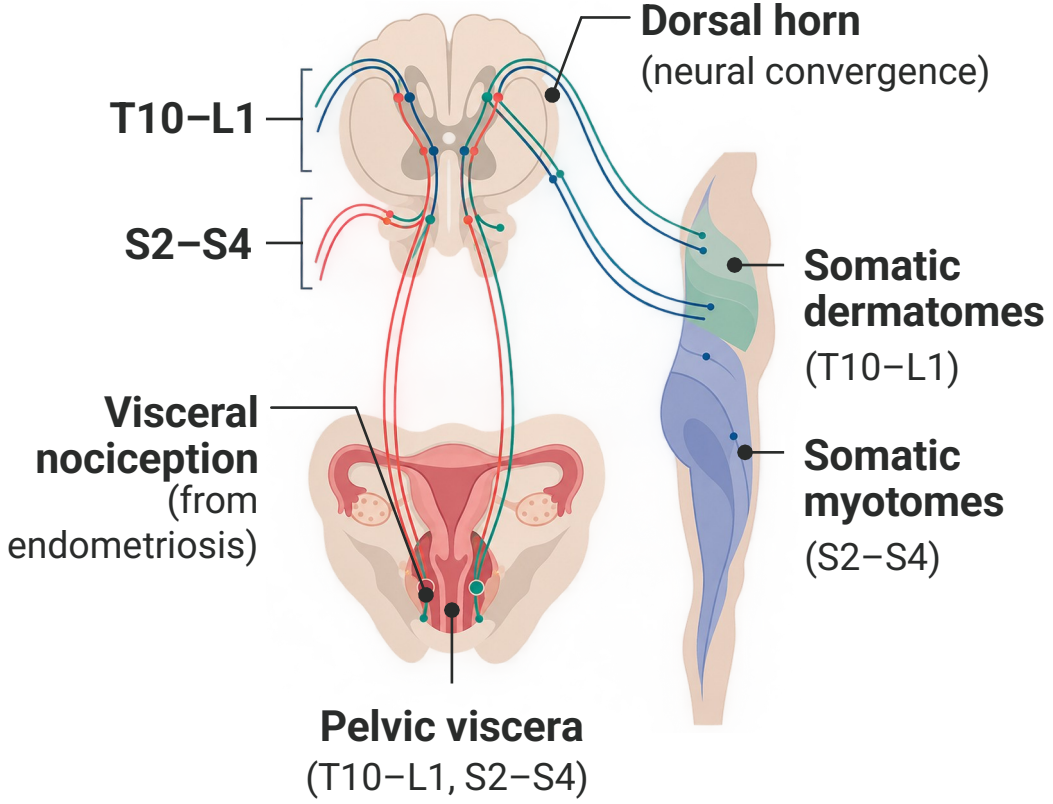
- Systemic inflammation impairs tendon remodeling: chronic pelvic tension correlated with metabolic distress



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Neural Pathways: Viscerosomatic Convergence

Visceral nociception triggers somatic motor output, driving pelvic floor overactivity and lumbar guarding.



Visceral Nociception
(pelvic organs)

↓

Somatic Motor Output
(via shared dorsal horn afferents)

↓

Pelvic Floor Overactivity
(muscle hypertonicity)

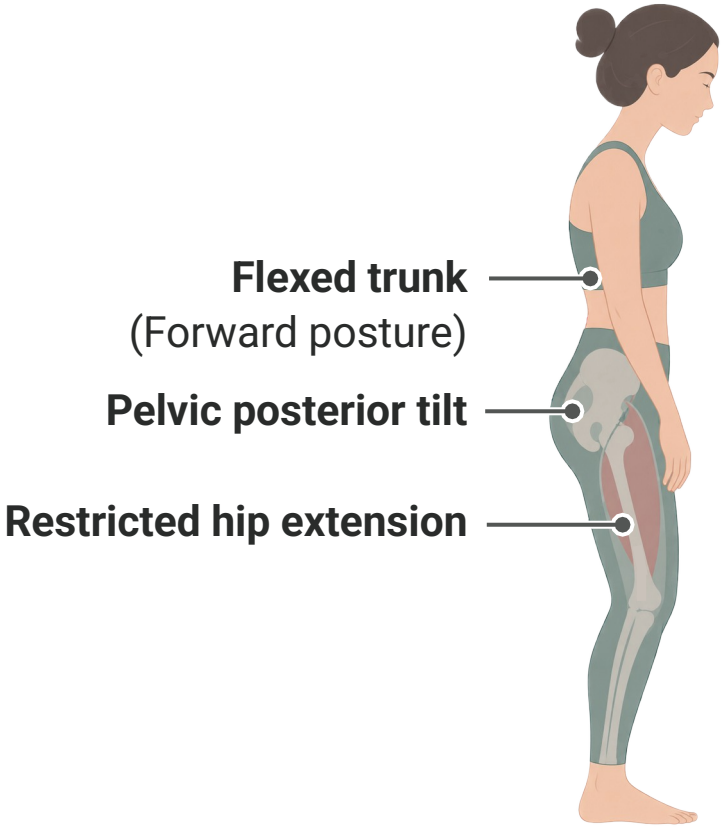
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Lumbar Guarding
(protective response)

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Biomechanical Adaptations in Endometriosis

Persistent abdominal guarding leads to altered diaphragmatic excursions, hip flexor tightness, and gait compensations.



Altered Diaphragmatic Excursions
(due to abdominal guarding)

↓

Hip Flexor Tightness
(limited hip extension)

↓

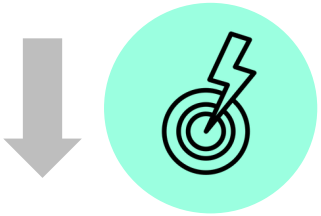
Gait Compensations
(altered movement patterns)

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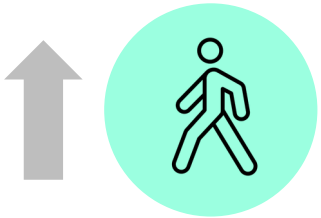
Manual and Visceral Therapy in Chronic Pelvic Pain

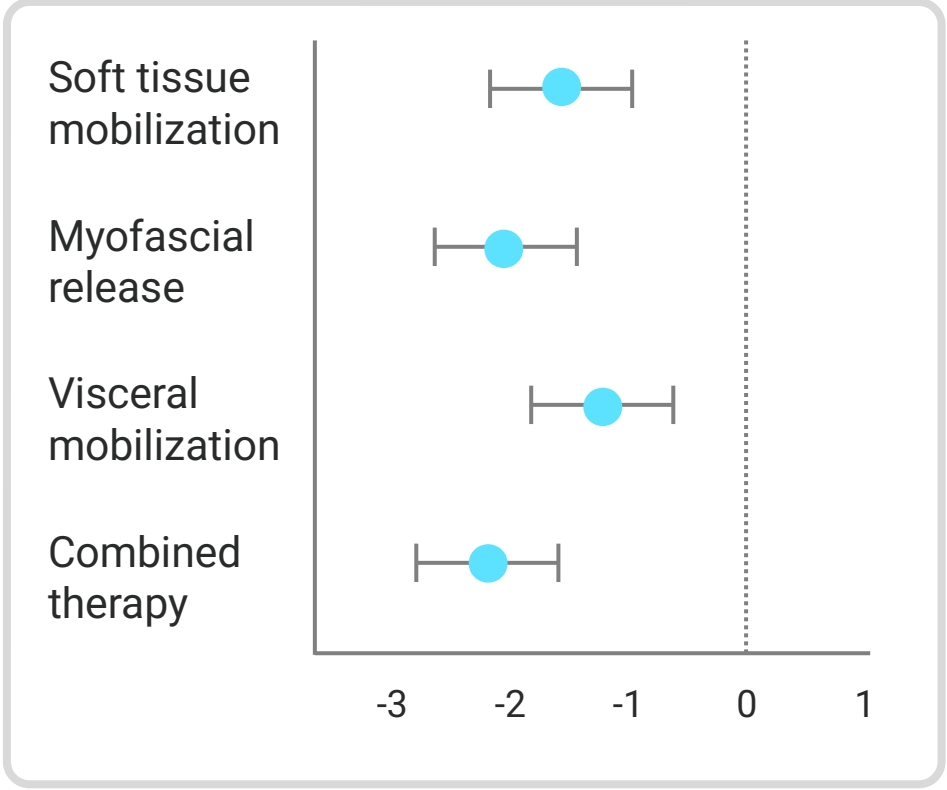
Targeted myofascial release decreases central sensitization and restores tissue glide.

Reduced pain sensitivity



Improved tissue mobility





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Case Study: 31 y/o Female With PMOS, LBP, and Infertility

1. Subjective Presentation

- Chronic pelvic pain
- Low back pain (LBP)
- Irregular menstrual cycles (PMOS)
- Difficulty conceiving (infertility)

2. Examination Findings

- Pelvic floor overactivity
- Lumbar and pelvic movement dysfunction
- PMOS-related hormonal profile and ovulatory dysfunction

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Case Study: 31 y/o Female With PMOS, LBP, and Infertility (cont.)

3. Interventions

- **Multiplanar exercise**
  - Strength, mobility, movement control
- **Metabolic loading**
  - Progressive strength and conditioning
- **Pelvic floor downtraining**
  - Breathwork, relaxation, neuromuscular reeducation
- **Lifestyle comanagement**
  - Nutrition, sleep, stress management

4. Outcome Metrics

- **Pain**
  - VAS (0–10)
- **Function**
  - Pelvic and lumbar mobility
- **Ovulation**
  - Menstrual cycles

Multiplanar exercise, metabolic loading, pelvic floor downtraining, and lifestyle comanagement led to pain resolution and ovulation recovery.

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Chapter Summary

- Exercise Interventions in PMOS: High-intensity interval training (HIIT) and progressive resistance training significantly improve insulin sensitivity (via GLUT4 translocation) and increase ovulatory rates
- Systemic inflammation impairs tendon remodeling, establishing a correlation between chronic pelvic tension, pelvic floor hypertonicity, tendinopathy, and low back pain
- Visceral nociception from pelvic pathology triggers shared dorsal horn afferents (T10–L1, S2–S4), driving somatic motor output, pelvic floor overactivity, and lumbar guarding
- Persistent abdominal guarding leads to altered diaphragmatic excursions, flexed trunk postures, posterior pelvic tilt, restricted hip extension, and gait compensations in individuals with endometriosis
- Combined manual therapy, visceral mobilization, multiplanar exercise, and lifestyle comanagement effectively reduce pain sensitivity, restore tissue mobility, and resolve clinical symptoms in complex patient presentations

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Chapter 3

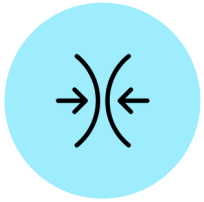
Clinical Interventions, Male Factors, and Collaborative Practice

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Male Infertility and Pudendal Neurovascular Mechanics

Dynamic compression at Alcock's canal reduces arterial inflow and disrupts testicular thermoregulation

01



Dynamic compression at Alcock's canal

02



Reduced arterial inflow to pudendal and testicular circulation

03



Disrupted testicular thermoregulation

04

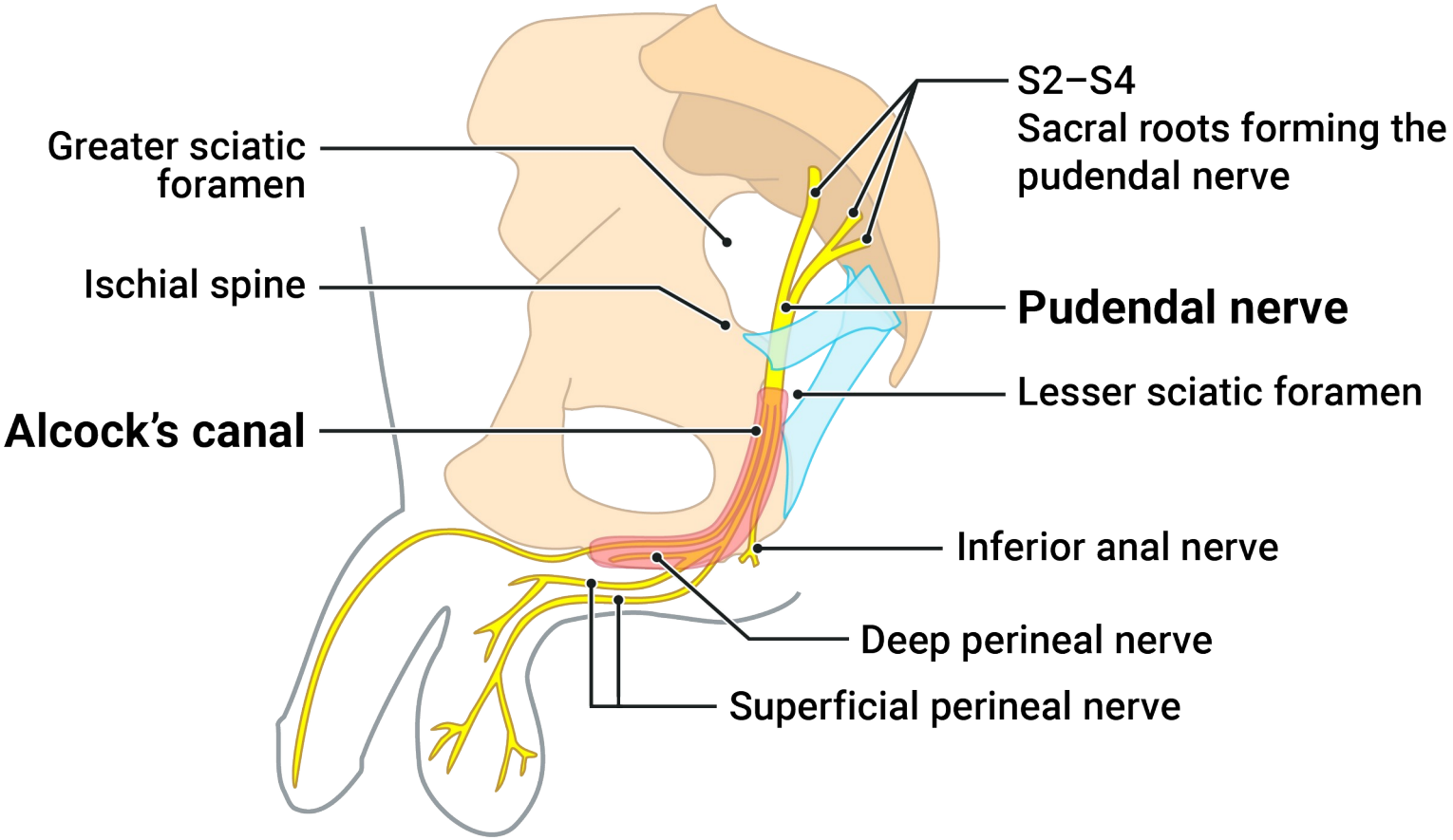


Potential impact on spermatogenesis and male fertility

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Male Infertility and Pudendal Neurovascular Mechanics (cont.)

Dynamic compression at Alcock's canal reduces arterial inflow and disrupts testicular thermoregulation.



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Pelvic Floor Therapy in Male Sexual and Reproductive Health

Pelvic Musculature			
Metric	**Preintervention**	**Postintervention**	**Change**
Pelvic muscle tone (μV)	12.8 ± 4.2	7.6 ± 2.9	↓ 40%
Penile blood flow (ml/min)	18.3 ± 6.7	27.9 ± 8.4	↑ 52%
Pain score (VAS)	6.2 ± 1.8	3.1 ± 1.4	↓ 50%
Ejaculatory latency (seconds)	92 ± 36	148 ± 52	↑ 61%

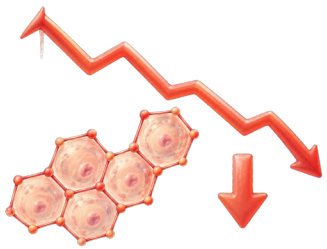
Manual Therapy			
Metric	**Preintervention**	**Postintervention**	**Change**
Pelvic muscle tone (μV)	13.4 ± 4.8	8.1 ± 3.2	↓ 39%
Penile blood flow (ml/min)	17.6 ± 5.9	26.8 ± 7.6	↑ 52%
Pain score (VAS)	5.8 ± 1.7	2.9 ± 1.3	↓ 50%
Ejaculatory latency (seconds)	88 ± 32	139 ± 48	↑ 58%

Downtraining hypertonic pelvic musculature improves blood flow, pain scores, and ejaculatory parameters.

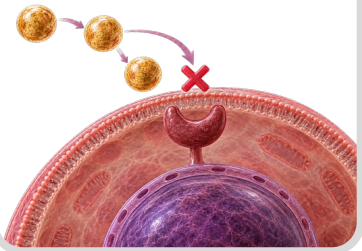
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Hypogonadism, Sarcopenia, and Muscle Degradation

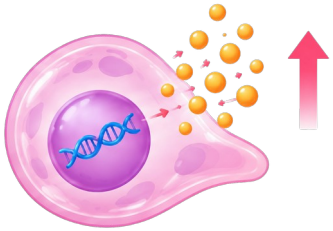
Testosterone Decline



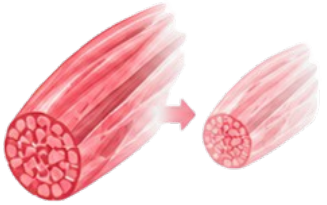
Less AR Binding



Increased Myostatin Expression



Sarcopenia



Age-related testosterone decline accelerates type II muscle fiber atrophy, balance deficits, and frailty.[1]

1. Kovacheva et al., 2010

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Comparative Lifespan Endocrine Shifts in Rehab

Hormonal Axis Shift	Primary Clinical Manifestation	Rehab and Functional Impact
Estrogen loss (postmenopause)	Accelerated bone turnover, collagen degradation[1]	Osteopenia, joint stiffness, pelvic organ prolapse risk
Testosterone decline / metabolic shift	Loss of type II motor units, visceral fat accumulation[2]	Sarcopenia, reduced gait speed, increased fall incidence
HPA axis dysregulation *(chronic stress)*	Cortisol elevation, sleep fragmentation, dysautonomia	Impaired wound healing, lower pain thresholds, fatigue

1. Chidi-Ogbolu & Baar, 2019
2. Cruz-Jentoft et al., 2019

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Case Study: 68 y/o Male With Hypogonadism, Sarcopenia, and Fall Risk

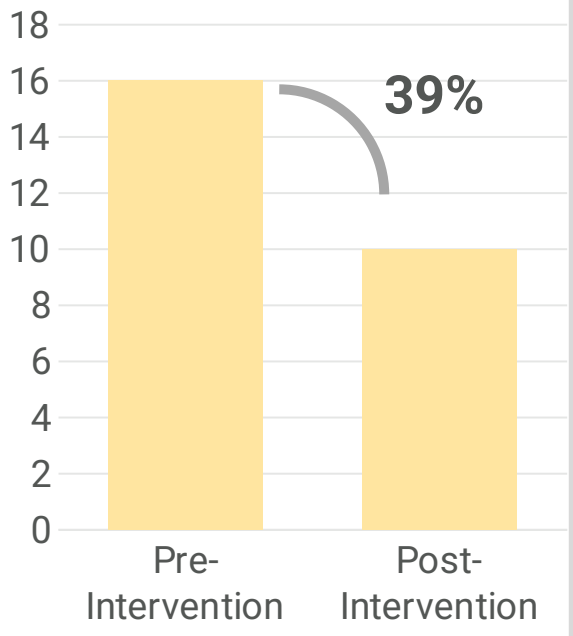
Patient Profile

Age:
68 years old

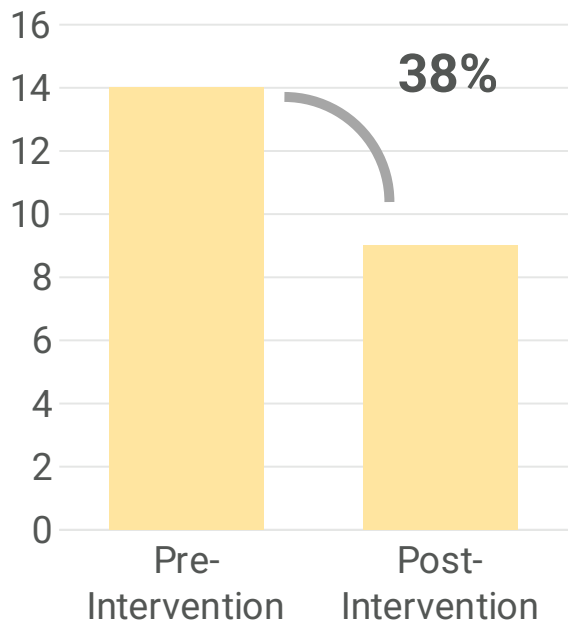
Condition:
Hypogonadism, sarcopenia, fall risk

Goal:
Improve functional mobility and reduce fall risk

Timed Up and Go (TUG)



5-Times Sit-to-Stand (5xSTS)



Integrated high-resistance velocity training, endocrine medical comanagement, and nutritional optimization restored functional mobility.

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Systemic Endocrine Screening Checklist

Red Flags

- Unexplained weight loss
- Metabolic crises
- Severe bone pain

Yellow Flags

- Irregular cycle
- Fatigue
- Slow healing



Systematic screening ensures safe clinical triaging and timely interprofessional medical referrals.

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Pelvic Rehabilitation in Male Factor Subfertility: Clinical Targets and Response Model

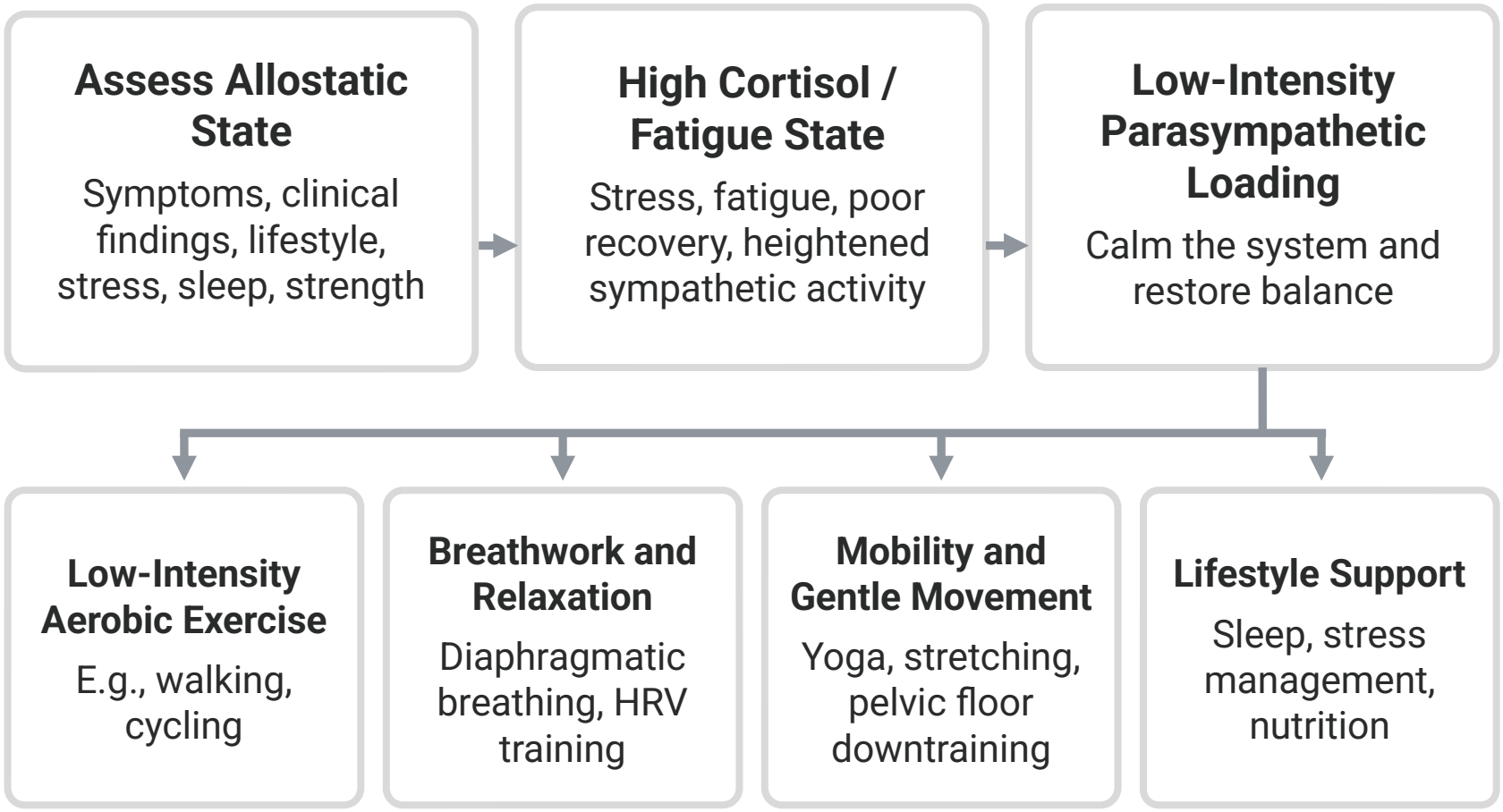
Parameter	Pelvic Muscle Biofeedback (Pre → Post)	Change	Manual Therapy / MFR (Pre → Post)	Change
Pelvic blood flow (PSV, cm/s)	19.4 ± 4.2 → 28.5 ± 5.1	↑ 47%	18.8 ± 4.0 → 27.9 ± 4.8	↑ 48%
Pain score (VAS, 0–10)	6.2 ± 1.5 → 3.1 ± 1.2	↓ 50%	6.4 ± 1.6 → 2.8 ± 1.1	↓ 56%
Ejaculatory latency (IELT, min)	0.6 ± 0.3 → 2.4 ± 0.8	↑ 300%	0.7 ± 0.4 → 2.2 ± 0.7	↑ 214%
Semen volume (mL)	1.9 ± 0.5 → 2.7 ± 0.6	↑ 42%	1.8 ± 0.4 → 2.6 ± 0.5	↑ 44%
Sperm motility (% Total)	31.2 ± 6.4 → 44.5 ± 7.1	↑ 43%	30.5 ± 5.8 → 43.1 ± 6.5	↑ 41%



Downtraining hypertonic pelvic musculature improves blood flow, pain scores, and ejaculatory parameters.

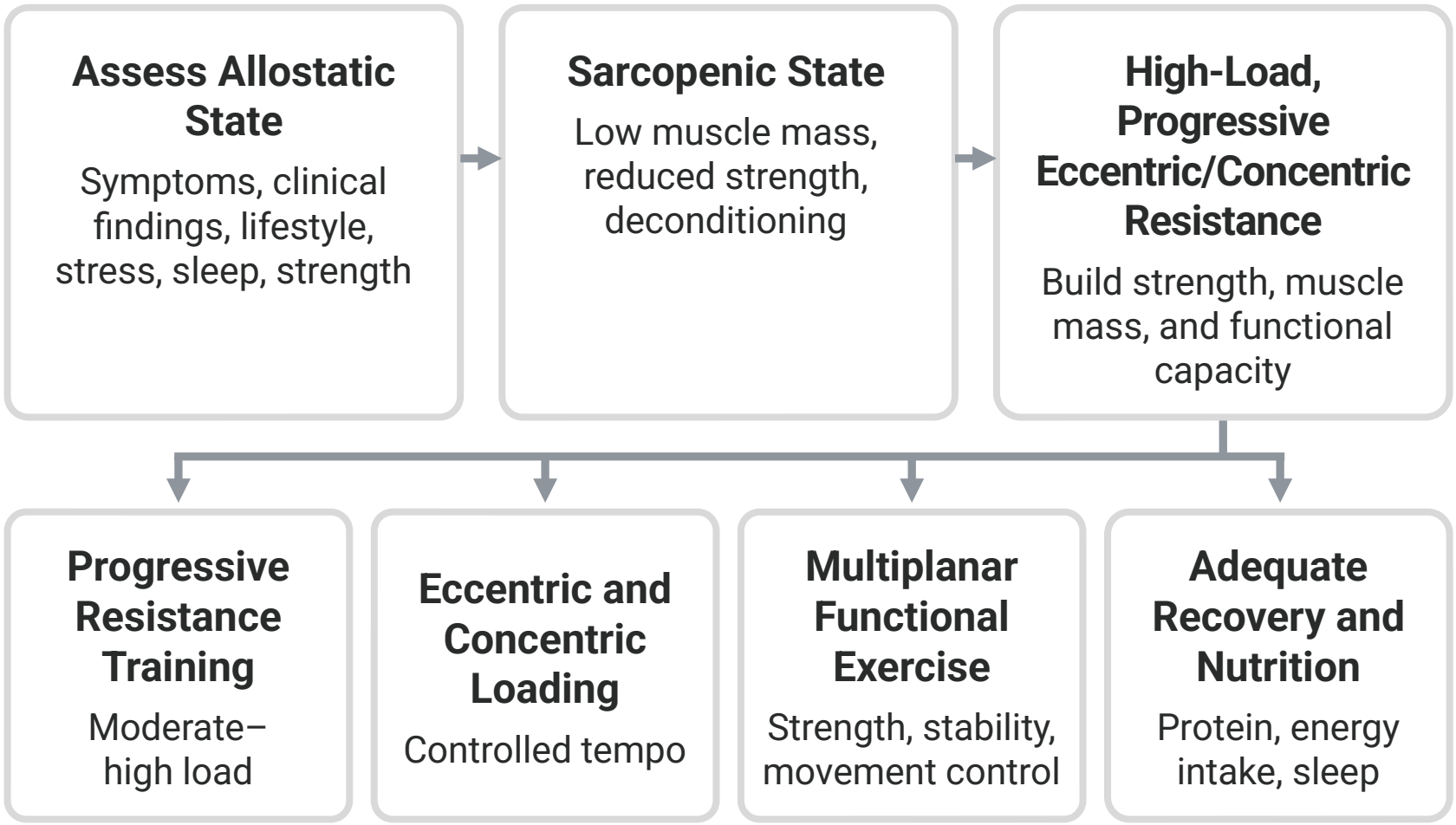
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Exercise Prescription: Matching Load to Allostatic State



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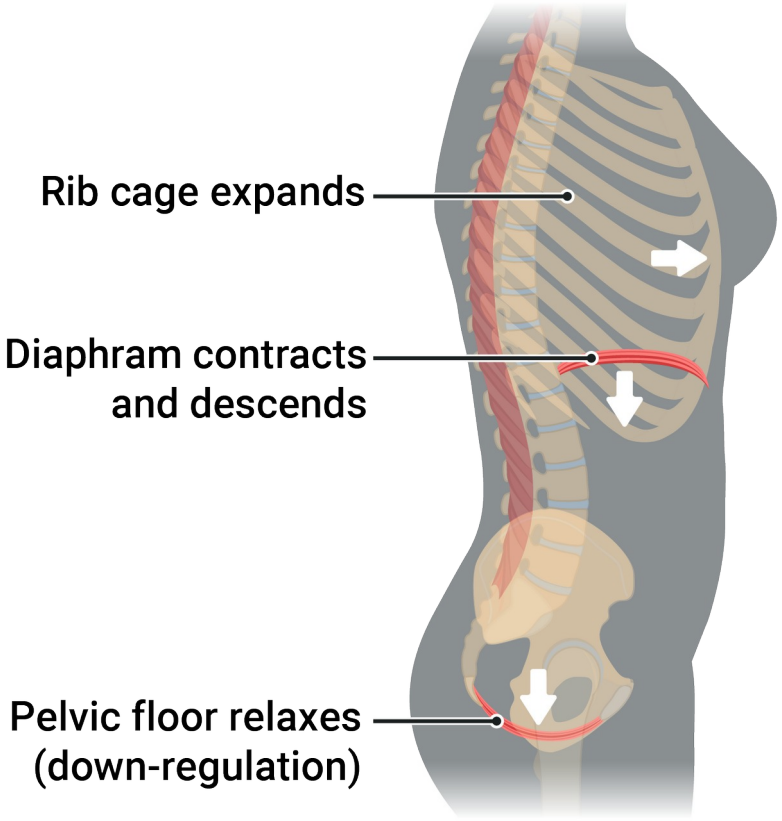
Exercise Prescription: Matching Load to Allostatic State (cont.)



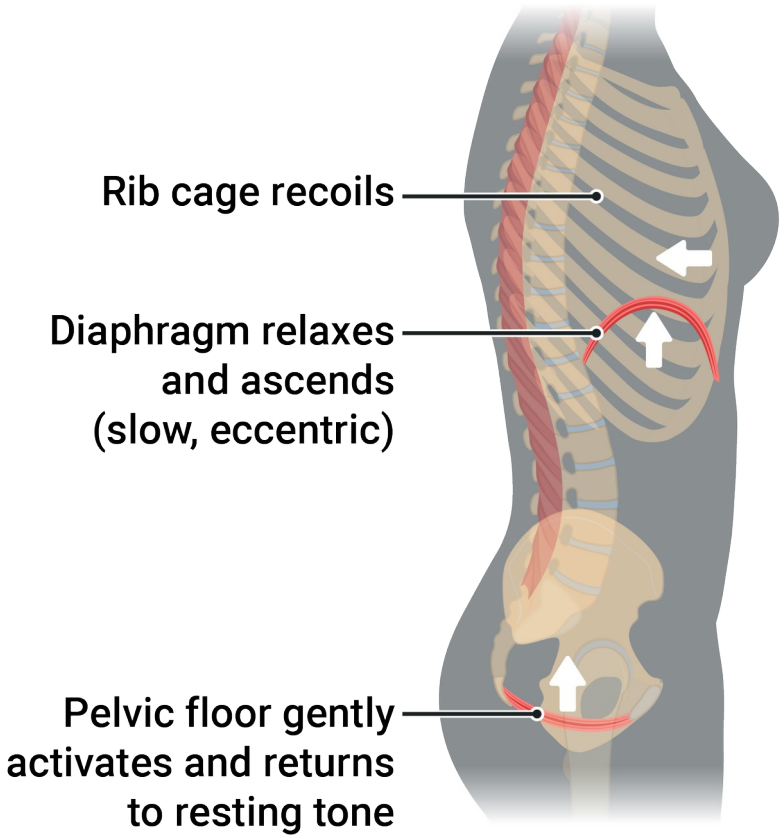
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ANS Regulation and Diaphragmatic Modulation

Inhalation
Diaphragm descends



Exhalation
Diaphragm ascends (slow, eccentric)



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ANS Regulation and Diaphragmatic Modulation (cont.)



Slow eccentric diaphragmatic breathing

Activates parasympathetic pathways, **lowering systemic tone and pelvic resting pressure**



Vagal tone

- Activates parasympathetic nervous system
- Reduces sympathetic overactivity



Systemic tone

- Lowers stress response
- Promotes relaxation and recovery



Pelvic floor down-regulation

- Reduces resting pressure
- Decreases muscle overactivity

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Multidisciplinary Care Pathways

Pelvic health physical and occupational therapy, coordinating with

Reproductive Endocrinology (REI)

- Fertility and hormonal evaluation
- Reproductive health management
- Shared care for complex endocrine cases

Urology

- Lower urinary tract symptoms (LUTS)
- Pelvic pain
- Sexual dysfunction
- Diagnostic evaluation and treatment

Endocrinology

- Hormonal assessment and optimization
- Metabolic and endocrine disorders
- Management of complex endocrine patients

Nutrition

- Nutritional assessment
- Diet and lifestyle guidance
- Support for hormonal and metabolic health
- Weight management

Establishing clear communication channels and referral **criteria enhances long-term outcomes for complex endocrine patients.**

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Chapter Summary

- Hypertonic pelvic floor overactivity can compress the neurovascular bundle at Alcock's canal
- Routine screening for endocrine red flags and yellow flags allows rehab clinicians to identify metabolic and androgen dysfunction early and triage appropriately
- Exercise prescription must respect neuroendocrine load
- Rehab professionals expand fertility outcomes not by diagnosing medical pathology, but by treating musculoskeletal, autonomic, and tissue-glide barriers while collaborating early with REI, urology, endocrinology, and nutrition

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Master Clinical Takeaways

Screen Hormonal Health

Screen hormonal health in chronic pain and consider endocrine factors in assessment and management

Match Exercise to Allostatic Load

Match exercise prescription to the individual's allostatic state (e.g., fatigue vs. sarcopenia)

Downtrain Pelvic Tension

Use breathing, mobility and targeted strategies to downtrain pelvic tension and reduce resting pressure

Collaborate Early

Collaborate early with REI, urology, endocrinology, and nutrition for comprehensive care

Focus on Long-Term Outcomes

Integrate screening, loading, downtraining, and comanagement to improve long-term outcomes for complex endocrine patients

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Bibliography

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