PHALANX ANABOLICS

THE HARM REDUCTION MANUAL

FREE SAMPLER

This is a free preview of the complete manual — an evidence-based educational and harm-reduction reference on performance-enhancing drugs. Edition 1, Version 1.0. 626 pages · Parts I–X · 67 units.

The pages that follow are drawn directly from the full work: the front matter and full table of contents, an excerpt from the Performance-Enhancing Drugs section, a complete chapter on cardiovascular monitoring, and the opening of the Myth Library.

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THE HARM REDUCTION MANUAL

SCIENCE · KNOWLEDGE · RESPONSIBILITY

Edition 1 · Version 1.0

An evidence-based educational & harm-reduction reference on performance-enhancing drugs.

Medical disclaimer

Medical disclaimer. This manual is an educational and harm-reduction resource. It is not medical advice, and it is not a substitute for assessment, diagnosis or treatment by a qualified healthcare professional. It does not recommend, endorse or encourage the use of any drug or substance, and it contains no dosing or usage instructions. Performance-enhancing drugs carry serious and sometimes irreversible health risks, and many are controlled substances whose possession or supply is regulated by law in most coun tries. Reference ranges and figures vary between laboratories and sources; interpret any result against the reporting laboratory's ranges and your clinician's advice. Always seek the advice of a qualified clini cian with any questions about your health, and in an emergency contact your local emergency services immediately. The publisher and contributors accept no liability for decisions made on the basis of this ma terial.

This edition presents the complete manual: Parts I–X. Every substantive claim is supported by a real, veri fied reference (Vancouver, with DOI/PMID) and carries a visible Research Confidence rating, within a strict harm-reduction scope — no dosing, no protocols, no instruction on use. The bookmarks panel of your PDF reader mirrors the contents below and jumps to any Part or chapter.

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PART IV · PERFORMANCE-ENHANCING DRUGS

Chapter 70

TESTOSTERONE

"The dose makes the poison."

— ATTRIBUTED TO PARACELSUS, 16TH CENTURY

70.1 Clinical overview

Testosterone is the principal male sex hormone (androgen) and the reference compound against which every other anabolic–androgenic steroid is measured. It is produced mainly by the Leydig cells of the testes under the control of the brain, and it circulates to almost every tissue in the body, where it supports reproductive function, muscle and bone, red-blood-cell production, libido, mood and energy.

In medicine, testosterone is a licensed treatment for men with **hypogonadism** — a genuine deficiency of the hormone caused by disease of the testes or of the brain's hormonal control centres.[4] Outside medicine, testosterone and its chemical relatives are used in supraphysiological amounts — quantities far greater than the body would ever make — to increase muscle size and strength. This chapter explains what testosterone is, how it works, what the evidence does and does not show about its effects, and, above all, the risks it carries and how those risks can be recognised and reduced. It contains no dosing information and no usage instructions; its purpose is understanding, not administration.

> ☆ **PHALANX INSIGHT**
>
> Testosterone is not an exotic drug — it is a hormone every human body already makes and depends on. That is precisely why using it in large external amounts is consequential: the body reads the extra hormone as its own and responds by shutting down its natural production. Understanding that single feedback principle explains most of what follows in this chapter.

70.2 Why it matters

Anabolic–androgenic steroids are among the most widely used performance-enhancing drugs in the world. A meta-analysis of population studies estimated a global lifetime prevalence of use of about 3.3% overall — roughly 6.4% among men and 1.6% among women — with much higher rates among people involved in recreational strength training.[1] Testosterone is the most commonly used of these agents and the foundation of the class.

Because use is common and largely unsupervised, the harm that follows is often preventable. Most of the serious problems associated with testosterone are not mysterious: they are predictable consequences of known physiology, they develop gradually, and they can be detected early through simple monitoring. A reader who understands the mechanisms in this chapter is better placed to recognise a warning sign, ask a clinician the right question, and act before a reversible problem becomes a permanent one.

70.3 History

The isolation and chemical synthesis of testosterone in 1935 was one of the landmark achievements of twentieth-century endocrinology. The hormone was first isolated from testicular tissue and, in the same year, synthesised independently by the chemists Adolf Butenandt and Leopold Ružička — work for which both were later recognised with the Nobel Prize in Chemistry.[2] Within a few years, testosterone was being investigated as a treatment for men whose bodies produced too little of it.

From these legitimate clinical origins, use spread — first into strength sports in the mid-twentieth century, and then far more widely into recreational training communities. The scientific understanding of testosterone's benefits in deficiency and its harms in excess has developed in parallel ever since, and it is that evidence base, rather than tradition or anecdote, that this chapter draws on.

DID YOU KNOW?

The word “anabolic” refers to tissue-building, and “androgenic” to the development of male characteristics. Chemists spent decades trying to separate the two properties — to make a purely anabolic steroid with no androgenic effect. No compound has ever fully achieved this, because both effects are produced through the same androgen receptor.

70.4 Chemistry

Testosterone is a **steroid** — a molecule built on the characteristic four-ring carbon skeleton shared by cholesterol, cortisol and the sex hormones. The body manufactures it from cholesterol through a series of enzymatic steps. Its structure is what allows it to pass through cell membranes and act inside the cell, and small modifications to that structure are what distinguish testosterone from the other steroids described in this Part.

Pure testosterone is broken down very rapidly by the liver, which is why it is not useful when simply swallowed.[3] Pharmaceutical preparations overcome this in two main ways. In injectable products, a fatty-acid chain (an **ester**) is attached to the testosterone molecule; the ester makes the compound oil-soluble and slows its release and breakdown, so that a single injection acts over a longer period before the ester is cleaved off and active testosterone is freed.[3] Other preparations use gels or patches applied to the skin. Understanding esterification matters here only as chemistry — it explains why different products behave differently in the body, not how any product should be used.

70.5 Mechanism of Action

Testosterone works by entering cells and binding to a protein called the **androgen receptor (AR)**. The hormone–receptor pair then moves into the cell nucleus and switches on specific genes, increasing the production of proteins — including the contractile proteins of muscle — and stimulating the bone marrow to make more red blood cells. This is the route by which testosterone builds tissue.

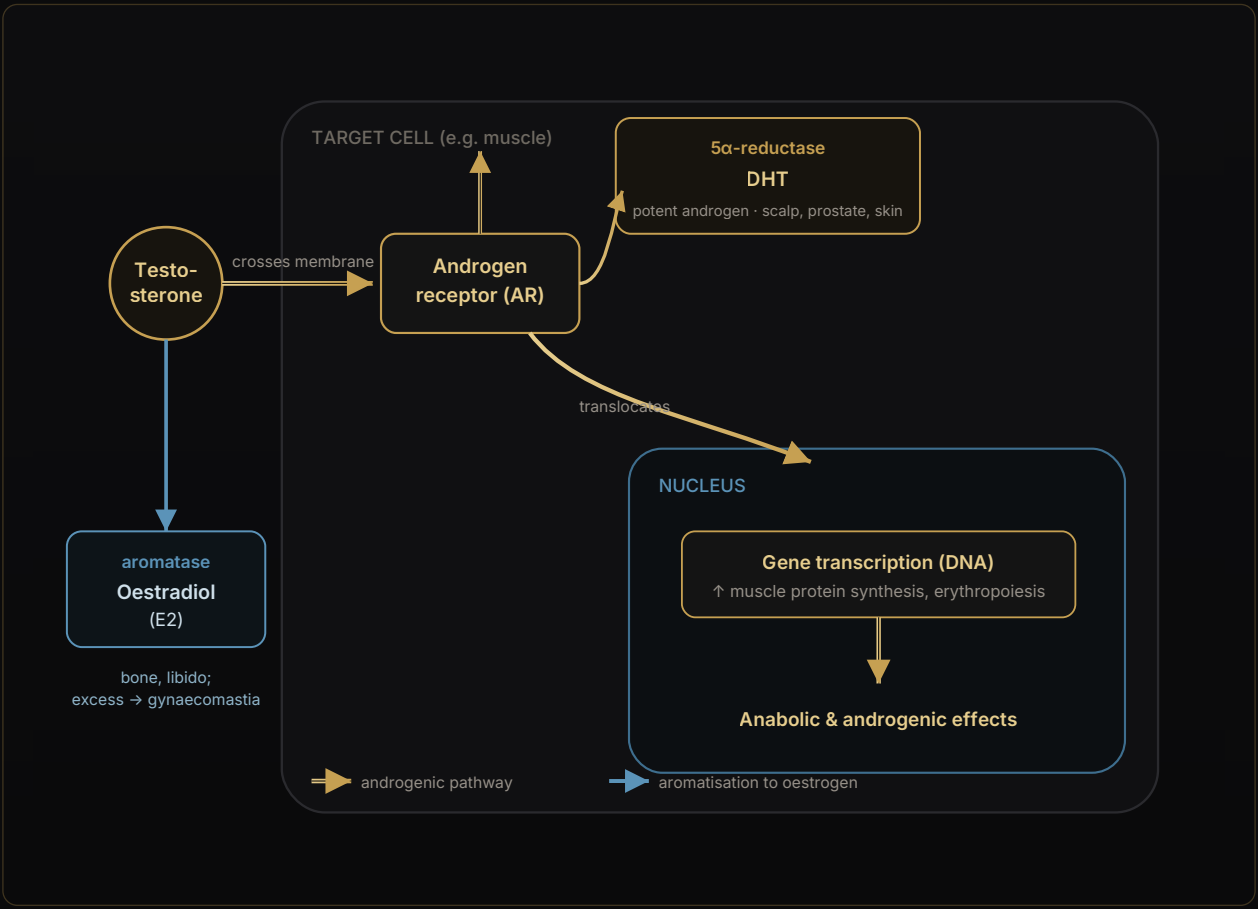

Figure 70.1 — Testosterone acts directly through the androgen receptor and also through two conversion pathways: aromatisation to oestradiol and 5α-reduction to dihydrotestosterone. Many of testosterone's effects — wanted and unwanted — are effects of these metabolites, not of testosterone itself.

Two conversion pathways are essential to understanding testosterone's effects. An enzyme called **aromatase** converts a portion of testosterone into **oestradiol**, the principal oestrogen; oestradiol is necessary in men for bone health, libido and other functions, but in excess it can cause breast-tissue growth (gynaecomastia).[14] A second enzyme, **5α-reductase**, converts testosterone into **dihydrotestosterone (DHT)**, a more potent androgen responsible for effects on the scalp, prostate and skin. Because these metabolites drive some of the most familiar side effects, testosterone cannot be understood as a single actor — it is the head of a small family of active hormones.

The control system: the HPTA

The body regulates its own testosterone through a feedback loop known as the **hypothalamic–pituitary–testicular axis (HPTA)**. The hypothalamus in the brain releases gonadotropin-releasing hormone (GnRH), which prompts the pituitary gland to release luteinising hormone (LH) and follicle-stimulating hormone (FSH); these in turn instruct the testes to produce testosterone and sperm. When testosterone rises, it signals back to the brain to reduce this output — a thermostat that keeps levels stable.

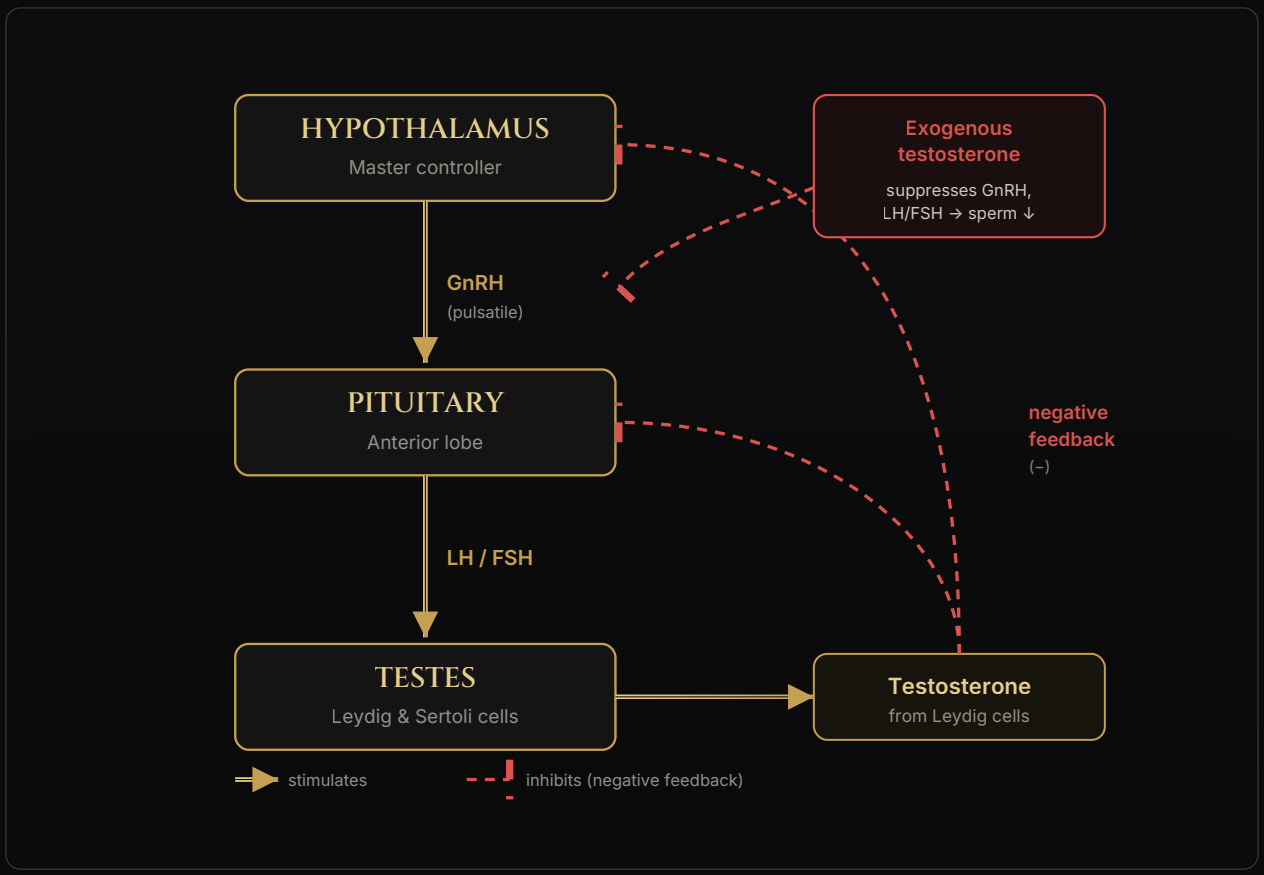

Figure 70.2 — The HPTA feedback loop. Introducing testosterone from outside the body is read by the brain as an abundance of the hormone, so it reduces its own signals (LH and FSH). Because those same signals maintain sperm production, suppression of the axis reduces fertility for as long as it persists.

> **CLINICAL NOTE**
>
> This feedback loop is the reason exogenous testosterone suppresses natural production and reduces sperm counts, often to very low levels. It is also why low LH and FSH on a blood test, alongside a high testosterone reading, is the classic signature of externally supplied androgen rather than a naturally high level.

70.6 Pharmacology

Once active testosterone is in the bloodstream, most of it travels bound to carrier proteins — chiefly **sex hormone-binding globulin (SHBG)** and albumin — with only a small "free" fraction able to act on tissues. The balance between bound and free hormone is clinically important, because two people with the same total testosterone can have quite different free levels depending on their SHBG.

Testosterone is cleared by the liver and its breakdown products are excreted in the urine. Different pharmaceutical forms — injections of various esters, skin gels, and others — differ mainly in how quickly the hormone appears and how long it persists, which shapes how stable blood levels are over time. This manual deliberately does not describe dosages, frequencies or regimens: the relevant point for harm reduction is that the pharmacological form influences how sharply hormone levels rise and fall, and that larger, less stable swings tend to be associated with more pronounced side effects.

70.7 Approved Medical Uses

● STRONG

Testosterone therapy is well established for one clear indication: **male hypogonadism**, in which the body cannot produce adequate testosterone because of a problem in the testes (primary hypogonadism) or in the hypothalamus or pituitary (secondary hypogonadism). The Endocrine Society's clinical practice guideline recommends making this diagnosis only in men who have both symptoms and unequivocally, consistently low morning testosterone confirmed on more than one occasion using a reliable assay, and it advises against treatment in several specific situations — for example untreated prostate cancer, a recent cardiovascular event, untreated severe sleep apnoea, or a man planning fertility in the near term.[4]

Testosterone also has defined uses in specific contexts such as delayed puberty and as part of gender-affirming care. In all of these, treatment is aimed at restoring or establishing physiological levels under medical supervision — a fundamentally different situation from the supraphysiological use described next.

EVIDENCE SNAPSHOT · TESTOSTERONE FOR SYMPTOMATIC DEFICIENCY

In men with genuine hypogonadism, restoring testosterone to normal levels reliably improves sexual desire and can improve mood, bone density and body composition.[4,7] The benefits are clearest in those who are genuinely deficient; they do not translate into a rationale for pushing an already-normal man's levels far above the physiological range.

● Research Confidence: Strong for symptomatic deficiency

70.8 Off-label and non-medical use

The great majority of testosterone used worldwide is not prescribed for deficiency. It is used — usually in supraphysiological amounts, often alongside other anabolic steroids — to increase muscle mass, strength and, sometimes, athletic performance or appearance. In this setting the goal is not to restore a normal level but to exceed it substantially, and it is this deliberate excess that generates the risk profile described later in the chapter.

It is important to state plainly what changes when testosterone is used this way. At physiological replacement levels the body's own production is being supplemented; at supraphysiological levels the body's own production is switched off entirely (Figure 70.2), the conversion pathways to oestradiol and DHT are driven harder, and red-blood-cell production, blood pressure and cholesterol handling all shift. The effects sought and the harms risked come from the same source: an amount of hormone the body was never designed to encounter.

70.9 Reported performance effects

● STRONG

That supraphysiological testosterone increases muscle size and strength is one of the few things about anabolic steroids that is firmly established by high-quality evidence. In a landmark randomised, placebo-controlled trial, healthy men given supraphysiological testosterone gained fat-free mass, muscle size and strength — and did so *both* with and without weight training, though the combination produced the largest gains.[5] A later controlled dose–response study confirmed that these effects, along with the rise in haemoglobin and the fall in fat mass, scale with the amount of hormone and the resulting blood concentration.[6]

The picture in older men treated at more modest levels is more measured: in the coordinated Testosterone Trials, treatment improved sexual function and mood but produced no significant benefit for vitality or walking distance.[7] The honest summary is that testosterone genuinely builds muscle in a dose-related way, but that “builds muscle” and “improves health, performance or wellbeing” are not the same claim, and the evidence supports the first far more strongly than the second.

PART V · HEALTH MONITORING

Chapter 99

CARDIOVASCULAR MONITORING

“The heart is the highest-stakes organ here — and the one least likely to warn you in advance.”

— ON WATCHING THE HEART

99.1 Why the heart comes first

Of everything worth monitoring, the heart carries the **highest stakes and the least reversibility**. Long-term anabolic-steroid use is associated with a thicker, less efficient left ventricle — reduced pumping (systolic) function and impaired relaxation (diastolic) function[2] — and with a greater burden of coronary plaque.[1] This chapter expands the "heart and vessels" panel of the monitoring map from Chapter 98: what changes in the heart, how it is told apart from harmless athletic adaptation, how it is detected, and what it means over time. As always, it is about recognition and knowing when to seek care — not protocols.

> ☆ **PHALANX INSIGHT**
>
> The defining feature of cardiac harm is that it is *quiet*. A person can feel strong, train hard and have no symptoms while the heart muscle thickens, its function slips, and plaque accumulates in the coronary arteries. The organ that matters most is also the one least likely to give an early warning you can feel — which is precisely why it is imaged and tracked rather than judged by how you feel. Feeling fine is not evidence that the heart is fine.

99.2 What changes in the heart

● **STRONG**

Two linked changes are well documented. First, the **left ventricle thickens** and its function declines — careful echocardiographic studies show reduced systolic performance and impaired diastolic relaxation in long-term users compared with non-users.[1,2] Second, this dysfunction is often **subclinical**: sensitive techniques such as tissue-Doppler and strain imaging pick up early myocardial impairment before any symptom appears.[3] The prospective HAARLEM study confirmed that steroid use induces measurable left-ventricular hypertrophy and functional change over a period of use.[4] The pattern matters because it can progress silently.

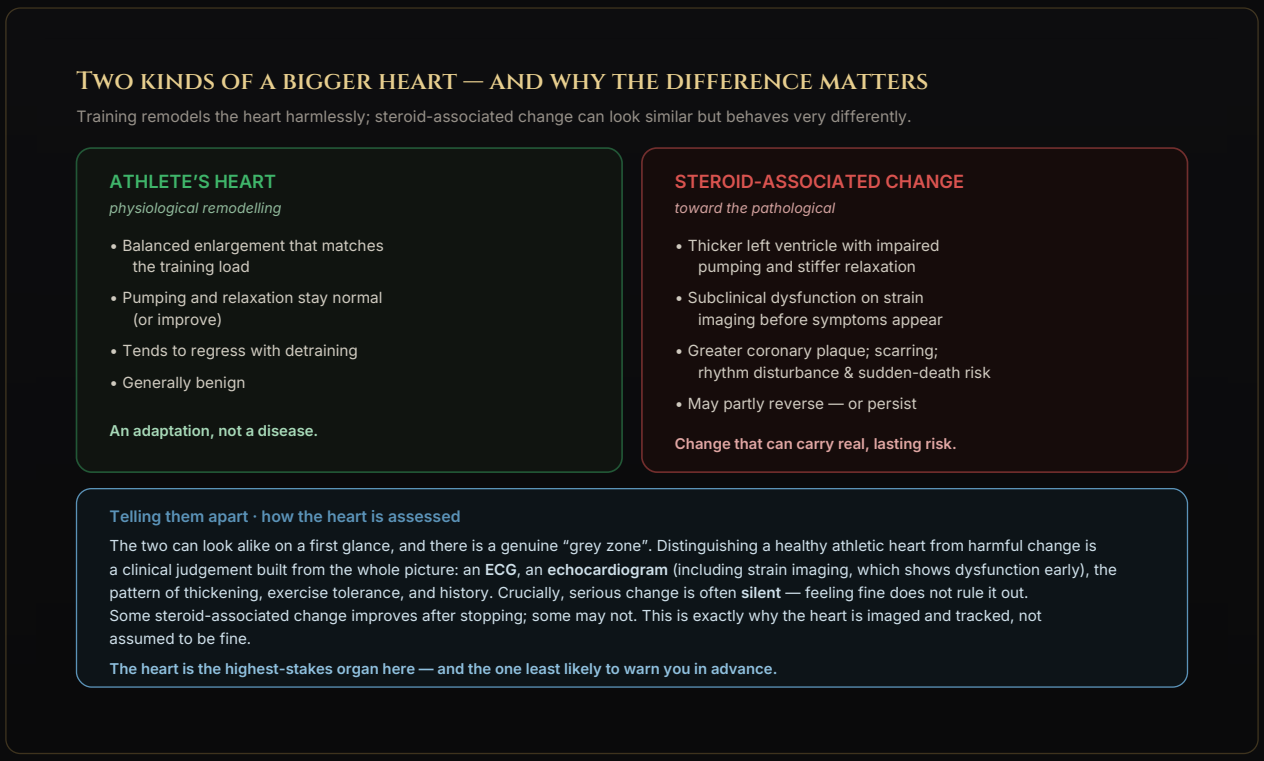

Figure 99.1 — Athletic adaptation versus steroid-associated cardiac change: they can look alike, behave very differently, and are told apart by imaging and clinical judgement — not by symptoms.

99.3 Athlete's Heart versus Pathology

A crucial and easily-confused distinction is between **"athlete's heart"** and pathological change. Endurance and strength training remodel the heart physiologically — a balanced enlargement that matches the workload, preserves or improves function, and tends to regress when training stops. This is **benign**.[6] The difficulty is that this healthy adaptation can superficially resemble the early stages of harmful hypertrophy or cardiomyopathy, creating a genuine **"grey zone"**. Distinguishing the two is a clinical judgement drawing on the pattern of thickening, the function on imaging, the response to exertion, and the history — not something to self-diagnose. It is one of the main reasons cardiac assessment in this context belongs with a clinician.

99.4 The Coronary Arteries

Beyond the muscle itself, anabolic steroids are associated with **coronary atherosclerosis** — plaque building in the arteries that feed the heart. Coronary-CT imaging in long-term users has shown greater plaque volume than in non-users,[1] and coronary calcification has been documented in steroid-using bodybuilders.[7] This is the substrate for heart attacks, and — like the muscle changes — it accumulates without symptoms until it is advanced. It is also why the lipid shift covered in Chapter 98 matters so much: the two processes are connected.

99.5 Rhythm and sudden events

The most feared cardiac outcomes are **arrhythmia, cardiomyopathy and sudden cardiac death**. Forensic case series have documented sudden cardiac death in anabolic-steroid users, with hearts showing hypertrophy and fibrosis (scarring) at autopsy,[8] and clinical series describe steroid-associated **heart failure**.[9] These are, mercifully, less common than the silent structural changes — but they are the reason the structural changes are taken seriously, because a scarred, hypertrophied heart is an electrically unstable one.

99.6 The blood vessels

The vascular system is part of the same picture. Steroid use is associated with increased **arterial stiffness** and elevated blood pressure,[11] and with **endothelial dysfunction** — the artery lining loses some of its ability to dilate normally, an early step toward atherosclerosis.[10] Blood pressure and vascular health get their own fuller treatment later in this part; here the point is that the heart and the vessels feeding and loading it are monitored together, not in isolation.

99.7 How the heart is assessed

Because the harm is silent, detection depends on **measurement, not symptoms**. The core tools are the **ECG** (electrocardiogram), which records the heart's electrical activity and can flag hypertrophy, strain patterns and rhythm problems, and the **echocardiogram**, which images the muscle's thickness and function — with **strain imaging** able to reveal dysfunction early, before it is otherwise apparent.[3,4] Exercise tolerance and history add context, and distinguishing benign adaptation from pathology draws all of these together.[6] None of this is a self-assessment: interpreting cardiac imaging is a specialist task, which is the practical reason cardiovascular monitoring means involving a clinician.

99.8 Does it reverse?

The honest answer is **sometimes, partly, and not always**. The HAARLEM study found that steroid-induced left-ventricular hypertrophy and dysfunction were **reversible to a degree** after stopping,[4] which is genuinely encouraging. But longer-term follow-up suggests some structural change can **persist**,[5] and established cardiomyopathy or coronary plaque does not simply vanish.[9] The uncertainty itself is the argument for monitoring: catching change early, while it is most likely to be reversible, is far better than discovering it once it is fixed.

⚠ RED FLAG · THIS IS AN EMERGENCY

Some cardiac symptoms are not "monitoring" matters but **emergencies** needing urgent help now: **chest pain or pressure**, especially spreading to the arm, jaw or back; **sudden breathlessness**; **fainting or collapse**; a **racing, pounding or irregular heartbeat** that will not settle; or signs of a **stroke** (face droop, arm weakness, speech difficulty). Do not wait for the next scan or blood test — contact emergency services. Separately, new breathlessness on exertion, swelling of the legs, or reduced exercise tolerance warrants prompt clinical assessment.

99.9 Cardiovascular monitoring at a glance

TABLE 99.1 — WHAT IS WATCHED, AND WHY

Target	How it is assessed	Why it matters
LV structure & function	Echocardiogram (incl. strain), ECG	Silent hypertrophy & dysfunction
Athlete's heart vs pathology	Pattern, function, history — clinician	The "grey zone" needs judgement
Coronary arteries	Imaging; lipid & risk context	Silent plaque → heart attack
Rhythm	ECG; symptoms of palpitation/syncope	Arrhythmia & sudden-death risk
Vessels	Blood pressure; vascular assessment	Stiffness & endothelial dysfunction
Over time	Repeat imaging; trend	Some change reverses, some persists

99.10 Common myths

COMMON MYTH

"My heart's fine — I do loads of cardio and feel great."

Fitness and feeling well do not rule out steroid-associated cardiac change, which is typically silent and detected only by imaging; a big heart from training is not the same as a healthy one.[1,6]

● Research Confidence: Strong

COMMON MYTH

"Any heart changes just go back to normal when you stop."

Some change is partly reversible after stopping, but not all of it — longer-term data show structural change can persist, and established plaque or cardiomyopathy does not simply resolve.[4,5]

Research Confidence: Moderate–Strong

99.11 Frequently Asked Questions

If I feel fine, is my heart okay? Not necessarily — the most important cardiac changes are silent and only show on ECG and echocardiography.[1,3]

Isn't a bigger heart just from training? Training does enlarge the heart benignly, but that adaptation can resemble harmful change; telling them apart needs clinical assessment.[6]

Will my heart recover if I stop? It may improve, sometimes substantially, but some change can persist — which is why earlier is better.[4,5]

99.12 Case Study

Case Study 99.1

A long-term steroid user, fit and symptom-free, has an echocardiogram that shows a thickened left ventricle. He assumes it is simply "athlete's heart" from years of training. But strain imaging shows his heart's pumping is subtly reduced, and his ECG shows a strain pattern.

> Feels well · LV wall thickened · Strain imaging: reduced function · ECG: strain pattern

How a clinician reasons through this. The thickened ventricle alone is ambiguous — it could be benign athletic remodelling. What tips the assessment is the whole picture: genuine athlete's heart preserves or enhances function, whereas here the strain imaging shows function is actually *reduced*, and the ECG adds a strain pattern. Together these point away from a purely physiological adaptation and toward pathological change — despite the person feeling completely well. This is exactly why symptoms are not the test and imaging is: the tools separated a benign possibility from a concerning reality. The clinician's next steps are proper cardiac evaluation and an honest conversation about the heart being the highest-stakes reason to reconsider exposure.

99.13 Decision tree

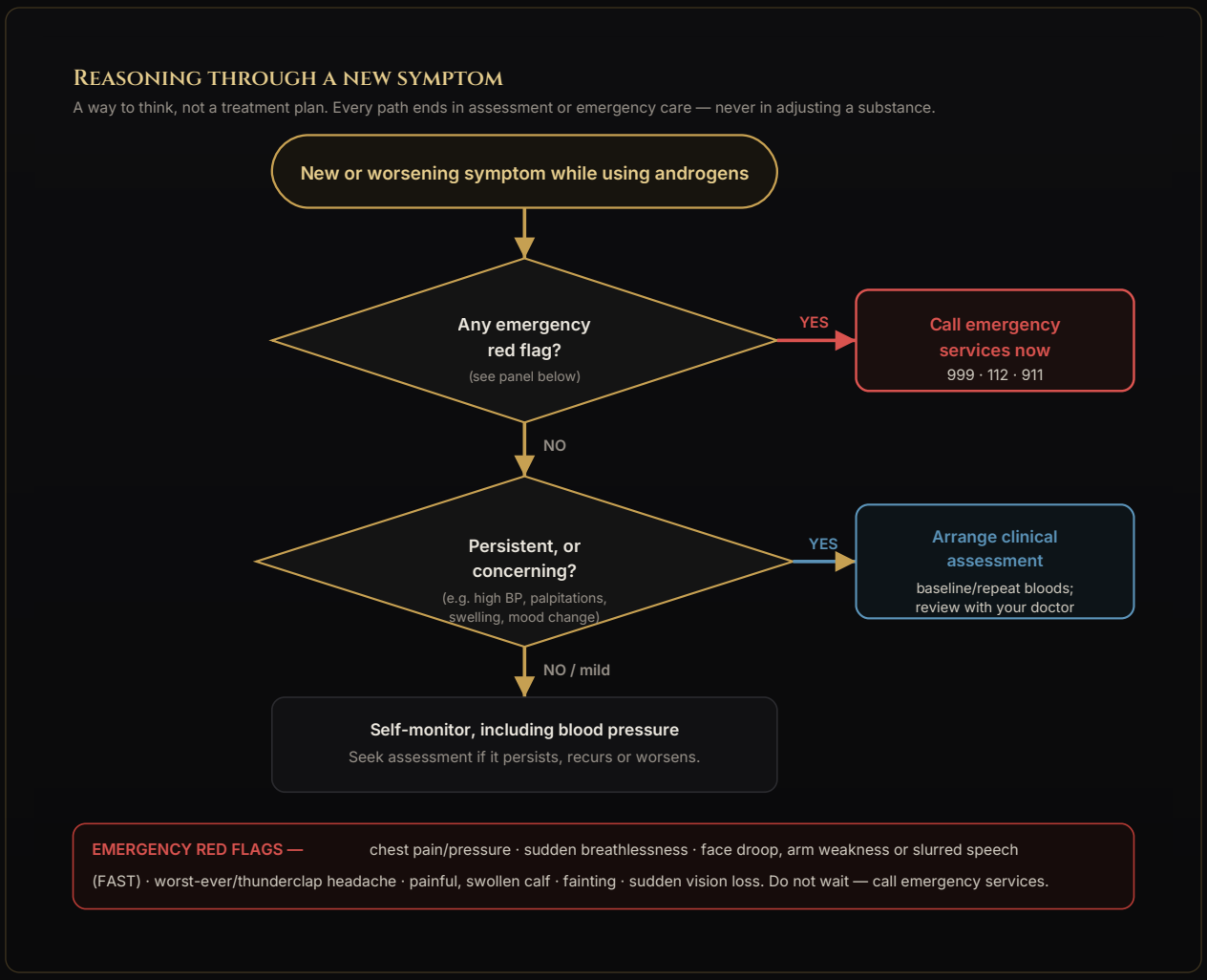

Figure 99.2 — Reasoning through a cardiovascular concern. Cardiac emergencies need help immediately; abnormal findings need prompt specialist assessment; ambiguous ones are interpreted and tracked.

99.14 Key Takeaways

KEY TAKEAWAYS

1. The heart carries the **highest, least-reversible stakes** — and its harm is usually **silent**.[1,2]
2. Steroid use is associated with a **thicker, less efficient left ventricle**, detectable early by strain imaging.[2,3,4]
3. Benign **"athlete's heart"** can resemble pathological change — telling them apart is a clinical judgement.[6]
4. **Coronary plaque** builds silently and connects directly to the lipid changes of Chapter 98.[1,7]
5. The worst outcomes — **arrhythmia, cardiomyopathy, sudden death** — are why the silent changes are taken seriously.[8,9]
6. The **vessels** stiffen and their lining is impaired — monitored alongside the heart.[10,11]
7. Detection is by **ECG and echocardiography**, interpreted by a clinician — not by how you feel.[3,6]
8. Change may **partly reverse** after stopping, but some can persist — earlier recognition is better.[4,5]

Medical disclaimer. This chapter is educational and not a substitute for assessment by a qualified clinician. It does not recommend or encourage use and contains no dosing or usage instructions. Interpreting cardiac tests, and deciding what to do about them, is a matter for a qualified professional; in an emergency, contact your local emergency services immediately.

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PART IX · THE MYTH LIBRARY

Overview

HOW TO READ A CLAIM

"A myth and a fact can sound exactly the same. The difference is not how confident the speaker is — it is what stands behind the words."

— ON WHY THIS PART EXISTS

IX.1 Why a whole part on myths

Almost no field runs on folklore the way this one does. Because so much of the knowledge is passed hand to hand — forum to gym floor to training partner — claims travel faster than evidence, and repetition does the work that proof should. Some of those claims are harmless. Others get people hurt: a belief that a drug is safe delays the check-up that would have caught a problem, a belief that a symptom is "just part of it" talks someone out of seeking help, and a belief about what protects you offers a **false safety the body does not honour**. This part exists to hold the most common and most consequential of these beliefs up to the light. It is not written to shame anyone — most myths are repeated in good faith by people who mean well — but to replace inherited certainty with something you can actually check.

> ☆ **PHALANX INSIGHT**
>
> The most valuable thing this part can give you is not a list of debunked claims to memorise — new myths appear faster than any book can catch them — but a **method**. If you can look at any claim, old or new, and ask where it comes from, what evidence stands behind it, who benefits if you believe it, and what would change your mind, you can evaluate the next myth yourself. The debunked list is the fish; the method is the fishing. Everything that follows in this part is really an extended worked example of that one transferable skill: reading a claim before you act on it.

IX.2 What a myth actually costs

It is tempting to treat a false belief as harmless until acted upon, but in this field the cost is direct and often physical. Myths do their damage in three recognisable ways. They cause **bad decisions** — someone chooses a drug, or a combination, on the strength of a claim that was never true. They create **false safety** — a reassuring myth ("this one's harmless", "you can always undo it later") removes the caution that would have kept a person monitoring their health. And they cause **delayed help** — a myth that reframes a warning sign as normal ("everyone's blood pressure runs high", "feeling low coming off just means you need more") keeps someone from a doctor at exactly the moment attention matters most. A myth, in other words, is not merely incorrect; it is expensive, and the bill is usually paid in health.

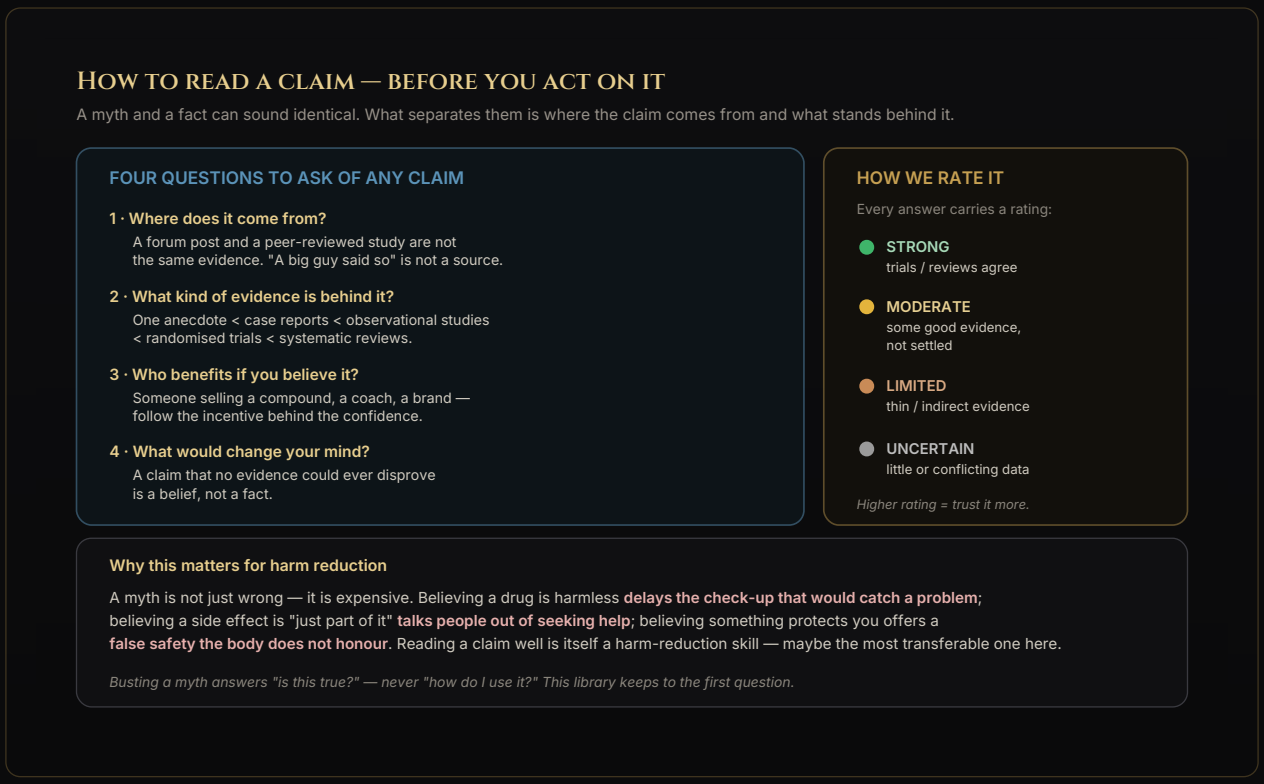

Figure IX.1 — The method this part teaches: four questions to ask of any claim, and the Research Confidence scale used to rate every answer.

IX.3 Four questions to ask of any claim

Reading a claim well comes down to four questions, and they work on anything — a forum post, a coach's advice, or a sentence in this very book. **First, where does it come from?** A peer-reviewed study and an anonymous forum post are not equivalent evidence, and "a big, successful guy said so" is not a source — impressive physiques are not proof of accurate physiology. **Second, what kind of evidence stands behind it?** There is a ladder here: a single anecdote sits below collected case reports, which sit below observational studies, which sit below randomised controlled trials, which sit below systematic reviews that pool many studies. **Third, who benefits if you believe it?** When the person making a claim also sells the compound, coaches the client, or profits from the brand, the confidence deserves extra scrutiny. **Fourth, what would change your mind?** A claim that no possible evidence could ever disprove is a belief, not a fact — and beliefs dressed as facts are exactly where myths live.

IX.4 How the Research Confidence ratings work

Throughout this book, evidence-based claims carry a small **Research Confidence** badge, and it is worth understanding what it means. A rating of

● STRONG

signals that well-conducted trials or systematic reviews broadly agree — you can lean on it.

● MODERATE
means there is some good evidence but the matter is not fully settled, so hold it a little more loosely.

● LIMITED
flags that the evidence is thin, indirect, or drawn from small or lower-quality studies. And

● UNCERTAIN
means the data are sparse or conflicting and honest people still disagree. The ratings are not a gimmick; they are an attempt to be transparent about how much weight any given statement can bear — including, importantly, admitting when the honest answer is "we do not really know."

IX.5 A NOTE ON HOW THESE MYTHS ARE ANSWERED

Every entry in this part follows the same shape: the **claim** stated plainly, as it is usually heard; an **answer** grounded in cited evidence; and a **Research Confidence** rating. Two principles govern that answer. The first is **fairness** — where a myth contains a grain of truth, this book says so rather than pretending the claim is pure nonsense, because a half-true myth is only dismantled by acknowledging the half that holds. The second is the book's unbroken rule: **busting a myth is not the same as teaching how to use a drug**. Correcting "this compound is harmless" with the evidence of its risks is education; it never tips into dosing, protocols or instructions. This part answers the question "is this true?" — never "how do I do it?"

IX.6 A PREVIEW OF THE CLUSTERS AHEAD

The myths in this part are grouped by theme so related beliefs can be answered together. **Safety and "natural" myths** tackle the reassurances — that certain drugs are harmless, that a substance is safe because it is "natural" or sold as a supplement, that harms are always reversible. **Steroid myths** address the beliefs specific to anabolic-androgenic steroids. **Ancillary and recovery myths** cover the drugs taken alongside — the "everything can be restored afterwards" claims. **Training and nutrition myths** address the foundational beliefs from Part VIII. **Health-monitoring myths** take on the ideas that keep people from bloodwork and check-ups. And **harm-reduction myths** confront the false shortcuts — the belief that some ritual makes a genuinely risky practice safe. To show the format at work, the rest of this overview busts a handful of the most important cross-cutting myths now.

IX.7 Five cross-cutting myths

COMMON MYTH · SAFETY

"If you're sensible about it, performance-enhancing drugs are basically safe."

An Endocrine Society scientific statement documents a broad range of adverse health consequences of performance-enhancing drugs across multiple organ systems — "sensible use" reduces some risks but does not make these drugs safe, and several harms are serious or irreversible.[1] The honest framing is risk reduction, never safety.

Research Confidence: Strong

COMMON MYTH · THE HEART

"Steroids don't really hurt your heart if you're young and train hard."

A controlled study found long-term anabolic-steroid use associated with left-ventricular dysfunction and coronary atherosclerosis in otherwise healthy, trained men — the cardiovascular toxicity is real and often silent, not cancelled by youth or fitness.[2]

Research Confidence: Strong

COMMON MYTH · DEPENDENCE

"You can just stop anytime — steroids aren't addictive."

Anabolic-androgenic steroid dependence is a recognised disorder: a meaningful minority of long-term users meet dependence criteria, driven by the body-image drive and a withdrawal syndrome that punishes stopping.[3] "Just stopping" is genuinely hard for many, and that difficulty is physiological, not a failure of willpower.

Research Confidence: Strong

COMMON MYTH · "JUST A FAT-BURNER"

"Fat-burners are mild — they're just strong supplements."

Some are genuinely dangerous drugs. 2,4-dinitrophenol (DNP) is a weight-loss agent with significant acute toxicity and a real risk of death, with no antidote — nothing about being sold for fat loss makes it a mere supplement.[4] Category ("fat-burner") tells you nothing about safety.

Research Confidence: Strong

COMMON MYTH · ORAL STEROIDS

"Oral steroids are fine short-term — pills are gentler than injections."

Being a pill is not being gentle. Oral 17α-alkylated steroids can cause serious liver injury; methasterone (superdrol), for example, is documented to cause cholestatic liver injury — the oral route adds a hepatic hazard the injectable form does not carry.[5]

Research Confidence: Strong

IX.8 How to use this part

You can read the Myth Library straight through, or dip into the cluster that matches a claim you have just heard. Either way, the intended takeaway is the same: not a memorised catalogue of right answers, but a habit of mind. When the next confident claim reaches you — and in this field it always will — run it through the four questions, notice who benefits, look for the evidence behind it, and check what rating an honest source would give. That habit will protect you long after any specific myth in these pages has been forgotten or replaced. The single most useful sentence in this part is not any one debunking; it is the reminder that **how sure someone sounds is not evidence of anything at all**.

IX.9 Key takeaways

KEY TAKEAWAYS

1. Myths cost real health — through **bad decisions, false safety and delayed help** — so reading claims well is a harm-reduction skill.
2. Ask four questions of any claim: **where it comes from, what evidence backs it, who benefits, and what would change your mind**.
3. Evidence has a **ladder** — anecdote below case reports below observational studies below trials below systematic reviews.
4. **Research Confidence** ratings show how much weight a claim can bear, including admitting when the answer is "uncertain".
5. Myth-busting answers **"is this true?"** — never "how do I use it?"; correcting a false safety claim is education, not instruction.[1]
6. The transferable lesson: **how confident someone sounds is not evidence** — the method outlasts any single debunked myth.

Medical disclaimer. This part is educational and evaluates common claims against published evidence. It is not medical advice and does not instruct on the use of any substance. Where a myth concerns your health, monitoring, or a decision about a drug, discuss it with a qualified clinician rather than relying on any claim — including a debunking — in isolation.

IX.10 References

1. Pope HG Jr, Wood RI, Rogol A, Nyberg F, Bowers L, Bhasin S. Adverse health consequences of performance-enhancing drugs: an Endocrine Society scientific statement. *Endocr Rev.* 2014;35(3):341–375. doi:10.1210/er.2013-1058. PMID:24423981.
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3. Kanayama G, Brower KJ, Wood RI, Hudson JI, Pope HG Jr. Anabolic-androgenic steroid dependence: an emerging disorder. *Addiction.* 2009;104(12):1966–1978. doi:10.1111/j.1360-0443.2009.02734.x. PMID:19922565.
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5. Shah NL, Zacharias I, Khettry U, Afdhal N, Gordon FD. Methasteron-associated cholestatic liver injury: clinicopathologic findings in 5 cases. *Clin Gastroenterol Hepatol.* 2008;6(2):255–258. doi:10.1016/j.cgh.2007.11.010. PMID:18187367.

CONTINUE READING

You have read a fraction of what the full manual contains. The complete reference runs to 626 pages across ten Parts — foundations and physiology, a full Bloodwork Academy, detailed chapters on every major compound class, health monitoring, injection medicine, medical emergencies, lifestyle, and a comprehensive Myth Library, glossary and index.

Every chapter carries real, verified references and a visible research-confidence rating. It is education and harm reduction throughout — never a guide to use.

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