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General Anaesthesia in Athletes: Physiological Adaptations, Pharmacological Nuances, and Clinical Management

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Abstract

Background. Years of structured, high-intensity conditioning leave a mark on far more than skeletal muscle. The heart of a trained competitor remodels, autonomic balance drifts towards vagal predominance, resting metabolic demand climbs, and body composition shifts decisively towards lean tissue. Taken together, these changes describe a patient who differs in several respects at once from the average person presenting for surgery, and each of them has at least a plausible bearing on the response to general anaesthesia.

Aim. To appraise and synthesise the evidence available on the perioperative care of athletes receiving general anaesthesia. Particular attention is given to haemodynamic and autonomic adaptation, the influence of body composition on drug disposition, preoperative screening, airway assessment and the Mallampati classification in athletic populations, the behaviour of induction and maintenance, the choice between intravenous and inhalational technique, the toxicology of performance-enhancing agents, and compliance with anti-doping regulation.

Material and methods. A narrative review of the scientific literature was carried out, drawing on anaesthesiology, cardiology and sports medicine sources.

Results. Dynamic endurance training produces eccentric hypertrophy with balanced four-chamber dilation and pronounced resting vagal tone, whereas static resistance work drives a concentric pattern. That high vagal tone plausibly underlies the susceptibility of highly trained endurance athletes to clinically significant bradyarrhythmias under anaesthesia, and several published case reports describe precisely this sequence of events. Lean body weight is a reasonable scalar to consider for hypnotic induction agents in very muscular patients, although the supporting evidence originates in obese rather than athletic cohorts and direct validation in athletes is still lacking. Measures of intrinsic muscular strength track with higher Modified Mallampati scores. Undisclosed use of performance-enhancing drugs adds a further layer of risk, including accelerated coronary disease, myocardial fibrosis and hypercoagulability, while the World Anti-Doping Agency Code imposes obligations of its own on the prescribing anaesthesiologist.

Conclusions. General anaesthesia in the athlete calls for an individualised, evidence-informed approach: physiological adaptation must be recognised for what it is, cardiovascular and airway assessment must be thorough, drug dosing must be cautious, and the team must be ready for a bradyarrhythmia that may appear without warning.

Keywords: general anaesthesia, athlete’s heart, sports anaesthesia, lean body weight, propofol, Mallampati score, bradyarrhythmias, anabolic steroids, WADA

Introduction

Professional, collegiate and serious recreational sport has expanded considerably over the past two decades, and with it the number of highly trained people who eventually need an operation [1; 3]. Prolonged athletic conditioning reshapes anatomy, metabolism and neurovegetative control in ways that serve one purpose, namely to optimise oxygen delivery, peripheral perfusion and musculoskeletal power output [8; 10]. Those same adaptations, however, do not simply disappear when the patient arrives in the anaesthetic room. They interact with the cardiovascular and pharmacological effects of general anaesthesia, occasionally in ways that are clinically inconvenient [1; 3; 7; 9].

For a long time the athlete presenting for surgery was treated as a straightforward case: young, fit, healthy, low risk [9]. The assumption is understandable but incomplete, because it passes over the electrophysiological, morphological and autonomic remodelling that defines this population [1; 7; 10].

High resting vagal tone, enlarged cardiac chambers, an altered autonomic set point and electrocardiographic variants that would be pathological in a sedentary patient all complicate the interpretation of preoperative findings. In a small number of published cases they have also been implicated in clinically significant bradyarrhythmias during general or neuraxial anaesthesia [2; 7; 8; 10]. Body composition adds a second dimension. Increased lean body mass, expanded intravascular volume, generous regional organ blood flow and very little adipose tissue may plausibly influence the volume of distribution, the clearance and the overall pharmacokinetic profile of both intravenous and inhalational agents [1; 6].

A third consideration is pharmacological rather than physiological. Covert use of performance-enhancing drugs (PEDs), including anabolic-androgenic steroids, growth factors, beta-2 agonists, central stimulants and masking agents, introduces drug interactions and multi-organ pathology that the anaesthetist may never be told about [1; 9; 11]. Substance misuse in this population has been linked to premature coronary atherosclerosis, myocardial fibrosis, biventricular dysfunction, hepatic impairment, hypercoagulability and altered airway anatomy [4; 9; 11]. At the same time, anyone caring for a competitive athlete has to work within the World Anti-Doping Agency (WADA) Code, since a perioperative prescription written without thought can produce an anti-doping rule violation [1; 3; 9].

This review sets out to draw together the current evidence on general anaesthesia in athletes. Nine areas are addressed: physiological and haemodynamic adaptation of the athletic

cardiovascular system; body composition and its pharmacokinetic consequences; preoperative assessment and the separation of occult pathology from training effect; the Modified Mallampati classification and anthropometric predictors of a difficult airway; induction phenomena, heart rate variability and autonomic shifts; intraoperative maintenance, drug handling and the choice between total intravenous anaesthesia and volatile agents; performance-enhancing drug toxicity; postoperative pain perception together with regional and local blockade; and finally anti-doping compliance and shared decision-making.

Materials and Methods

A narrative review was undertaken of 14 published scientific works, comprising randomised clinical trials, prospective cohort investigations, physiological studies and specialised clinical reports, supplemented by three further review articles addressing related physiological and anaesthetic questions. The literature examined spans the years 1989 to 2026 and is drawn from international anaesthesiology, cardiology and sports medicine journals.

Data and clinical observations were extracted and then grouped into thematic sections: cardiovascular remodelling, autonomic dynamics, upper airway morphology, pharmacokinetics and dosing scalars, induction and maintenance of anaesthesia, the toxicology of performance-enhancing agents, postoperative recovery, and regulatory compliance. Because the review is narrative rather than systematic, no formal quality scoring or meta-analytic pooling was attempted, and the synthesis presented below should be read with that limitation in mind.

Results

1. Physiological and haemodynamic adaptation in athletes

Repeated haemodynamic loading during training produces a set of morphophysiological changes in the cardiovascular system known collectively as exercise-induced cardiac remodelling (EICR), or more colloquially as the athlete’s heart [1; 8; 10]. Which pattern develops depends largely on the balance of dynamic (isotonic) and static (isometric) work that a given sport demands [8; 9].

Dynamic endurance disciplines such as distance running, road cycling, rowing and cross-country skiing impose sustained volume overload [8; 9; 10]. Cardiac output is high, left ventricular stroke volume rises substantially, and systemic vascular resistance is normal or reduced [8; 9]. The heart answers with eccentric hypertrophy: balanced dilation of all four

chambers together with proportional thickening of the walls [8; 9; 10]. In elite endurance competitors the left ventricular end-diastolic diameter often exceeds 55 to 60 mm [8; 9; 10]. What is striking is that despite this degree of enlargement, resting systolic function remains normal or low-normal while diastolic filling is supranormal, a consequence of enhanced myocardial compliance and elastic recoil [8; 9; 10].

Static resistance sports, among them Olympic weightlifting, throwing events and bodybuilding, load the heart in a different way. Sustained muscular contraction and repeated Valsalva manoeuvres raise both systolic and diastolic pressure and generate transient surges in systemic vascular resistance [8; 9]. The resulting stimulus favours concentric remodelling, meaning thicker ventricular walls without meaningful cavity dilation [8; 9; 10]. Wall thickness of 13 to 14 mm is not unusual in strength athletes [8; 10]. It is worth noting that the blood pressure response itself varies with the type of activity undertaken, which is one reason why training history matters during preoperative assessment [16].

Mixed disciplines occupy the middle ground. Soccer, rugby, basketball and ice hockey combine dynamic and static demands, and the phenotype that follows is correspondingly intermediate, with eccentric chamber enlargement accompanied by mild concentric thickening [8; 9]. Across all of these groups, resting stroke volume is markedly elevated, which allows a normal resting cardiac output of 5 to 6 L/min to be sustained at heart rates as low as 30 to 45 beats per minute [1; 7; 8; 10]. Plasma volume is expanded, systemic capillary density is greater, and skeletal muscle oxidative capacity is enhanced [1; 8; 10].

2. Body composition and the pharmacokinetics of anaesthetic drugs

The competitive athlete's body composition departs from that of sedentary and general surgical populations in a fairly predictable way [1; 6]. Lean body weight (LBW) accounts for a high proportion of total mass, skeletal muscle is expanded, total body water is increased, blood and plasma volumes are enlarged, and the percentage of body fat is low [1; 6; 9]. Since body mass index (BMI) relates total weight to height squared and takes no account of what that weight consists of, muscular athletes are routinely classified as overweight or even obese despite carrying almost no adipose tissue [1; 6]. Any dosing decision anchored to BMI alone therefore starts from a misleading premise.

These characteristics alter how anaesthetic drugs distribute [1; 6]. Lean tissues, that is skeletal muscle, brain, liver, kidneys and heart, receive roughly 22% of resting cardiac output, whereas poorly vascularised adipose tissue takes only about 5% [1; 6]. Where lean mass is high, early

distribution into vessel-rich tissue is accelerated and the apparent volume of distribution for water-soluble and moderately lipophilic drugs is larger [1; 6]. Training-related changes in cardiac output, organ perfusion and renal and hepatic physiology can be expected to influence distribution and clearance as well, although the precise magnitude of that influence for individual anaesthetic agents in highly trained subjects has not been established [1; 6].

This brings us to the practical question of which weight to dose against. The case for scaling propofol to lean body weight rests primarily on studies in patients with obesity, and its relevance to the muscular athlete is inferential rather than demonstrated. In the randomised trial reported by Ingrande and colleagues [6], LBW proved an appropriate scalar for propofol induction in morbidly obese subjects, whereas dosing to total body weight (TBW) produced larger administered doses, higher peak plasma concentrations and more post-induction hypotension. Extrapolating from an obese cohort to a lean and highly muscular one is not a trivial step, and although LBW-based dosing seems a sensible starting position in the athlete, its validity in this population remains an open question that deserves direct study [1; 6].

Where bioimpedance analysis is not available, LBW can be estimated with the Janmahasatian formula [1; 6]:

- Males: $\text{LBW (kg)} = (9.27 \times 10^3 \times \text{TBW}) / (6.68 \times 10^3 + 216 \times \text{BMI})$
- Females: $\text{LBW (kg)} = (9.27 \times 10^3 \times \text{TBW}) / (8.78 \times 10^3 + 244 \times \text{BMI})$

Neuromuscular blocking agents follow different rules. Succinylcholine distributes into extracellular water and is hydrolysed by pseudocholinesterase, so its dose should be calculated on total body weight (1.0 mg/kg TBW) if a complete block is to be obtained [1; 6]. For non-depolarising agents such as rocuronium and vecuronium, dosing is best individualised according to patient characteristics and the pharmacology of the particular drug. Evidence on altered requirements in muscular athletes is thin, and for that reason objective quantitative neuromuscular monitoring should guide both administration and the confirmation of adequate recovery rather than any assumed dose per kilogram [1; 9].

Table 1. Physiological adaptations in athletes and their potential implications for general anaesthesia.

Parameter	**Athletic adaptation**	**Potential implications for general anaesthesia**
Resting heart rate	Decreased	Greater baseline bradycardia; interpretation should take athletic conditioning into account
Stroke volume	Increased	Cardiac output may be preserved despite a low heart rate
Vagal tone	Increased	Possible greater susceptibility to autonomic disturbance at induction
Plasma volume	Increased	Potential influence on drug distribution
Lean body mass	Increased	May affect volume of distribution and weight-based dosing
Body fat	Decreased	Potentially altered distribution of lipophilic drugs
Cardiac output during exercise	Markedly increased	Greater cardiovascular reserve
Airway soft tissue and neck musculature	Increased	Potentially more difficult airway management

Parameter	Athletic adaptation	Potential implications for general anaesthesia
Oxygen consumption	Increased	Faster desaturation during apnoea

3. Preoperative assessment and clinical screening

Preoperative evaluation of an athlete demands a working knowledge of training-related physiology, and for two reasons that pull in opposite directions: normal athletic variants must not be mistaken for disease, and genuine occult pathology must not be dismissed as a training effect [1; 3; 8; 9; 10].

Resting electrophysiological changes are found in up to 80% of competitive athletes [9; 10]. Elevated parasympathetic tone, reduced resting sympathetic drive and intrinsic remodelling of the sinoatrial node together explain most of what appears on the trace [1; 2; 7; 10]. A resting 12-lead electrocardiogram may show sinus bradycardia of 30 to 50 bpm, sinus arrhythmia, junctional escape rhythms, first-degree atrioventricular block with a PR interval above 200 ms, or Mobitz type I second-degree block [8; 9; 10]. Morphological variants are equally common: incomplete right bundle branch block appears in 35% to 50% of endurance athletes as a consequence of right ventricular dilation, and isolated high QRS voltage satisfying Sokolow-Lyon criteria for left ventricular hypertrophy (S wave in V1 plus R wave in V5 greater than 35 mm) is a frequent incidental finding [2; 7; 8; 9; 10]. Early repolarisation, with concave ST elevation in V2 to V4 in white athletes or domed ST elevation followed by T-wave inversion in V1 to V4 in Black athletes, is likewise physiological [2; 7; 8; 9; 10].

The difficulty lies in telling benign remodelling apart from hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy (ARVC) and the inherited channelopathies [8; 9; 10]. An athlete whose left ventricular wall thickness falls into the diagnostic grey zone of 13 to 15 mm requires full evaluation before anyone concludes that all is well [8; 10].

Several findings favour physiological adaptation: symmetric hypertrophy, balanced four-chamber dilation, supranormal early diastolic filling with an E/A ratio above 2, preserved global longitudinal strain better than minus 18%, absence of late gadolinium enhancement on cardiac magnetic resonance imaging, and normalisation of repolarisation abnormalities during

exercise testing [8; 9; 10]. The opposite pattern, that is asymmetric septal hypertrophy, left atrial enlargement disproportionate to ventricular size, impaired diastolic relaxation, reduced strain and mid-wall fibrosis, points towards cardiomyopathy [8; 9; 10].

History taking should be directed at exertional syncope or presyncope, chest pain, palpitations, and any unexplained fall in performance, together with a family history of premature sudden cardiac death [1; 7; 8; 9; 10]. Laboratory work covering haematocrit, electrolytes, renal and hepatic function and glucose helps to identify occult dehydration, electrolyte disturbance or organ dysfunction related either to heavy training or to substance use [1; 9]. Two further conditions deserve specific enquiry, namely exercise-induced bronchospasm and obstructive sleep apnoea, both of which are over-represented in high-ventilation sports and among collision athletes with large neck circumferences [1; 9].

4. Upper airway assessment and the Mallampati classification in athletes

Airway management in this population carries anatomical challenges of its own [1; 4; 9]. Competitors in strength, power and contact sports, rugby, American football and combat disciplines among them, frequently present with hypertrophied neck musculature, bulky deltoids and limited cervical extension [1; 4; 9]. Add pharyngeal soft-tissue enlargement, a short thyromental distance and macroglossia, the last of which may be aggravated by growth hormone misuse, and mask seal, bag-mask ventilation and direct laryngoscopic view can all be compromised [1; 9].

The Modified Mallampati Score (MMS) remains the standard bedside instrument for grading the visibility of oropharyngeal structures and estimating the likelihood of a difficult intubation [4]. Erbesler and colleagues [4] examined the relationship between muscular strength, hand anthropometry and Mallampati class in elite track athletes and non-athletic controls, and reported that particular strength and dimensional measures were associated with the higher classes III and IV [4].

Their data show a significant correlation between pinch strength of the thumb (PST) and Mallampati class ($r = 0.392$, $p = 0.001$) [4]. A multilayer perceptron neural network model identified pinch strength of the little finger (PSLF) as the leading predictor in women, with 100% normalised importance, fingertip to root digit 3 (FTR3) as the dominant predictor in men, again at 100%, and hand breadth at thumb (HBT) as the most important variable across the sample as a whole [4]. Hand breadth at thumb, fist circumference, wrist circumference and the finger pinch strengths were all significantly greater in elite athletes than in controls [4]. Global

handgrip strength (HGS), by contrast, was not an independent predictor once sex and athletic status had been controlled for, a result the authors themselves describe as running against expectation [4]. Wrist extension angle (WEA) was significantly reduced in those with MMS III and IV, which the authors attribute to differences in joint flexibility and soft-tissue structure [4].

The practical message is that airway assessment in muscular and elite athletes should combine the conventional predictors with an appreciation of the patient's build [1; 4; 9]. Where a large neck circumference, high pinch strength and an elevated Mallampati score coincide, difficult mask ventilation and a restricted direct laryngoscopic view should be anticipated rather than discovered, and ramped positioning aids, a video laryngoscope and advanced supraglottic rescue devices should be to hand before induction begins [1; 4; 9].

5. Induction of general anaesthesia: autonomic shifts and haemodynamic behaviour

Induction is the point of greatest vulnerability for the athletic patient, chiefly because it dismantles a resting autonomic balance that has taken years to establish [1; 2; 7]. Endurance athletes maintain resting cardiac output through a large stroke volume paired with a slow heart rate, an arrangement sustained by strong vagal efferent activity and low sympathetic outflow [1; 7; 10]. Intravenous hypnotics and opioids produce central sympatholysis; baseline sympathetic tone is withdrawn while vagal tone is left unopposed [1; 2; 7; 12].

The consequence may be pronounced bradycardia and, occasionally, nodal escape rhythms or cardiac arrest [1; 2]. Published reports describe well-trained endurance athletes, marathon runners and triathletes among them, who developed sudden and profound sinus arrest or asystole during routine induction or spinal anaesthesia in the absence of hypoxia, hypercapnia or surgical stimulation [2; 7]. Dominici and co-workers [2] described a high-level triathlete with a resting rate of 40 bpm in whom persistent bradyarrhythmia during propofol and alfentanil induction required 1.25 to 1.5 mg of intravenous atropine before a stable rate could be restored and held. Kreutz and Mazuzan [7] reported asystole in a marathon runner under spinal anaesthesia; upper thoracic sympatholysis unmasked the underlying vagal predominance and precipitated an arrest that needed closed-chest compressions and repeated doses of atropine.

Power spectral analysis of heart rate variability (HRV) offers a quantitative window on these effects [12; 14]. The technique separates fluctuations in heart rate into a low-frequency component (LF, 0.04 to 0.15 Hz), which reflects combined sympathetic and parasympathetic

regulation, and a high-frequency component (HF, 0.15 to 0.40 Hz), which is taken to represent cardiac vagal modulation alone [12; 14]. Their ratio is an established, if imperfect, index of sympathovagal balance [12; 14].

Riznyk and colleagues [12] compared thiopental with propofol during fentanyl-based induction in 100 patients. Fentanyl reduced total spectral power and LF power while leaving HF power intact, a pattern consistent with preferential suppression of cardiac sympathetic tone [12]. Propofol given afterwards lowered total power and HF power further but preserved LF power, which suggests that it reduces cardiac parasympathetic activity more than sympathetic activity [12]. Thiopental behaved differently again: normalised LF power rose, HF power fell, the LF/HF ratio increased, and the overall picture was one of a vagolytic effect accompanied by an increase in sympathetic activity [12].

Win and colleagues [14] examined intravenous propofol and midazolam sedation using high-resolution maximum entropy HRV analysis together with spectral entropy. Propofol sedation reduced total power and the LF/HF ratio and increased normalised HF power during recovery, indicating parasympathetic dominance with depressed sympathetic modulation [14]. Midazolam produced the reverse, with reduced normalised HF power and a rising LF/HF ratio during recovery, that is persistent sympathetic dominance [14].

Applied to a patient whose baseline vagal tone is already high, this evidence suggests that a fentanyl and propofol induction may deepen parasympathetic dominance and blunt baroreflex compensation at the same time [1; 2; 12; 14]. Preparation therefore matters more than usual: continuous electrocardiographic and arterial pressure monitoring, and atropine, glycopyrrolate and ephedrine drawn up and immediately available rather than in a drawer [1; 2; 7; 9].

6. Intraoperative maintenance, drug handling and choice of technique

Once anaesthesia is established, the athlete's physiology continues to shape ventilation, drug disposition, temperature control and electrophysiological stability [1; 9; 13].

Baseline oxygen consumption and carbon dioxide production are both raised in muscular subjects [1; 9]. After induction and neuromuscular blockade, thoracic wall compliance may be reduced by bulky pectoral and intercostal musculature, so higher inflating pressures and an adjusted minute volume are often needed to hold normocapnia and adequate oxygenation [1; 9]. Should apnoea occur, the metabolic appetite of a large muscle mass shortens the time to

desaturation appreciably, and the margin for a leisurely intubation is correspondingly narrower [1; 9].

Drug handling during maintenance reflects high cardiac output and expanded lean tissue [1; 6]. Flow-limited agents are cleared rapidly by the liver and redistributed quickly into tissue [1; 6]. Muscle relaxant requirements may also differ: apparent resistance to non-depolarising agents has been attributed to upregulation of acetylcholine receptors in trained muscle, which is another argument for quantitative train-of-four monitoring rather than dosing by habit, both to secure surgical relaxation and to exclude residual paralysis at the end of the case [1; 9].

The choice between total intravenous anaesthesia (TIVA) with propofol and a volatile technique using sevoflurane, desflurane or isoflurane involves a genuine trade-off, and comparative data on haemodynamic variability between the two approaches continue to accumulate [1; 9; 13; 17].

TIVA with propofol offers stable maintenance, leaves baseline cardiac conduction largely undisturbed and does not prolong the QT interval [9; 13]. That is a real advantage in an athlete with repolarisation variants or resting QT prolongation, where any additional proarrhythmic burden is unwelcome [9; 13]. Propofol also has intrinsic antiemetic properties, reduces postoperative nausea and vomiting, and tends to produce a smooth emergence, which is no small consideration in a large and powerfully built patient [1; 3; 9; 13]. Against this, propofol uncouples complexes I and II of the mitochondrial electron transport chain. Propofol infusion syndrome (PRIS) is rare, but prolonged high-dose infusion is best avoided in the critically ill or metabolically depleted athlete [9; 13].

Volatile agents, sevoflurane in particular, prolong the QTc interval in a concentration-dependent manner [9; 13]. In a patient with resting bradycardia, left ventricular hypertrophy or pre-existing repolarisation abnormality, that prolongation may raise the risk of torsades de pointes and other ventricular tachyarrhythmias [9; 13]. Volatiles nevertheless cause less peripheral vasodilation than a propofol infusion [13]. This matters in one specific and uncommon scenario: the athlete who comes to theatre soon after strenuous exercise, in whom persistent exercise-induced muscle vasodilation can produce marked post-exercise hypotension [1; 13]. Here a volatile technique may preserve systemic vascular resistance more effectively than TIVA [13].

Thermoregulation is also disturbed, most obviously in the immediate post-exercise state [1; 9; 13]. Hard exertion displaces central thermoregulatory set-points and promotes cutaneous heat

loss [1; 9; 13]. Superimpose anaesthetic inhibition of vasoconstriction and core hypothermia follows readily, with knock-on effects on coagulation and drug metabolism and a tendency to shiver on emergence [1; 9; 13]. Continuous core temperature monitoring and active warming are not optional in this group [1; 9].

7. Performance-enhancing drugs: toxicity and anaesthetic hazard

Covert use of performance-enhancing drugs is among the more difficult problems in sports anaesthesia, partly because the history is so often incomplete [1; 3; 9; 11]. Nor is the problem confined to elite sport; survey data point to substantial misuse among amateur competitors, bodybuilders, recreational gym users and fitness enthusiasts [1; 9; 11]. Several pharmacological classes are involved, each with its own perioperative signature [1; 9; 11].

Anabolic-androgenic steroids (AAS), including testosterone esters, nandrolone, stanozolol, oxandrolone and methandrostenolone, are taken to promote muscle hypertrophy and strength [1; 9; 11]. Perry and colleagues [11] reviewed the cardiovascular consequences of steroid misuse and grouped the mechanisms of harm under four headings: atherogenic, thrombotic, vasospastic and direct myocardial injury [11]. Dyslipidaemia is characteristic, with suppression of high-density lipoprotein alongside a rise in low-density lipoprotein, and the net effect is accelerated coronary atherosclerosis [11]. A procoagulant state develops in parallel through increased synthesis of clotting factors, enhanced platelet aggregation and elevated thromboxane A2, raising the risk of acute myocardial infarction, ischaemic stroke and venous thromboembolism [1; 9; 11].

The same review identified direct myocardial consequences of chronic use, namely left ventricular hypertrophy, collagen deposition and patchy fibrosis [11]. Across imaging and autopsy series, up to 83% of chronic users showed left ventricular systolic dysfunction with an ejection fraction below 55%, together with cardiomyopathy and an increased tendency to ventricular arrhythmia and sudden cardiac arrest [11]. Histologically, steroid-related fibrosis provides the arrhythmogenic substrate on which lethal ventricular fibrillation can develop [11]. In the perioperative period this may present as wide swings in blood pressure, acute intraoperative cardiac failure, or an emergence marked by aggression and frank psychosis [1; 3; 9; 11]. Orally active 17-alpha alkylated preparations carry their own liability: raised transaminases, cholestasis, peliosis hepatis and impaired hepatic drug metabolism [1; 9; 11].

Other classes raise different concerns [1; 9].

Protein and peptide hormones, among them human growth hormone (hGH), insulin-like growth factor-1, insulin and human chorionic gonadotrophin, are used for their anabolic and anti-catabolic effects [1; 9]. Chronic hGH misuse produces acromegalic features, macroglossia and pharyngeal hypertrophy included, which together can create severe upper airway obstruction and a genuinely difficult intubation [1; 9]. Exogenous insulin brings the risk of profound intraoperative hypoglycaemia, a diagnosis easily masked by anaesthesia and capable of causing irreversible neurological injury before it is recognised [1; 9].

Stimulants such as amphetamines, ephedrine, cocaine and high-dose caffeine are taken to sharpen alertness, shorten reaction time and assist weight loss [1; 9]. Acute intoxication produces hypertension, tachycardia, hyperthermia and an increased minimum alveolar concentration requirement for volatile agents [1; 9]. Chronic use has the opposite effect, depleting central catecholamine stores and predisposing to refractory intraoperative hypotension that fails to respond to indirect sympathomimetics and calls instead for a direct alpha agonist such as phenylephrine or noradrenaline [1; 9; 17].

Beta-2 agonists, oral clenbuterol and fenoterol in particular, are used for bronchodilation and for beta-2 mediated anabolism [1; 9]. Clenbuterol toxicity manifests as severe sinus tachycardia, ventricular arrhythmia, hypokalaemia, lactic acidosis and QTc prolongation, a combination that raises the risk of perioperative cardiac arrest [1; 9].

Oxygen transport enhancers, meaning recombinant human erythropoietin, blood transfusion and xenon, are favoured by endurance athletes seeking greater red cell mass and oxygen-carrying capacity [1; 9]. Supraphysiological haematocrit above 50% to 55% increases blood viscosity and predisposes to systemic hypertension, coronary thrombosis, cerebral infarction and postoperative deep vein thrombosis [1; 9]. Preoperative hydration and active thromboprophylaxis are indicated [1; 9].

Diuretics and masking agents, furosemide, spironolactone and probenecid among them, are used either to dilute urine ahead of testing or to achieve rapid weight loss in weight-categorised sports [1; 9]. Their abuse leaves the patient hypovolaemic, hypokalaemic and hyponatraemic, a state in which induction can precipitate haemodynamic collapse [1; 9].

8. Pain perception, regional anaesthesia and local anaesthetic blocks

Postoperative analgesia in the athlete has to serve recovery without introducing side effects that delay it [1; 3; 5; 9]. Two neurobiological mechanisms are usually invoked to explain the variability of pain perception in this group [1; 3; 9].

The first is endorphinergic. Intense exercise raises circulating beta-endorphins and downregulates central opioid receptors [1; 3; 9]. After injury or surgery, enforced inactivity causes endogenous endorphin release to fall abruptly, receptors upregulate in rebound, and opioid sensitivity becomes unpredictable [1; 3; 9]. Postoperative requirements consequently range from marked sensitivity at one extreme to considerable tolerance at the other [1; 3; 9].

The second is behavioural. Athletes in competitive contact sports acquire a learned tolerance of pain, and some will under-report on a visual analogue scale in the hope of hastening discharge and return to play [3; 5; 9]. A low score should not always be taken at face value.

Regional anaesthesia and peripheral nerve blockade offer targeted analgesia with reduced systemic opioid exposure, but their use in competitors calls for a frank discussion beforehand [1; 3; 5; 9]. Permanent neurological injury is rare, in the order of 0.04% after neuraxial anaesthesia and under 3% after peripheral nerve blockade, yet even a transient neuropraxia or motor deficit lasting a few weeks can derail a career that depends on precise movement [1; 3; 9].

Gultekin and colleagues [5] reviewed 1,970 local anaesthetic painkilling injections given to 540 elite athletes across professional rugby league, soccer, American football and Australian football. Safety, they found, depends heavily on where the needle goes [5]. Injections into non-weight-bearing structures, the acromioclavicular joint (440 injections), hand and fingers (362), sternoclavicular joint and sternum (203) and iliac crest (110), were associated with good long-term outcomes [5]. On follow-up averaging five years, 68% of athletes reported no residual pain in the injected area, 93% expressed satisfaction with the procedure, and 61% said they would repeat it without hesitation [5].

The picture at load-bearing sites is less reassuring. Injection of the knee and ankle, or of the ligamentous structures of the wrist and thumb base, was associated with higher rates of long-term complication [5]. Reported events included complete rupture of the Achilles tendon, adductor longus and flexor digitorum, plantar fascia rupture, chronic joint instability and accelerated osteoarthritis [5]. The mechanism proposed is straightforward enough: abolishing

protective nociceptive feedback in a primary load-bearing joint allows uninhibited mechanical overloading, and a partial structural injury becomes a complete one [5].

9. Anti-doping regulation, WADA compliance and shared decision-making

Anyone anaesthetising a competitive athlete works within the WADA Prohibited List, which distinguishes substances banned at all times from those banned in competition only [1; 3; 9].

- Agents permitted perioperatively include propofol, etomidate, ketamine, midazolam, thiopental, the local anaesthetics lidocaine, bupivacaine and ropivacaine, muscle relaxants, paracetamol, non-steroidal anti-inflammatory drugs and direct alpha agonists such as phenylephrine [1; 9].
- Banned in competition are the potent opioids (fentanyl, alfentanil, sufentanil, morphine, pethidine, oxycodone, hydromorphone), central stimulants including ephedrine and adrenaline, and, in precision disciplines such as archery and shooting, beta-blockers [1; 9].
- Banned at all times are systemic glucocorticosteroids given orally, intravenously or intramuscularly, recombinant erythropoietin, human growth hormone, insulin in the non-diabetic athlete, anabolic steroids, diuretics, and intravenous infusions exceeding 100 mL per 12-hour period unless given during a hospital admission, a surgical procedure or a clinical investigation [1; 9].
- Where a prohibited substance is clinically necessary, fentanyl for surgical analgesia or systemic dexamethasone being the obvious examples, the anaesthesiologist should document the indication in detail and help the athlete lodge a Therapeutic Use Exemption (TUE) with the relevant national anti-doping organisation [1; 3; 9].

Decisions about eligibility, surgery and anaesthetic planning are best framed within the five pillars of shared decision-making described by Martinez and colleagues [8]:

Knowledge. A working command of sports physiology, cardiac remodelling, anaesthetic pharmacology and anti-doping rules [8].

Humility. Recognising the limits of one's own expertise and involving sports cardiologists, orthopaedic surgeons and team physicians when the findings are ambiguous [8].

Respect. Taking seriously the athlete's autonomy, personal goals and individual tolerance of risk [8].

Teamwork. Bringing the athlete, family, coaching staff and team medical personnel into the same conversation [8].

Communication. Recording risk-benefit discussions, perioperative plans and TUE submissions in a form that will stand up to later scrutiny [8].

10. Discussion

Taken as a whole, the evidence reviewed here indicates that anaesthetising an athlete is less a matter of applying special rules than of understanding why the usual assumptions may not hold. Four areas account for most of the difference: cardiovascular remodelling and autonomic variability; body composition and drug disposition; the toxicity of performance-enhancing agents; and the regulatory and communicative demands that surround competitive sport [1; 6; 8; 11].

Cardiovascular and autonomic adaptation is the first of these and probably the most consequential. Resting bradycardia in the trained athlete reflects both high vagal tone and intrinsic sinoatrial remodelling, so it is not simply a matter of parasympathetic excess that can be argued away [1; 2; 7; 10; 12]. Induction agents and opioids strip away sympathetic tone and leave that vagal predominance exposed, which is the common thread running through the reported cases of severe bradyarrhythmia, junctional rhythm and asystole [1; 2; 7; 12; 14]. The practical implications are modest but non-negotiable: review the preoperative electrocardiogram rather than glancing at it, monitor haemodynamics continuously, and have an anticholinergic within arm's reach at induction [1; 2; 7; 9]. It is also worth remembering that chronic training represents a sustained adaptive load on metabolic and stress-response systems more broadly, and that this wider context may modulate the perioperative response in ways not captured by cardiovascular measurements alone [15].

Pharmacokinetic reasoning argues against total body weight as a scalar for intravenous induction hypnotics in the very muscular patient [1; 6]. Dosing propofol to TBW overestimates what the central compartment requires, and the predictable results are excessive plasma concentrations, rapid loss of consciousness and post-induction hypotension [6]. Scaling to lean body weight with the Janmahasatian formula avoids the overshoot and tends to preserve haemodynamic stability [1; 6]. The caveat, restated because it is easily lost, is that this recommendation is extrapolated from obese cohorts and has not been tested directly in athletes; it is a reasonable default, not a validated one.

Airway assessment deserves more attention than it usually receives in a fit young patient. Neck muscularity, restricted cervical mobility and higher Mallampati scores, the last of which correlate with pinch strength and hand dimensions in the data of Erbesler and colleagues, together justify preparing for difficult mask ventilation and having a video laryngoscope available [1; 4; 9]. Covert PED use compounds all of the above, adding accelerated coronary disease, left ventricular dysfunction and hypercoagulability to the list of things that must be screened for and, where present, actively managed with thromboprophylaxis [1; 9; 11]. Finally, adherence to WADA regulation and a structured approach to shared decision-making are what keep the anaesthetic care not only safe but defensible and aligned with what the athlete is actually trying to achieve [1; 3; 8; 9].

Several limitations should temper these conclusions. The evidence base is small, heterogeneous and dominated by case reports and physiological studies conducted in non-athletic populations; randomised data obtained in athletes are essentially absent. Much of what is recommended above therefore rests on mechanistic reasoning rather than on outcome data, and that distinction should be preserved when these observations are translated to the bedside.

11. Conclusions

General anaesthesia in the athlete calls for an individualised, evidence-informed approach that takes account of physiological adaptation, gives due weight to cardiovascular and airway assessment, treats drug dosing with caution, and anticipates bradyarrhythmia rather than reacting to it. The athletic heart and resting hypervagotonia should be managed proactively if intraoperative bradyarrhythmia and asystole are to be avoided.

Induction hypnotics such as propofol are better titrated against lean body weight than total body weight, with the aim of limiting cardiovascular depression, while acknowledging that direct evidence in athletic populations has yet to be generated.

Performance-enhancing drug toxicity requires deliberate screening and, where indicated, thromboprophylaxis. Compliance with WADA regulation and a structured framework for shared decision-making complete the picture, and together they help ensure that perioperative care is safe, effective and supportive of the athlete’s return to health and to competition.

Disclosure

Author contributions

Conceptualization: Karolina Pyrtek-Kajzer, Klaudia Kapelka, Julia Raszewska Methodology: Karolina Pyrtek-Kajzer, Klaudia Kapelka, Julia Anna Swierczek Investigation: Julia Anna Swierczek, Weronika Targosz, Gabriel Mandera Resources: Aleksandra Wesolowska, Adam Perek, Julia Raszewska Data curation: Adam Perek, Karolina Pyrtek-Kajzer, Klaudia Kapelka Writing, rough preparation: Wiktor Jablonski, Klaudia Kapelka, Aleksandra Wesolowska Writing, review and editing: Karolina Pyrtek-Kajzer, Dawid Sobanski, Julia Anna Swierczek Visualisation: Weronika Targosz, Julia Raszewska, Karolina Pyrtek-Kajzer Supervision: Klaudia Kapelka, Gabriel Mandera, Weronika Targosz Project administration: Dawid Sobanski, Aleksandra Wesolowska, Adam Perek

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In preparing this work the authors used Gemini by Google for language editing and grammar correction only. After using this tool the authors reviewed and edited the text as required and take full responsibility for the substantive content of the publication.

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