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Venous Thromboembolism in Athletes, Including Paget–Schroetter Syndrome:

A Narrative Review

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Abstract

Background. Venous thromboembolism (VTE), comprising deep vein thrombosis (DVT) and pulmonary embolism (PE), is traditionally linked to immobility, surgery and malignancy, yet it also affects young, apparently healthy athletes. Exercise-related cardiovascular protection coexists with transient exercise-induced hypercoagulability and sport-specific mechanical and environmental exposures, creating a distinctive, easily overlooked risk profile.

Aim. To synthesise the medical evidence on VTE in athletes, with particular attention to Paget–Schroetter syndrome (effort thrombosis of the axillary–subclavian vein), and to critically appraise its pathophysiology, diagnosis, treatment and return-to-play.

Material and methods. Narrative review of case reports, case series, cohort studies and topical reviews addressing hemostasis, thrombophilia, upper- and lower-extremity DVT, PE and anticoagulation in athletic populations.

Results. Athletes are exposed to all three arms of Virchow’s triad through repetitive microtrauma, hemoconcentration, venous stasis during travel and immobilisation, and post-exertional inflammation, frequently compounded by inherited thrombophilia, hormonal therapy or occult malignancy. Paget–Schroetter syndrome typically affects young overhead athletes and is best managed by anticoagulation, catheter-directed thrombolysis and first-rib resection; anticoagulation alone carries high rates of residual obstruction and recurrence. Standard clinical decision rules and athlete physiology, notably a low resting heart rate, may mislead and delay the diagnosis of DVT and PE.

Conclusions. A high index of suspicion, athlete-adapted diagnostics, individualised anticoagulation and structured return-to-play planning are essential. VTE in athletes is both a physical and a psychosocial burden requiring multidisciplinary care.

Keywords: venous thromboembolism, deep vein thrombosis, pulmonary embolism, Paget–Schroetter syndrome, effort thrombosis, athletes, anticoagulation

1. Introduction

Venous thromboembolism (VTE) is a common and clinically significant condition that primarily affects the venous system of the lower limbs, although in selected cases it also involves the upper limbs or the inferior vena cava. The disease comprises two closely related clinical entities, deep vein thrombosis (DVT) and pulmonary embolism (PE), and thrombus formation is driven by the components of Virchow's triad: venous stasis, an imbalance between procoagulant and anticoagulant factors, and endothelial injury [1]. The annual incidence of VTE in European populations is approximately 150 per 100,000 people, that is one to two per 1,000 people per year, and recognised risk factors include advanced age, hospitalisation, prolonged immobilisation, major surgery, malignancy, hormonal contraception and trauma [1]. Thrombosis is one of the main causes of death worldwide and the most common cause among non-communicable diseases; almost ten million people suffer a thromboembolic event each year, provoked by surgery, cancer and other risk factors, although a large proportion of events are unprovoked [2].

Pulmonary embolism is the most feared manifestation of the disease. It occurs when one or more of the pulmonary arteries become blocked by a thrombus displaced most often from the deep veins of the legs, disrupting normal physiology and, when the clot burden is substantial, producing severe haemodynamic instability. The primary determinant of outcome is the effect on the right ventricle and its capacity to adapt: right ventricular afterload can rise acutely because of both mechanical obstruction and hypoxic vasoconstriction, and the leading cause of mortality in acute PE is right ventricular dysfunction and subsequent failure [3]. Because the symptoms of PE are frequently non-specific and may mimic myocardial infarction, heart failure or pneumonia, the diagnosis is often not obvious, and this diagnostic difficulty is magnified in populations not traditionally regarded as being at risk [3].

The stereotypical VTE patient is sedentary, recently operated, bedridden or elderly, and most clinicians readily recognise this profile. Athletes appear to defy these expectations. Regular physical activity is one of the best established protective factors against cardiovascular disease, and the paradox that vigorous exercise might promote rather than prevent thrombosis is counterintuitive. Nevertheless, thromboembolic events do occur in young, apparently healthy, highly conditioned individuals. A retrospective review of injury reports from the four major North American professional leagues identified 55 VTE events between 1999 and 2016, of which 19 were upper-extremity DVT, 15 lower-extremity DVT, 15 PE and 6 combined DVT with PE; the mean age at the time of the event was 29.3 years, and the mean time lost from play was 6.7 months, with seven athletes never returning to competition and one death [4].

These figures, drawn from media reports and therefore almost certainly an underestimate, demonstrate that VTE in athletes is neither anecdotal nor benign.

The apparent contradiction between the cardiovascular benefits of exercise and the occurrence of thrombosis in athletes is resolved by recognising that competitive and endurance sport exposes participants to a distinctive combination of thrombogenic influences. These include repetitive mechanical microtrauma to the vessel wall, transient exercise-induced hypercoagulability, hemoconcentration from dehydration, systemic inflammation, prolonged venous stasis during long-distance travel to and from competition, and periods of immobilisation following injury or orthopaedic surgery. When these acquired exposures are superimposed on inherited thrombophilia, hormonal therapy or occult malignancy, the balance can tip toward pathological clotting even in a physically elite person [4].

Within this broad topic, one entity is particularly emblematic of the athletic population: Paget-Schroetter syndrome (PSS), also termed effort thrombosis or venous thoracic outlet syndrome, a primary thrombosis of the axillary-subclavian vein associated with repetitive, strenuous use of the upper extremity. Thoracic outlet syndrome comprises three variants according to which neurovascular structure is compressed as it exits the thoracic cavity: neurogenic (approximately 95% of cases), venous (approximately 4%) and arterial (approximately 1%); PSS is the effort-related subset of the venous variant [5]. It has a yearly incidence of one to two per 100,000 people and accounts for 1-4% of all venous thrombosis episodes, most commonly affecting young, otherwise healthy men in their early thirties, with athletes such as baseball players, swimmers and weightlifters and manual workers performing repetitive overhead motion at particular risk [5]. Clinicians must be familiar with its symptoms, physical examination findings and initial management, because early intervention can be limb-saving and occasionally life-saving [6].

This narrative review synthesises the medical evidence on VTE in athletes, with a particular emphasis on Paget-Schroetter syndrome. It examines the pathophysiology of exercise-related hemostatic change; the acquired and genetic risk factors that predispose athletes to thrombosis; the presentation, diagnosis, treatment and outcomes of effort thrombosis of the upper extremity; the spectrum of lower-extremity and central venous thrombosis in athletes; the diagnostic pitfalls of pulmonary embolism in this population; the principles of anticoagulation and return to play; and the psychosocial burden of thrombotic disease on young competitors. Throughout, the aim is to compare and critically appraise the available literature, which is dominated by case reports and small series, and to identify where consensus exists and where important gaps remain.

2. Venous thromboembolism in athletes: burden and paradox

The epidemiology of VTE specifically within athletic populations is poorly defined. Established incidence data for VTE, one to two events per 1,000 person-years in the general adult population, are derived from cohorts in which athletes are not separately identifiable, and there is currently no reliable estimate of incidence in the athlete subset [4]. This uncertainty reflects several factors: the rarity of the event, difficulties with diagnosis and clinical suspicion, and the possibility that some events undergo self-resolution before they are detected. Case reports and small series therefore remain the principal source of knowledge, and they are inherently prone to publication and selection bias.

Despite these limitations, a coherent picture emerges. The largest descriptive study to date, the review of professional American sport leagues, found that VTE led to an average of 6.7 months lost from play, that the overall return-to-play rate was 85.4%, and that athletes with upper-extremity DVT returned to play significantly faster (mean 4.3 months) than those with lower-extremity DVT (5.9 months), PE (10.8 months) or combined DVT and PE (8.2 months) [4]. Major League Baseball had a significantly higher number of upper-extremity DVT than the other sports, and all affected baseball players were pitchers, consistent with the biomechanical demands of repetitive overhead throwing. Identifiable risk factors, such as a known or family history of a clotting disorder, prior clot, recent injury or surgery, were present in only 22 of 55 events, so that a majority of episodes had no major identifiable provoking factor, suggesting a more subtle predisposition related to the effects of strenuous exercise, travel and the sport itself [4].

Athletes are susceptible to a wide variety of vascular injuries, secondary either to repetitive motion or to high-speed collision, and the reported spectrum extends well beyond classic venous thrombosis to include arterial and unusual venous events across many sports [7]. The literature documents effort thrombosis and vascular injury in baseball, swimming, wrestling, weightlifting, martial arts, American football, hockey, rowing and gymnastics, as well as in more unexpected settings such as golf, complicated by subclavian artery dissection, racquet and ball sports associated with ulnar and radial artery thrombosis, billiards, umpiring, jogging, backpacking associated with portal vein thrombosis, and trampolining and skiing associated with popliteal vessel injury, and a fatal pulmonary embolism has been reported after an isolated anterior cruciate ligament tear [7]. This breadth underlines that the mechanical and haemodynamic stresses of sport can injure the vasculature in territory-specific ways, and that a diagnosis of vascular injury, although seldom the first consideration in an athlete with limb

pain, must remain on the differential to avoid catastrophic limb-threatening or life-threatening outcomes [7].

Professional athletes are exposed to a number of acquired conditions associated with an increased risk of thrombosis, including tissue trauma from contact during play, hemoconcentration from dehydration, circulatory stasis secondary to bradycardia associated with high cardiopulmonary fitness, and prolonged immobilisation during travel or after injury [4]. Frequent long-distance travel is a phenomenon particular to elite sport, with games played nationwide and internationally; several studies have documented an increased risk of VTE with travel exceeding four to six hours, and athletes often travel immediately after competition, in the prothrombotic post-exercise state. High altitude, relevant to teams that train or compete at elevation, adds a further layer of risk through hypoxia and hemoconcentration [4]. The recognition that athletes are simultaneously protected by their cardiovascular fitness and exposed by the demands of their sport frames the central paradox that runs through the whole of this review.

The epidemiological contribution of travel and environment has been examined in the professional-league dataset. Studies in the general population report a roughly four-fold increased risk of VTE after four hours of travel and an increased risk after six hours of air travel, and athletes frequently travel immediately after competition in a prothrombotic post-exercise state; marathon studies have shown that the combination of air travel exceeding four hours and endurance running induces a prothrombotic shift, with an increase in procoagulant relative to fibrinolytic activity [4]. Although no association was found between a team's geographic location and VTE risk, the National Hockey League accounted for the largest number of events, a finding linked to the extensive travel of hockey players and possibly to the cold playing environment, given that several studies have detected a higher frequency of DVT in winter months, attributed to cold-induced prothrombotic changes, vasoconstriction and the association between winter respiratory infection and hypercoagulability [4]. Heightened awareness of the signs and symptoms of VTE is therefore warranted for athletes who undertake frequent long-haul travel and for those who train and compete at elevation [4].

3. Exercise, hemostasis and the pathophysiology of thrombosis in the athlete

3.1. Virchow's triad in the athletic context

Thrombus formation in any patient reflects the interplay of the three arms of Virchow's triad, and each arm is engaged in the athlete in ways that are specific to sport. Venous stasis arises

from prolonged travel, bed rest after injury and the venous compression that accompanies certain anatomical variants and repetitive postures. Hypercoagulability arises transiently from post-exercise hemoconcentration and inflammation and may be amplified by inherited or acquired thrombophilic states. Endothelial injury arises from the repetitive microtrauma inflicted on vessel walls by high-force, high-repetition movement and by direct contact trauma. As has been emphasised in the athletic literature, dehydration and subsequent polycythaemia exemplify hypercoagulability, travel to competition and bed rest after injury illustrate venous stasis, and repetitive microtrauma to the venous and arterial vasculature demonstrates vessel-wall injury; an athlete can reasonably be exposed to at least one and up to all three components of the triad [8].

This framework applies equally to the upper and the lower limb. In the upper extremity, repetitive retroversion, hyperabduction and extension of the arm impose excessive strain on the subclavian vein, leading to microtrauma of the endothelium and activation of the coagulation cascade, the mechanistic basis of Paget-Schroetter syndrome [5,6]. In the lower extremity, the popliteal, posterior tibial and peroneal veins are susceptible to intimal trauma from the sudden hyperextension and torsion experienced during a soccer kick or tackle, or to direct compressive forces from contact [7]. Deep venous thrombosis is classically related to venous stasis, intimal injury and coagulation diathesis, and each of these can be produced by athletic activity [7].

3.2. Acute and chronic exercise-induced hemostatic responses

Hemostasis is a tightly regulated system that preserves blood fluidity under normal conditions while enabling rapid clot formation after vascular injury, maintained through interactions between procoagulant mechanisms (platelet activation, thrombin generation and fibrin formation) and anticoagulant and fibrinolytic pathways involving tissue plasminogen activator, plasmin, antithrombin and the protein C system [9]. Physical activity is protective against cardiovascular disease in the general population, yet accumulating evidence indicates that strenuous exercise, particularly prolonged endurance activity, can transiently shift the hemostatic balance toward a procoagulant state [9]. Endurance athletes are exposed to physiological conditions known to influence coagulation, including dehydration, hemoconcentration, systemic inflammation and endothelial shear stress.

Acute bouts of strenuous endurance exercise induce measurable activation of several components of the hemostatic system. Circulating platelet counts increase by approximately 15-25% immediately after competition through splenic contraction and catecholamine-mediated mobilisation, accompanied by elevated expression of platelet activation markers such as P-selectin and glycoprotein IIb/IIIa; marathon studies have documented substantial post-

exercise increases in thrombin-antithrombin complexes and D-dimer [9]. Exercise duration is a critical determinant: short bouts lasting less than one hour produce mild and rapidly reversible changes, whereas efforts exceeding two to four hours, such as marathon or Ironman segments, can sustain elevations of thrombin generation markers for several hours, with thrombin-antithrombin complexes remaining raised for four to six hours after a full marathon. These prolonged responses are attributed in part to metabolic acidosis from lactate accumulation, which lowers blood pH and enhances factor Xa activity, and to cytokine release such as interleukin-6, which stimulates plasminogen activator inhibitor-1 production [9].

Hydration status strongly modifies these responses. Even moderate dehydration can concentrate coagulation factors by approximately 15-20%, increase blood viscosity and reduce circulating antithrombin, and athletes who become substantially dehydrated during endurance events demonstrate significantly higher D-dimer and factor VIII levels than adequately hydrated participants; controlled oral rehydration can attenuate post-exercise fibrinolytic activation by nearly half [9]. Environmental and demographic variables further modulate the response, with high temperatures increasing sweat-induced fluid loss and altitude stimulating hypoxia-related pathways that raise haemoglobin and haematocrit [9].

In contrast to these acute pro-thrombotic shifts, habitual endurance training produces adaptive antithrombotic changes. Regular aerobic training enhances endothelial function through increased nitric oxide bioavailability, improves fibrinolytic potential with higher resting tissue plasminogen activator activity and lower plasminogen activator inhibitor-1, reduces platelet aggregability by 10-25% in response to ADP and collagen, and lowers resting inflammatory markers such as C-reactive protein and interleukin-6 [9]. Trained athletes also return to baseline hemostatic parameters more rapidly after exercise, within 12-24 hours compared with up to 48 hours in sedentary individuals. These adaptations are partially reversible, however, and periods of detraining of three to six weeks reduce endothelial nitric oxide availability and increase platelet reactivity [9]. The net effect is best described as a biphasic model: acute strenuous exercise induces transient hypercoagulability, whereas chronic training confers long-term protection. Understanding this physiological paradox is central to interpreting the occurrence of VTE in otherwise healthy competitors [9].

Several experimental and observational studies have quantified this response. Investigations in marathon runners have reported significant increases in coagulation factors and fibrinolytic markers immediately after competition, indicating that endurance events activate coagulation and fibrinolysis simultaneously in a dynamic regulatory response; analyses of platelet activation have demonstrated increased expression of markers such as P-selectin after

prolonged exercise, consistent with sympathetic activation and elevated shear stress; and studies of long-distance trail running have documented significant rises in thrombin-antithrombin complexes and other coagulation markers [9]. Comparisons across event types are informative: whole-blood analyses of marathon, triathlon and long-distance cycling found that coagulation was activated across all three, but significant platelet activation occurred only in the running-intensive events, correlating with the greater thrombotic risk associated with long-distance running, a distinction that a global measure of clot firmness alone cannot capture and that argues for dedicated platelet assessment in future work [9,10]. Endurance training itself modifies these responses, enhancing fibrinolytic capacity through increased endothelial release of tissue plasminogen activator, so that well-trained athletes recover from transient prothrombotic changes approximately twice as fast as untrained individuals [9].

Molecular case studies reinforce the concept that intense effort can unmask latent hemostatic disorders. In an elite basketball player who suffered an occlusive femoral vein thrombosis, an integrated biochemical, haematological and genetic work-up revealed marked thrombocytosis and a hypercoagulable thrombin-generation profile that classical clotting tests failed to detect, ultimately attributed to essential thrombocythaemia carrying a CALR exon 9 deletion complicated by acquired von Willebrand disease [8]. The authors underline that exercise is generally thought to protect against thrombosis through the controlled balance between exercise-activated coagulation and fibrinolysis, yet athletes are exposed to many acquired risk factors, including trauma, immobilisation, long-haul travel, dehydration and post-exercise hemoconcentration, that may predispose them to thrombotic events, and that the transient exercise-induced increase in clotting, platelet clumping and fibrinolytic activity can combine with an underlying disorder to precipitate an event [8].

3.3. Insights from ultraendurance sport

Ultraendurance events push the hemostatic system to extremes, and recent work with whole-blood viscoelastic testing has begun to characterise the resulting profile. Prolonged running activates coagulation and, to a degree, the counterbalancing fibrinolysis, producing a transiently rebalanced state; however, some evidence suggests the balance may tip unfavourably during recovery, with coagulation remaining elevated while fibrinolysis wanes [10]. In elite competitors sampled before and within about three hours of an 86.9 km mountain trail race, a shift toward a hypercoagulable, inflammation-associated profile was detected, with prolonged prothrombin time coupled with reduced factors V, VII and X but a markedly shortened activated partial thromboplastin time driven by elevated factors VIII and IX and von

Willebrand factor; plasminogen fell and alpha-2-antiplasmin rose alongside a spike in D-dimer, a pattern consistent with an inflammatory fibrinolysis-shutdown seen in critical illness, and post-race clots were firmer and more resistant to lysis [10]. Although none of these athletes developed clinical thrombosis, these findings illustrate that an ultraendurance race triggers coagulation changes qualitatively similar to an acute inflammatory or hypercoagulable state, and that a second hit such as dehydration or a long-haul flight after the event may tip the balance toward symptomatic thrombosis [10].

4. Acquired and genetic thrombotic risk factors in athletes

4.1. Inherited thrombophilia

Inherited thrombophilias represent one of the most important non-modifiable risk factors, and their contribution to VTE in athletes is a recurring and unresolved theme. Factor V Leiden confers a three- to seven-fold increased lifetime risk of VTE, and when combined with physiological stressors such as dehydration or prolonged immobilisation during travel, this predisposition may substantially increase thrombotic risk [9]. Other clinically important inherited defects include the prothrombin G20210A mutation and deficiencies of the natural anticoagulants protein C, protein S and antithrombin, all of which can amplify exercise-induced hemoconcentration and endothelial activation [9].

The synergy of multiple defects is powerfully illustrated by the case of an amateur football player who developed a post-traumatic lower-limb DVT after a blunt injury and was found to be double heterozygous for factor V Leiden and prothrombin G20210A in the setting of a strong family history of thromboembolism [11]. Heterozygous carriers of factor V Leiden and of the prothrombin gene mutation have, respectively, a five- and three-fold higher risk of first venous thrombosis compared with the general population, and the co-existence of both defects, a rare event occurring in roughly 0.1% of people, is associated with an eighteen- to twenty-fold higher risk [11]. The authors argue that thrombotic genetic testing in athletes who experience a DVT can inform patient management, for example by justifying prolonged anticoagulation or the avoidance of trauma-related sport during treatment, particularly when a strong family history is present and other provoking factors are absent [11]. This position is not universally accepted, and controversy persists regarding the extent of prothrombotic genetic testing in a young, otherwise healthy athlete with a provoked event; nonetheless, the identification of a high-risk genotype can shift an apparently low-risk, trauma-provoked first event into a category warranting extended therapy [11].

Not all thrombophilia work-ups are positive, and their absence does not exclude effort thrombosis. In several reported athletes with axillary-subclavian or lower-limb thrombosis, extensive hypercoagulability panels, including factor V Leiden, prothrombin gene mutation, protein C and S, antithrombin, lupus anticoagulant and antiphospholipid antibodies, were entirely negative, and the event was attributed to mechanical and exertional factors alone [12,13]. The interpretation of thrombophilia testing is further complicated by anticoagulant therapy itself: assays for protein S, protein C and lupus anticoagulant can be affected by direct oral anticoagulants, so that borderline abnormal values obtained during treatment may be uninterpretable, as was explicitly noted in a young cyclist whose protein S activity was low while on apixaban [14].

4.2. Malignancy and cancer-associated thrombosis

Malignancy is one of the strongest acquired risk factors for VTE, and although it is uncommon in young athletes it must remain on the differential, particularly for events in unusual locations or without a clear mechanical cause. Cancer-associated thrombosis, historically termed Trousseau's syndrome, reflects a hypercoagulable state driven by tumour-derived procoagulants, and VTE develops in up to 20% of cancer patients over the course of their disease, with cancers of the pancreas, brain, lung, ovary and haematological malignancies carrying the highest risk [15]. The mechanisms are multifactorial and include tumour expression of tissue factor and release of tissue-factor-bearing microvesicles, cancer procoagulant that directly activates factor X, plasminogen activator inhibitor-1 overexpression, and mucin-mediated platelet activation [15]. Cancer patients treated with vitamin K antagonists have a roughly three-fold increased risk of VTE recurrence and a two- to six-fold higher risk of bleeding compared with non-cancer patients, and international guidelines therefore favour low-molecular-weight heparin, and increasingly direct oral anticoagulants, for at least three to six months [15].

The relevance of occult malignancy to the athletic population is exemplified by a 47-year-old male weightlifter who presented with upper-limb DVT initially attributed to Paget-Schroetter syndrome, in whom staging imaging unexpectedly revealed a papillary renal cell carcinoma [16]. Because roughly 30% of cases of upper-extremity DVT are secondary to malignancy, and because venous thromboses of the upper limbs are more frequently associated with occult cancer than those of the lower limbs, the authors stress the importance of a complete evaluation to exclude malignancy in patients with unusual VTE, even when a sport-related mechanical explanation is available [16]. A comparable message emerges from an endurance runner whose

venous thromboembolism was reported in the context of Lynch syndrome and prior colon cancer, underscoring that athletes carry the same medical comorbidities as their less active peers [17].

4.3. Hormonal, environmental and pharmacological factors

Combined oral contraceptives, hormone therapy and pregnancy are established provoking factors for VTE and are highly relevant to female athletes. In an athletic 40-year-old marathon runner who developed both a dural venous sinus thrombosis and a calf DVT, the thrombotic risk profile combined recent cancer history, oral contraceptive use (Yaz) and the athletic exposures of hemoconcentration, dehydration, muscular injury and systemic inflammation; the contraceptive was discontinued at diagnosis because of its association with venous thrombosis [17]. This case also illustrates that endurance athletes may possess a higher pain threshold and may write off leg symptoms that a sedentary person would report, so that DVT is discovered incidentally, and that scoring systems such as the Wells score, which award a point for cancer and none for athletic exposures, may systematically underestimate their risk [17].

Environmental stressors amplify these responses. High ambient temperatures increase sweat-induced fluid loss and hemoconcentration, and altitude exposure above 2,000 m stimulates hypoxia-related pathways that raise haemoglobin and haematocrit after acclimatisation, increasing blood viscosity and procoagulant activity [9]. A striking example is a professional baseball pitcher whose effort thrombosis presented as pulmonary embolism at an altitude of more than 5,200 feet; high-altitude thrombosis is thought to be multifactorial, involving a hypobaric hypoxic atmosphere, low humidity and adaptive hyperfibrinogenaemia, and altitude should be considered a contributory factor in athletes competing at elevation [18]. Pharmacological and supplement-related exposures specific to sport, including anabolic steroids, unregulated workout supplements, and polycythaemia induced by altitude training or exogenous red-cell administration and stimulation such as blood doping or erythropoietin use, have all been proposed as contributors to hypercoagulability in athletes and belong in the differential diagnosis of VTE in this population [14].

4.4. Psychological stress

An emerging and less familiar risk factor is psychological stress, which is highly relevant to competitive athletes exposed to intense performance pressure. A growing body of epidemiological work links psychosocial stress and stress-related psychopathology to VTE. Post-traumatic stress disorder has been associated with abnormal symptomatic venous clotting in national registry data, women with the highest post-traumatic stress symptom burden have

an almost two-fold greater risk of VTE than those unexposed to trauma, and long working hours, disaster exposure and intimate partner violence have all been associated with increased VTE incidence and recurrence [19]. The proposed mechanism is stress-induced hypercoagulability, in which activation of the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis increases secretion of catecholamines, cortisol and vasopressin, tipping the antithrombotic-prothrombotic balance of the vessel wall and blood toward clotting [19]. Although this literature is not athlete-specific, the high psychological demands of elite competition make it a plausible and underexplored contributor to thrombotic risk in athletes, warranting awareness of psychological stress as part of the etiological assessment [19].

5. Paget-Schroetter syndrome (effort thrombosis of the upper extremity)

5.1. Definition, history and epidemiology

Paget-Schroetter syndrome is a condition of the upper extremity characterised by the formation of a deep vein thrombus in the subclavian or axillary vein. It was first described in detail by James Paget in 1875, and in 1884 Leopold von Schrotter was the first to propose vascular trauma from repetitive overhead activity as its etiology; Hughes later coined the eponym in the mid-twentieth century after compiling numerous case reports [20]. It is uncommon, affecting fewer than 0.5-1 in 100,000 individuals annually, and those most affected are athletes or manual labourers who perform repetitive overhead activities [20]. Upper-extremity DVT overall accounts for approximately 5-10% of all DVT and can be classified as primary (idiopathic or effort-related) or secondary; PSS has classically been characterised as a primary etiology accounting for more than half of all primary cases, although it is increasingly recognised that anatomical compression at the costoclavicular junction is a truly extrinsic factor, which would reclassify the syndrome as a secondary cause [20]. Secondary causes of upper-extremity DVT include indwelling catheters and pacemakers, malignancy and hypercoagulable states, and these must be excluded before a diagnosis of PSS is accepted [20]. Case series enrich the demographic picture drawn from individual reports. A German series of 34 patients with PSS over a ten-year period found that eight demonstrated a clear relationship between the thrombosis and a sporting activity, and a systematic review of PSS in athletes reported that the majority were male, that the average age was 24 years and that roughly one third had no relevant past medical history, confirming that effort thrombosis is predominantly a disease of young, healthy, active individuals [21]. Across the reported athletic cases, symptom onset is usually acute or subacute and follows a discrete episode of arm exertion, and

the classic triad of venous distension, arm swelling and aching pain, sometimes accompanied by bluish discolouration and prominent subcutaneous collateral veins, recurs consistently [6,21]. Recognition of this stereotyped presentation in a young athlete with recent repetitive overhead activity should prompt immediate evaluation, because the interval to effective thrombolysis is short and outcomes deteriorate once the thrombus organises [6].

The demographic is consistent across series: young, otherwise healthy males in their late twenties to early thirties predominate, and the dominant arm is most often affected. A systematic review of PSS in athletes reported an age range of 16-56 years with an average of 24 years, a male predominance of approximately 80%, and about one third of patients having no relevant past medical history [21]. Sports repeatedly implicated include baseball, particularly pitching, swimming, wrestling, weightlifting, gymnastics, martial arts, rowing and throwing events, and occupations involving continuous overhead work such as electricians, plumbers and mechanics, sometimes termed industrial athletes, carry similar risk [6,11].

5.2. Anatomy and pathogenesis

The thoracic outlet is bordered superiorly by the clavicle, inferiorly by the first rib, medially by the costoclavicular ligament and laterally by the anterior scalene muscle; as the axillary vein passes over the first rib and under the clavicle it becomes the subclavian vein, which then traverses this narrow costoclavicular space [5]. Chronic compression of the vein between the clavicle and first rib during arm abduction leads to venous endothelial damage, inflammation, scarring and potential thrombosis. Most patients with venous thoracic outlet syndrome have a costoclavicular ligament that inserts more laterally, and hypertrophy of the subclavius and anterior scalene muscles further contributes to venous compression and injury [5]. In athletes, hypertrophy of the overhead musculature, particularly the anterior scalene and subclavius, can compress the subclavian vein as it passes through the thoracic inlet, and this compression is especially evident in anatomical variants where the costoclavicular ligaments extend more laterally [20].

Beyond static compression, repetitive microtrauma is central to the pathogenesis. Repeated abnormal and strenuous interaction between the venous system and the structures of the thoracic outlet, evidenced by the dense external fibrosis and adhesions commonly found around the vein at surgery, induces intimal hypertrophy and a cycle of partial thrombosis and recanalisation that precedes the clinical manifestation of the syndrome [20]. As the arm moves, the vein is stretched and small tears occur, causing further damage and predisposing to thrombosis; over time progressive intrinsic injury produces fibroelastic strictures, turbulent

flow and clot formation, with extensive collateral formation [11]. Interestingly, cervical ribs, which are more posterior structures, are typically implicated in neurogenic or arterial thoracic outlet syndrome rather than in venous effort thrombosis, although they and other bony or ligamentous anomalies can contribute [11]. Unrecognised thrombophilic states contribute to thrombus formation in a large proportion of primary upper-extremity DVT but in only a minority of effort-related cases, reinforcing the mechanical basis of PSS [20].

5.3. The spectrum of presentation: from elite athletes to occupational and low-intensity exposures

The clinical presentation of PSS is relatively uniform. Patients typically present with acute or subacute swelling, heaviness, aching pain, redness or bluish discolouration of the affected upper extremity, often within 24 hours of an inciting activity, together with dilated, visible superficial veins across the shoulder and upper arm, a sign sometimes attributed to Urschel; palpable cord-like thrombosed veins may be felt in the axilla [5,6]. Because collateral veins can partially decompress the limb, some patients are initially asymptomatic or paucisymptomatic, and complaints in overhead athletes can mimic shoulder or elbow problems, presenting as loss of velocity or control, arm heaviness or a dead arm [11]. Symptom onset frequently follows a discrete precipitating event related to arm exertion, although trivial daily activities can also trigger the syndrome [13].

The reported case literature spans the full spectrum from elite competitors to occupational and remarkably low-intensity exposures, and this breadth is instructive. Classic athletic cases include a young collegiate and recreational swimmer whose repetitive over-the-head strokes produced retroversion, hyperabduction and extension of the arm with resulting axillary-subclavian thrombosis [22], a Division I collegiate and professional context in which effort thrombosis has been documented in baseball, swimming, wrestling and weightlifting [6], and a judo tutor who noticed an acutely red, swollen and painful arm after a routine training session [23]. Weightlifting and resistance training are recurrent themes: an avid weightlifter presented with initial and recurrent left subclavian thrombosis and right pulmonary emboli that required surgical reconstruction only two weeks after presentation [20], and a case of upper-limb DVT in a weightlifter prompted a search that uncovered occult renal carcinoma [16].

Equally instructive are the non-athletic and low-intensity presentations, which demonstrate that the label effort thrombosis should not be restricted to competitive sport. A 33-year-old waiter developed subclavian vein thrombosis attributed to maintaining shoulder abduction while carrying several plates for hours, with dynamic magnetic resonance venography confirming

obliteration of the subclavian vein within the costoclavicular triangle during shoulder abduction but not in the neutral position [24]. A 67-year-old competitive bowler developed multi-level upper-extremity DVT the morning after a prolonged game, in an atypical location distal to the classic costoclavicular site, and a 25-year-old barista whose work involved lifting 30-pound bags of coffee overhead developed classic subclavian-axillary thrombosis with first-rib compression [25]. A house painter who performed repetitive abduction, flexion and external rotation of the shoulder developed effort-induced DVT of the basilic, axillary and subclavian veins [12], and a firefighter who was also an avid runner developed extensive right subclavian, axillary, brachial and basilic thrombosis after his routine six-mile run, with venography confirming venous thoracic outlet syndrome [21]. These reports collectively emphasise that a high index of suspicion is required whenever spontaneous upper-limb venous thrombosis follows repetitive or sustained arm activity, regardless of the intensity of the activity or the athletic status of the patient [12,21,24,25].

A further nuance concerns the arm affected. Although PSS characteristically involves the dominant extremity, effort thrombosis can occur in the non-dominant arm when a sport imposes repeated bilateral loading. An 18-year-old collegiate offensive lineman, whose position required repeated forward-flexed elevation of both arms to maintain a pass block, sustained a non-dominant-arm effort thrombosis, a distribution the authors regarded as unsurprising because both extremities were subjected to the same repetitive action [26]. A small number of reports also describe an absence of common predisposing factors and even involvement of the left, non-dominant arm after aerobic exercise, again with a negative hypercoagulable work-up, reinforcing the concept that minor thoracic inlet abnormalities combined with strenuous activity can suffice [13].

5.4. Diagnosis

The gold standard for diagnosing upper-extremity DVT is venography, but compression duplex ultrasonography is the first-line imaging modality because it is non-invasive, inexpensive and reproducible, with reported sensitivity of 78-100% and specificity of 82-100% [5,11]. Key sonographic features include non-compressibility of the vein, absence of Doppler flow and, as the thrombus organises, increasing echogenicity, together with prominent peripheral collateral veins; positional manoeuvres may be required to reproduce dynamic compression [11]. A fresh clot may be echolucent and therefore not directly visualised, so the lack of compressibility is a key diagnostic feature [11]. Importantly, ultrasound can miss a central thrombus lying directly beneath the clavicle because of acoustic shadowing, and duplex may be falsely negative in the

proximal subclavian vein, as occurred in a young weightlifter whose venous Doppler and repeated ultrasounds of all extremities were negative despite strong clinical suspicion, with the diagnosis ultimately confirmed by venography [20].

When ultrasound is inconclusive but clinical suspicion remains high, computed tomographic or magnetic resonance venography can confirm the diagnosis and interrogate the surrounding anatomy, and each has drawbacks: CT venography carries contrast and radiation exposure, while MR carries high cost and limited availability [5]. Dynamic imaging that reproduces the patient's habitual provocative posture is particularly valuable; in the waiter described above, time-of-flight MR venography demonstrated that the subclavian vein was patent in the neutral shoulder position but compressed within the costoclavicular triangle during abduction, directly visualising the mechanism and guiding preventive advice about postural change [24]. A chest radiograph to exclude a cervical rib and a point-of-care ultrasound to assess Doppler flow are useful initial tools in the emergency department [20]. Plasma D-dimer may be elevated in upper-extremity DVT but has low specificity, ranging from 14-60%, and is not recommended as a confirmatory test, although it may serve as an adjunct [5].

A thrombophilia work-up is not required for diagnosis and is not routinely recommended, but it is prudent when the thrombosis is unexplained or a family history is present, and it may help identify an etiology and inform the duration of anticoagulation; when performed, it should ideally precede heparin initiation or be undertaken after an appropriate interval off anticoagulation, and typically includes factor V Leiden, prothrombin G20210A, antithrombin, protein C and protein S, lupus anticoagulant, and anticardiolipin and anti-beta-2-glycoprotein antibodies [5,20]. Cancer screening after unexplained upper-extremity thrombosis follows recommendations similar to those for the lower limb, with routine screening not advised but consideration given to a thorough history, examination, routine laboratory studies and age- or sex-appropriate investigations in genuinely unexplained cases [5].

The differential diagnosis of a suspected VTE in an otherwise healthy young athlete is broad and must be considered explicitly, because several benign conditions mimic thrombosis and, conversely, thrombosis mimics musculoskeletal injury. For unilateral limb swelling the differential includes lymphoedema, malignancy, haematoma and infection, and specific athletic risk factors that should be sought include Paget-Schroetter syndrome in those with repetitive upper-extremity movement, tissue injury from contact sport, immobilisation after injury or orthopaedic surgery, polycythaemia from altitude training or exogenous erythropoietic stimulation, and possible hypercoagulability from anabolic steroids or unregulated supplements [14]. In the lower limb, exertion-induced rhabdomyolysis may be

mistaken for effort thrombosis, as in the woman with post-exertional calf DVT whose mildly elevated creatine phosphokinase and negative urine myoglobin helped exclude significant rhabdomyolysis while the persistent oedema prompted the Doppler that confirmed the diagnosis [27], and occult bony injury or rupture of a popliteal cyst must be excluded by imaging in the post-traumatic patient [1]. Conversely, effort thrombosis of the arm can masquerade as shoulder tendinitis or muscular strain, and thoracic outlet syndrome as cervical or brachial-plexus pathology, so that a deliberate vascular assessment is required whenever the clinical picture is atypical [6].

5.5. Pulmonary embolism complicating Paget-Schroetter syndrome

Historically, PE was thought to be rare in upper-extremity DVT, but this view has been revised, and awareness of the phenomenon is important. Reported rates of PE in association with PSS range from 20-30%, and although the absolute risk is smaller than for lower-extremity DVT, it is clinically meaningful [5]. The most dramatic illustration is a 25-year-old professional baseball pitcher whose only presenting complaints were dizziness and shortness of breath, without any upper-extremity symptoms; he was found to have segmental pulmonary emboli and, on ultrasound, an acute subclavian and axillary effort thrombosis, with venography confirming complete occlusion of the subclavian vein on forward shoulder flexion [18]. This case demonstrates that effort thrombosis can present silently in the arm and manifest first as a symptomatic and potentially fatal PE, and that physicians caring for throwing athletes should include effort thrombosis in the differential diagnosis of unexplained dyspnoea and fatigue [18]. The subacute PSS case reported by O'Toole and colleagues similarly featured concurrent right-sided pulmonary emboli discovered on presentation despite an initially non-diagnostic upper-limb ultrasound, and the authors emphasise that clinicians must consider life-threatening PE and favour an aggressive approach because delayed diagnosis and conservative management carry real risk [20].

5.6. Treatment

The management of PSS involves three components, deployed in combination according to the patient's age, functional status and the age of the thrombus: systemic anticoagulation, endovascular thrombolysis or thrombectomy, and surgical thoracic outlet decompression [20]. Immediate systemic anticoagulation after diagnosis is the first step, and the 2016 CHEST guidance recommends direct oral anticoagulants such as dabigatran, rivaroxaban, apixaban or edoxaban over vitamin K antagonists for VTE without cancer, and a minimum three-month

course after any upper-extremity DVT [5]. Anticoagulation alone, however, is generally inadequate for PSS: it does not lyse the existing thrombus, and symptomatic relief is related more to the development of collateral veins than to resolution of the clot, so that up to 75% of patients have residual venous obstruction, symptoms persist in as many as 91% and permanent disability affects a substantial minority [11]. A recent meta-analysis found that 46% of patients treated with anticoagulation alone have persistent symptoms [20].

A more aggressive strategy involving catheter-directed thrombolysis followed by surgical decompression is therefore superior in patient-reported outcomes such as symptom resolution and return to work [5]. Thromboses are conventionally classified as acute (less than 14 days old), subacute (14 days to three months) or chronic (older than three months), and this timing strongly influences success [20]. Catheter-directed thrombolysis is most effective for acute clots, with early success rates of 75-84%, and its efficacy diminishes rapidly as the thrombus organises, so that treatment of a clot older than about two weeks is much less successful, with reported success as low as 29% at two to twelve weeks [5]. For subacute and organised thrombi that resist thrombolysis, mechanical and pharmacomechanical approaches are increasingly used; a case treated with mechanical aspiration thrombectomy using a Penumbra device as an adjunct to catheter-directed thrombolysis achieved successful recanalisation with reduced tissue-plasminogen-activator exposure, shorter time to lysis and lower cost [28], and the ClotTriever mechanical thrombectomy device has been used in the acute-subacute phase before decompression [29].

Definitive treatment of the underlying anatomical compression is surgical decompression, most commonly transaxillary or paraclavicular first-rib resection with anterior scalenectomy, with or without middle scalenectomy, and removal of a cervical rib if present [5,20]. Because resolution of the presenting thrombus does not prevent recurrence where anatomy predisposes to it, and because chronic recurrence leads to vascular scarring and vessel obliteration that is rarely amenable to surgical correction, prompt decompression is advocated in good surgical candidates [20]. The combination of thrombolysis and first-rib resection has been shown to prevent recurrence in approximately 96% of cases, and success rates of 90-95% have been reported when PSS is diagnosed and treated promptly with early decompression [5,20]. Angioplasty and venous stenting as sole therapies have generally performed poorly because of the extrinsic nature of the compression, with stent fracture, deformation and re-thrombosis, and stenting is not recommended before rib resection [11]. An adjunctive temporary arteriovenous fistula has been used in selected reconstructions to reduce recurrence and permit early detection of complications [18].

The optimal timing and intensity of surgery, and the role of a more conservative approach, remain debated. A systematic review found that some authors regard rib resection as potentially overtreating a subclavian lesion, yet more than 40% of conservatively treated patients ultimately require rib resection for recurrent thrombosis, suggesting that an aggressive approach may be more efficient and even safer [20]. The subacute PSS case with rapid two-week recurrence and clot extension despite mechanical thrombectomy and anticoagulation led its authors to advocate an equally aggressive approach to subacute as to acute thrombi, arguing that the small likelihood of overtreatment is outweighed by the risks of recurrence, post-thrombotic syndrome, vessel fibrosis and pulmonary embolism [20]. Others describe successful non-operative management in selected patients when collateral development alleviates symptoms, so that management must be individualised [13].

A notable minority of cases challenge the paradigm that decompression is always required. An 18-year-old offensive lineman with non-dominant-arm effort thrombosis, no traumatic injury and no external compression was treated successfully with four weeks of anticoagulation alone, returning to unrestricted football with no recurrence at 18 months, although the authors cautioned that this approach was chosen by a multidisciplinary team on the basis of the specific clinical picture and should not be generalised to all patients [26]. Similarly, a woman with left, non-dominant axillary-subclavian thrombosis and a negative hypercoagulable work-up was managed with anticoagulation alone and was asymptomatic with resolved clot at two months, in the absence of any anatomical obstruction [13]. These reports indicate that in the absence of demonstrable anatomical compression, and after careful multidisciplinary assessment, anticoagulation alone may occasionally suffice, whereas the presence of extrinsic compression strongly favours decompression [13,26].

The surgical literature provides more granular outcome data than individual reports. In a series of 32 high-level athletes undergoing surgical management for venous thoracic outlet syndrome, seven required repeat surgery, the risk of recurrent thrombosis or reoperation was relatively high at 22%, and the average return to play was 4.4 months, with no difference in return-to-play time based on the level of the athlete or the sport [6]. It has been estimated that approximately half of patients treated with decompression and venolysis alone retain a patent vein, and a temporary arteriovenous fistula has been recommended in the majority to reduce the risk of re-thrombosis and to permit early detection of complications, as was performed in the professional pitcher who subsequently returned to five further seasons at the major-league level [11,18]. Rehabilitation after decompression typically permits passive range of motion during anticoagulation, strengthening by about one month, a light throwing programme at eight

weeks and interval throwing at twelve weeks, with an average return to sport of around 3.5 months [11]. Anticoagulation for three to six months after recanalisation and decompression is recommended, with ultrasonography used to monitor progress [11].

5.7. Outcomes, recurrence and post-thrombotic syndrome

The principal long-term complication of PSS is post-thrombotic syndrome, characterised by chronic pain, heaviness and swelling of the affected extremity, which occurs in 7-46% of patients with upper-extremity DVT and is more common in primary than in secondary disease [5]. Residual deep venous thrombosis and chronic disability have been described in up to 40-90% of PSS patients treated conservatively, and recurrent or chronic thromboembolic pulmonary complications can also occur [24]. Early treatment correlates with improved patient-reported outcomes: success rates of 90-95% are achievable with prompt diagnosis and immediate decompression, and near-full recovery at one year with low recurrence rates has been reported after first-rib resection and scalenectomy [5]. In competitive athletes managed with catheter-directed thrombolysis, first-rib resection and systemic anticoagulation, return to the previous level of competition has been reported at a mean of approximately 3.5 months, although the risk of recurrent thrombosis or reoperation can be as high as 22% [11]. The consistent message across the surgical literature is that rapid diagnosis and a proactive, multimodal approach optimise both venous patency and the athlete's return to sport [5,6,11].

6. Lower-extremity and central venous thrombosis in athletes

6.1. Trauma-related lower-extremity deep vein thrombosis

Although lower-extremity DVT with a traumatic sporting injury in otherwise healthy active adults is seldom reported, trauma is one of the most common causes of lower-extremity DVT, and athletic vascular injury must be considered during the initial examination [7]. The classic illustration is a 42-year-old former semi-professional soccer player who sustained a posterior lower-extremity contusion during a tackle and was diagnosed with a traumatic DVT of the distal femoral, popliteal and distal calf veins, confirmed on duplex ultrasound and D-dimer; his only major thrombotic risk factor was the traumatic injury, and he was treated with unfractionated heparin and warfarin for six months plus compression stockings for nine months [7]. The popliteal, posterior tibial and peroneal veins are susceptible to intimal trauma from the sudden hyperextension and torsion of a soccer kick or tackle, and the popliteal vessels are particularly vulnerable to direct, shearing and muscular compressive forces because of their anatomical position [7]. This report also emphasises that failure to recognise a vascular injury can have catastrophic limb-threatening or life-threatening implications, and that a complete

bilateral duplex sonography is the primary diagnostic method, augmentable by evidence-based risk-factor analysis [7].

Post-traumatic DVT can also unmask inherited thrombophilia, as in the double-heterozygous football player discussed earlier, in whom a blunt lower-limb injury during a match precipitated a posterior tibial vein thrombosis on a background of factor V Leiden and prothrombin G20210A [11]. This intersection of mechanical trauma and genetic predisposition reinforces the argument for considering thrombophilia testing in athletes with apparently provoked thrombosis and a suggestive family history [11]. The soccer-player case additionally highlights the long tail of morbidity, with the patient continuing to experience intermittent lower-leg discomfort and knee effusion one year after the event, and the general observation that intrinsic fibrinolysis preserves venous valve function poorly, so that valves become fibrotic and fixed after a few weeks trapped in clot, predisposing to post-thrombotic syndrome [7].

6.2. Effort-related and exertion-induced lower-extremity thrombosis

Just as effort thrombosis affects the upper extremity, unaccustomed or intense lower-limb exertion can precipitate DVT even without direct trauma, a much rarer and less well recognised entity. A previously sedentary 25-year-old woman developed anterior tibial and popliteal vein thrombosis within 24 hours of walking 25 km in a single day, with the only laboratory abnormality being a mildly elevated creatine phosphokinase, and with no thrombophilia, contraceptive use or family history; the authors postulate that unaccustomed exertion caused muscle damage and local muscle spasm that, with continuous exertion, produced vessel injury and thrombosis [27]. They note that only a handful of cases of lower-limb venous thrombosis after intense physical activity had previously been reported, and that awareness of lower-extremity effort thrombosis is needed so that this diagnosis is considered when a person involved in exercise or sport presents with leg pain or swelling [27]. This mirrors the postulated pathophysiology of Paget-Schroetter syndrome, in which heavy exertion causes microtrauma to the vessel intima and activates the coagulation cascade, with significant thrombosis occurring after repeated insults, especially where mechanical compression coexists [27].

6.3. Central and iliac vein compression syndromes

Compression of the central veins can produce lower-limb or pelvic thrombosis in athletes through mechanisms analogous to thoracic outlet compression in the arm. A 22-year-old competitive cyclist presented with extensive right lower-extremity DVT caused by iliac vein compression between a hypertrophied psoas muscle, the common iliac artery and the L5

vertebral body, the first reported case of DVT from psoas muscle hypertrophy in a young cyclist [14]. Young cyclists commonly develop psoas hypertrophy from training, and the authors argue that this mechanism should be considered in cyclists presenting with lower-limb DVT; the patient also carried a homozygous factor V Leiden mutation, so that the etiology was viewed as a combination of extrinsic compression, possible vascular injury from repetitive cycling and an elevated baseline coagulopathic risk [14]. Management combined anticoagulation, a temporary detraining period and, later, venography with intravascular ultrasound and balloon dilation, although a stent was deliberately withheld given the risk of stent complications from ongoing muscular compression in a young athlete [14].

A second central presentation is a 22-year-old long-distance collegiate runner who developed acute right iliofemoral venous outflow obstruction on a background of bilateral internal iliac (hypogastric) vein thrombosis, in the absence of anatomical abnormality or hypercoagulable disorder, while taking combined oral contraceptives [30]. She had reported progressively worsening hip pain despite reduced training and presented with phlegmasia; she was treated with thrombolysis-free mechanical thrombectomy using ClotTriever and aspiration devices, with complete flow restoration, and the contraceptive was discontinued [30]. This case demonstrates that competitive athletes can develop extensive proximal DVT, that right-sided iliofemoral DVT carries a higher risk of subsequent PE, and that early minimally invasive mechanical thrombectomy is an emerging option that avoids the bleeding risk associated with thrombolysis while aiming to prevent post-thrombotic syndrome [30].

6.4. Intramuscular calf-vein thrombosis: an under-recognised entity

Thrombosis of the intramuscular veins of the calf, the soleal and gastrocnemius veins, is an uncommon and frequently overlooked manifestation of VTE that is highly relevant to trauma patients, including athletes, in whom orthopaedic injuries dominate the initial assessment [1]. A physically active 36-year-old man sustained a knee injury while skiing and presented with three weeks of persistent knee and shin swelling; magnetic resonance imaging and an initial ultrasound of the popliteal system showed no significant intra-articular pathology and no thrombosis, yet a subsequent comprehensive Doppler examination of the lower limb revealed extensive thrombosis of the soleal and medial gastrocnemius muscle veins with preserved patency of the larger deep and superficial trunks [1]. The key sonographic criteria are non-compressibility, intraluminal echogenic material, vein dilatation (typically above 4-5 mm for soleal and gastrocnemius branches) and absence of colour flow, and the treatment in this patient was low-molecular-weight heparin with subsequent follow-up [1].

This entity carries important lessons for sports medicine. Physicians not infrequently evaluate the venous system in a cursory manner, restricting attention to the larger deep and superficial veins and neglecting the intramuscular component, yet intramuscular thrombi connect with the tibial venous system and drain directly into the popliteal vein, so that they can propagate proximally and embolise to the pulmonary circulation [1]. Persistent post-traumatic calf swelling, even with a normal MRI and an unremarkable initial Doppler, therefore warrants targeted ultrasound of the soleal and gastrocnemius veins, and a comprehensive evaluation of the superficial, deep and intramuscular venous systems should be standard in trauma patients with unexplained calf oedema [1]. Although the progression risk of isolated intramuscular thrombosis is lower than that of proximal DVT, the dominant strategy of active surveillance with follow-up Doppler must be balanced against evidence that some isolated intramuscular thromboses progress to proximal DVT and PE, so that early anticoagulation may benefit selected patients [1].

The choice of imaging modality for suspected calf and intramuscular thrombosis reflects a hierarchy of usefulness. Doppler ultrasound with compression assessment is the current gold standard for evaluating intramuscular calf veins, being non-invasive and allowing assessment of both proximal and distal segments, though it requires a skilled sonographer and adequate compression at more than three levels [1]. Contrast phlebography, once the diagnostic gold standard, is now largely obsolete because it is invasive and carries the risks of intravenous contrast; magnetic resonance imaging can differentiate acute from chronic thrombus and is useful when ultrasound is inconclusive, with acute thrombus appearing hyperintense on T1-weighted sequences; and computed tomography, while involving radiation and iodinated contrast, is preferred when thrombosis is localised to the iliac or pelvic veins [1]. An interdisciplinary approach involving radiologists and phlebologists, with deliberate assessment of the deep, superficial and intramuscular venous systems, is critical for timely diagnosis, particularly in post-traumatic patients [1].

6.5. Postoperative and orthopaedic thrombosis in the young athlete

Orthopaedic injury and surgery, common in athletes, are potent provoking factors, and DVT can complicate the postoperative course even in adolescents in whom it is least anticipated. A fit 16-year-old basketball player developed an infrapopliteal DVT of the fibular and posterior tibial veins four days after open reduction and internal fixation of a displaced tibial tuberosity fracture combined with closed reduction of forearm fractures, presenting with fever and severe calf pain after compartment syndrome and infection had been excluded [31]. His thrombotic

risk factors were few, comprising an open orthopaedic surgery with poly-fracture, postoperative immobilisation in casts and an equivocal infection, most of which are present in many orthopaedic patients [31]. He was treated with therapeutic enoxaparin and intravenous cefazolin, mobilised in a splint, and had resolution of the thrombus on repeat Doppler at nine and eighteen days [31].

This case highlights several points of general relevance. Paediatric orthopaedic trauma surgery carries a higher rate of VTE than elective orthopaedic surgery, the risk of VTE after trauma rises toward adult levels from about 16 years of age, and conventional risk-stratification scores such as the Wells and Caprini scores perform poorly in identifying hospitalised orthopaedic children at increased risk, so that clinical vigilance and sound judgement, supported by a low threshold for D-dimer testing, are what protect a patient from a rare and unexpected DVT [31]. Had the complication been overlooked, a morbid or fatal pulmonary embolism could have resulted in a young person with his future ahead of him, and the authors call for research to refine risk stratification, treatment and safe return-to-sport planning in this growing patient group [31]. The general principles of thromboprophylaxis after orthopaedic surgery, discussed further below, are directly applicable to the surgically treated athlete [32].

6.6. Post-thrombotic syndrome and long-term outcomes

The long-term consequences of lower-extremity DVT are dominated by the post-thrombotic syndrome, which is particularly relevant to athletes for whom participation in sport is central to quality of life. Anticoagulation prevents thrombus propagation and pulmonary embolism but has no fibrinolytic activity, and radiographically demonstrable clot lysis occurs in only about half of anticoagulated patients, with complete resolution in fewer than 5%; slow intrinsic fibrinolysis does not preserve venous valve function, and valves become fibrotic and fixed after a few weeks trapped in clot [7]. The resulting post-thrombotic syndrome is characterised by brawny oedema, stasis dermatitis, hyperpigmentation, induration, ulceration and chronic leg pain, and is associated with a considerable burden of chronic pain and disability, with approximately 40% of the total cost of treating DVT spent on the post-thrombotic syndrome [7]. In one long-term study, 82% of patients with a first DVT suffered recurrent symptoms over a mean follow-up of 6.6 years, and four-level DVT, calf-vein thrombosis, ipsilateral recurrence and insufficient anticoagulation were of prognostic significance for clinically relevant symptoms up to 10-20 years after the first event [7].

There is growing evidence that the early clot lysis provided by thrombolytic therapy is more likely to preserve valve function, decreasing the likelihood of recurrence and of the post-

thrombotic syndrome, and acute thrombolytic treatment has demonstrated improved therapeutic efficacy and a decrease in post-DVT symptoms [7]. This rationale underlies the increasing use of catheter-directed and mechanical approaches in extensive proximal DVT, although randomised trials in the general population have shown that adding pharmacomechanical catheter-directed thrombolysis to anticoagulation increases major bleeding without consistently lowering the risk of post-thrombotic syndrome, so that the balance of benefit and harm remains debated [30]. For the athlete, the preservation of venous function and the avoidance of chronic post-thrombotic symptoms are of particular importance, because participation in sport is usually a central component of quality of life, and validated instruments have been developed to measure health-related and treatment-related quality of life in patients with DVT [7].

7. Pulmonary embolism and diagnostic pitfalls in the athlete

Pulmonary embolism is the most dangerous manifestation of VTE and, because its symptoms are non-specific and may be attributed to the exertion of sport, it is easily missed in athletes. The diagnostic evaluation rests on assessing pre-test probability, categorised as low, intermediate or high using the Wells or Geneva scoring systems, followed by D-dimer and imaging, most commonly computed tomographic pulmonary angiography; risk stratification is central to management because the leading cause of death is right ventricular dysfunction and failure, and key predictors of short-term mortality include overall clinical status, right ventricular dysfunction and myocardial injury indicated by elevated troponin [3]. The problem in athletes is that these clinical tools were derived and validated in the general population and may not perform reliably in individuals whose baseline physiology differs.

The most instructive example is a 20-year-old Division I collegiate basketball player who presented with pleuritic chest pain and was diagnosed with PE despite being negative by the Pulmonary Embolism Rule-Out Criteria (PERC) [33]. The PERC rule, which is 97% sensitive when applied to patients already judged to be at low risk, uses a heart-rate threshold of fewer than 100 beats per minute, but this athlete's presenting heart rate of 79 beats per minute was correctly recognised as a relative tachycardia against his true resting rate of 47-60 beats per minute; a D-dimer was ordered specifically because of his athletic status, was elevated, and led to a computed tomographic angiogram that demonstrated a segmental PE [33]. Had the relative tachycardia not been appreciated, the athlete would have been deemed PERC-negative, no D-dimer would have been obtained, and the diagnosis would have been missed [33]. Elite athletes

are known to have lower-than-average resting heart rates, and this caution should extend to any patient with a low resting rate from other causes, such as beta-blockade [33].

This case crystallises three lessons for the assessment of possible PE in athletes. First, it is a misconception that a healthy, well-appearing athlete is devoid of risk factors for VTE, because the inherent nature of competitive athletics exposes individuals to conditions that promote thrombosis, mapped onto Virchow's triad as hemoconcentration and dehydration causing hypercoagulability, extensive travel and limb immobilisation causing stasis, and musculoskeletal trauma and surgery causing endothelial damage [33]. Second, vital signs that appear normal for a typical patient may be abnormal in an athlete with high physiologic reserve, so that clinical decision rules must be interpreted in light of the patient's own baseline [33]. Third, the population's high pain tolerance and fitness can lead providers to place PE and DVT low on the differential, precisely the error that risks a missed diagnosis of a life-threatening condition [33]. In the reported athlete, PE was ultimately attributed to recent air travel or an arthroscopic meniscus repair, and he was successfully treated with three months of oral anticoagulation and returned to play, with a recommendation to take aspirin on days of team travel [33].

Pulmonary embolism can also arise from unexpected sources and coexist with other thrombotic events. Broader reviews of non-obvious PE remind clinicians that the disease can present with only mild chest pain, arm pain or fatigue, that it may be misattributed to stress, fatigue or viral infection in younger patients without apparent risk factors, and that it may be overlooked after orthopaedic surgery because dyspnoea is considered a common postoperative event [3]. The endurance runner with a dural venous sinus thrombosis and concurrent calf DVT underscores that a single athlete may harbour thrombosis in multiple territories simultaneously, and that a thromboembolic event may be the first, life-threatening manifestation of an underlying condition [17]. Given these difficulties, proper risk assessment, the judicious use of advanced imaging, and a thorough history remain essential to timely diagnosis and treatment [3].

Sex-specific differences in PE have also been described and are relevant to the mixed-sex athletic population. In a prospective cohort followed over a decade, men were more likely to have cancer and unprovoked PE, whereas women more often presented with transient risk factors such as older age, recent surgery, injury or immobilisation and hormone therapy, although adverse effects of treatment at 30 days did not differ between the sexes [3]. The broader literature on non-obvious PE additionally catalogues mechanisms that may occasionally affect physically active people, including thrombosis provoked by central venous catheter placement, hormonal therapy, pregnancy and the puerperium, and paradoxical

embolism through a patent foramen ovale, all reinforcing that a thorough history and a low threshold for imaging are required whenever PE is possible [3].

8. Anticoagulation and pharmacological treatment

Anticoagulation is the cornerstone of VTE treatment, and its principles apply to athletes with important modifications. The management of VTE proceeds in three phases: initial management to prevent clot propagation, primary treatment for three to six months given the high early risk of recurrence, and secondary prevention, with the total duration and intensity individualised according to whether the event was provoked or unprovoked [2]. The anticoagulant armamentarium has evolved over a century from unfractionated heparin, through low-molecular-weight heparin and vitamin K antagonists, to the direct oral anticoagulants, and factor XI inhibitors are now in clinical development as a potential means of preventing thrombosis while preserving physiological haemostasis [2].

Unfractionated heparin acts by binding antithrombin and inhibiting thrombin and factor Xa, but its variable dose response, need for activated partial thromboplastin time monitoring, and risks of heparin-induced thrombocytopenia and osteopenia limit its use [2]. Low-molecular-weight heparins bind antithrombin and factor Xa with higher bioavailability, more predictable dosing and fewer episodes of heparin-induced thrombocytopenia, allowing outpatient administration [2]. Vitamin K antagonists inhibit the vitamin-K-dependent factors II, VII, IX and X and proteins C and S, but require International Normalised Ratio monitoring, have a narrow therapeutic index and numerous food and drug interactions [2]. The direct oral anticoagulants, dabigatran (a direct thrombin inhibitor) and rivaroxaban, apixaban and edoxaban (factor Xa inhibitors), do not require routine coagulation monitoring, have a wide therapeutic window, and reduce the risk of intracranial haemorrhage relative to warfarin, although they depend partly on renal elimination and carry a somewhat higher risk of gastrointestinal bleeding; specific reversal agents (idarucizumab for dabigatran and andexanet alfa for the factor Xa inhibitors) are now available [2]. In practice, athletes with VTE have been treated across the full range, from unfractionated heparin bridged to warfarin, through enoxaparin, to the direct oral anticoagulants apixaban and rivaroxaban, reflecting the diversity of clinical circumstances in the reported literature [4,26].

The particular challenge in athletes is the intersection of anticoagulation with the bleeding risk inherent in contact and non-contact sport. The management of DVT and PE in athletes is complicated because contact sports such as cycling, climbing and hang-gliding carry a possibility of bodily injury during anticoagulation, and shared decision-making between the

athlete and their providers is imperative to convey the risks and benefits of anticoagulation and continued exercise [14]. Previous authors have proposed regimens of intermittent anticoagulation for athletes at risk of thromboembolism, timing doses around competition so that the plasma anticoagulant level is at a safe nadir during participation, although the long-term efficacy and safety of these strategies remain unknown [14]. Duration of anticoagulation is likewise individualised; for provoked events such as effort thrombosis treated with decompression, a minimum of three months is typical, whereas an identified high-risk thrombophilia, such as combined heterozygosity for factor V Leiden and prothrombin G20210A, may justify extended or indefinite therapy [11]. In cancer-associated thrombosis, low-molecular-weight heparin and increasingly the direct oral anticoagulants are preferred, with treatment continued for at least three to six months and beyond in active disease [15].

9. Return to play and physical activity during anticoagulation

A conspicuous gap in the literature concerns when and how athletes should return to training and competition after VTE. There are currently no discrete recommendations for return to play in VTE guidelines, nor among specific return-to-play guidelines for athletes with cardiovascular conditions, an important void given that DVT and PE occur in both amateur and professional athletes and that PE can cause sudden death in young athletic populations [14]. A scoping review of physical activity for patients with DVT and PE on anticoagulation, encompassing 26 studies, found that clear paediatric guidance is lacking and that most evidence is extrapolated from adults; it concluded that participation in low- to moderate-intensity non-contact activity while on anticoagulation appears to be safe, supported by three randomised controlled trials among other studies [34].

The available evidence supports a graduated approach. A widely cited framework recommends a gradual return to activities of daily living during the first three weeks of anticoagulation, followed from weeks four to six by a progression from non-weight-bearing to nonimpact to impact-loading activity, with close monitoring of symptoms and a return to the last asymptomatic level if symptoms recur; contact or collision sport is generally deferred until anticoagulation is complete [34]. For elite athletes who wish to continue contact sport, an intermittent anticoagulation dosing strategy has been proposed after the first three months of uninterrupted treatment, timing the last dose so that the plasma level is at a safe nadir during participation and resuming anticoagulation one to two hours afterward in the absence of trauma; this approach is based on expert opinion, requires careful counselling about the risks of bleeding and recurrence, and may be inappropriate for sports with significant contact at

every game or for patients at high recurrence risk [34]. For upper-extremity effort thrombosis specifically, some authors focus on surgical correction of the venous thoracic outlet syndrome and avoidance of the aggravating activity until it is corrected, while a non-operative approach with individualised return may be appropriate when collateral development alleviates symptoms [34].

The graduated frameworks in the literature share a common structure derived from the natural history of thrombus organisation. A frequently cited protocol developed for a young triathlete recommended three weeks of activities of daily living, during which maintenance endurance training such as tethered swimming or hand-ergometer cycling could be considered, followed by a staged reintroduction of sport beginning with non-weight-bearing activity in week four, the addition of nonimpact loading in week five, and the gradual introduction of running in week six, with the athlete advised to return to the last asymptomatic level if symptoms recurred [34]. A comparable programme progressed from walking to light jogging to running over weeks four to six, with return to non-contact sport at week seven and to contact sport only after completion of anticoagulation [34]. These frameworks are supported by controlled evidence that supervised, low- to moderate-intensity exercise beginning within days of diagnosis is safe: a randomised trial of supervised exercise starting five to seven days after DVT reported no thrombosis recurrence or other complications over six months, and further trials and cohorts of early-initiation exercise and pulmonary rehabilitation after PE reported no serious adverse events attributable to exercise [34].

Classification of activities by contact risk and intensity assists clinical decision-making. Non-contact activities in which trauma is not expected are distinguished from limited-contact, contact and collision sports in which the frequency and intentionality of impact rise progressively, and intensity is graded as light (under three metabolic equivalents), moderate (three to 5.9 metabolic equivalents and 65-75% of maximal heart rate) or vigorous (six or more metabolic equivalents and 76-96% of maximal heart rate) [34]. On this basis, participation in low- to moderate-intensity non-contact activity while on anticoagulation is regarded as safe, whereas contact and collision sport, which carry the greatest risk of bleeding-relevant impact, is generally avoided during anticoagulation unless an intermittent dosing strategy is adopted [34]. The intermittent strategy has been applied in practice, for example in an 18-year-old baseball catcher who, after three months of uninterrupted anticoagulation, timed his rivaroxaban doses around competition and returned to catching and outfield play without significant bleeding, although such approaches require individualised assessment and may be inappropriate for sports with significant contact at every game [34].

The older sports-medicine literature reached similar conclusions from first principles. General guidance for sedentary individuals allows a gradual return to daily activities over about six weeks, with no contact activity during anticoagulation, and a theoretical framework based on the natural history of thrombosis in animal models proposed combining graduated return to activity with anticoagulation and regular physician re-evaluation [7]. An athlete wishing to return to contact or collision sport should be informed of the possible increased risk of recurrent DVT above general-population estimates, and because there is no evidence specifically investigating the risk of traumatic collision and DVT recurrence, the patient and physician should make an informed, shared decision that weighs the individual risk profile, the likelihood of recurrence, athletic goals and the importance of the particular sport to quality of life [7]. Importantly, appropriately supervised physical activity after a previous VTE is generally safe and may support recovery and vascular function, provided training intensity is carefully managed and medical guidance is followed [9].

10. The psychosocial burden of venous thromboembolism in athletes

Beyond its physical consequences, VTE imposes a substantial and often underappreciated psychosocial burden on athletes, particularly the young. Because athletic identity, the degree to which a person defines themselves through sport, is consolidated during adolescence, a diagnosis that threatens sport participation can precipitate a profound sense of identity loss and significant mental-health challenges [35]. A qualitative study of adolescent athletes who sustained a DVT identified four themes: DVT posed a threat to sport participation through acute symptoms, anticoagulation-related restriction from contact sport and long-term complications such as post-thrombotic syndrome; the diagnosis and enforced separation from sport produced significant mental-health challenges including depression, anxiety, fear of recurrence and, in one athlete, stress-related pseudo-seizures; DVT functioned as an invisible disability that others, including teachers, coaches and employers, did not understand; and physical, psychological and transition support through a multidisciplinary team was needed to improve care [35].

These findings have direct clinical implications. Athletes described the diagnosis as disheartening and heartbreaking, particularly when advised to give up their sport, and some experienced a constant fear of death from the thrombus or anticoagulation; access to a psychologist, when available, was highly valued and, in some cases, transformative, and finding activities the athlete could safely perform, rather than only listing prohibitions, was identified as an important part of care [35]. The clinicians treating VTE, most often

haematologists as the first point of contact, are therefore urged to attend to both the physical and the psychological dimensions of the disease, to deliver the diagnosis and its implications with sensitivity, to monitor emotional responses, to connect athletes with mental-health resources, and to plan a transition either back to sport or, where necessary, out of it, with awareness of the loss involved [35]. The parallel epidemiological literature on psychological stress as a possible contributor to VTE further suggests that the relationship between mental health and thrombosis in athletes may be bidirectional, with stress both potentially promoting thrombosis and resulting from it, reinforcing the case for integrated psychological care [19,35]. The relationship between psychological distress and VTE appears to operate in both directions and to have at least a partly causal basis. Diagnosed depression has been reported to predict subsequent VTE, and patients with a previous VTE are at greater risk of depression, while a large collaborative Mendelian randomisation study demonstrated a positive genetic association between major depressive disorder and VTE that was independent of known risk factors, implicating shared biological mechanisms including genes involved in cytokine and immune response [19]. A prospective study of patients referred for thrombophilia investigation found that those with high levels of depressive symptoms had a four-fold greater risk of recurrent VTE, and studies of perceived stress and intimate partner violence have identified moderate and high stress as independent predictors of recurrence [19]. For the athlete, in whom competition imposes substantial and sustained psychological demands and in whom a thrombotic diagnosis itself precipitates significant distress, these findings reinforce the argument that psychological factors should be incorporated into both the etiological assessment and the ongoing care of VTE, and that mental-health support is a component of comprehensive management rather than an optional adjunct [19,35].

11. Prevention, prophylaxis and monitoring

11.1. Thromboprophylaxis in the surgically treated athlete

Because orthopaedic surgery is common in athletes and is a potent provoking factor, the principles of perioperative thromboprophylaxis are directly relevant. VTE is one of the frequent and dangerous complications of orthopaedic and trauma surgery, and its genesis is multifactorial, with orthopaedic procedures acting on all three components of Virchow's triad through altered blood flow, vessel-wall injury and a shift toward hypercoagulability [32]. Risk factors combine patient-related elements, such as genetic predisposition, obesity, active malignancy, trauma, prior VTE, venous insufficiency and, importantly, advancing age, with lifestyle factors such as smoking, oral contraceptive or hormone-replacement use, prolonged

immobilisation, travel and dehydration, and with the type of operation performed [32]. Procedures around the pelvis, hip and knee arthroplasty and proximal femoral fracture carry particularly high risk; without prophylaxis, the frequency of VTE after hip replacement is 40-60% and after knee replacement up to 85%, whereas appropriate prophylaxis reduces this to roughly 1-10% [32].

The paediatric athlete deserves specific consideration, because the evidence base is thin and the pharmacology differs. Postoperative VTE is rare in children, with one analysis of paediatric surgical patients finding an event rate of 0.10%, but paediatric orthopaedic patients, particularly those aged 15-17 years, have an increased risk compared with other hospitalised children, and paediatric orthopaedic trauma surgery carries a higher incidence still, a recent meta-analysis reporting 29 VTE events per 10,000 compared with 14 for elective orthopaedic surgery [31]. No anticoagulant is formally approved for use in children, and most data are extrapolated from adults; published safety data exist for unfractionated heparin, low-molecular-weight heparin, fondaparinux and vitamin K antagonists, whereas direct oral anticoagulants remain investigational in children, and treatment of any symptomatic paediatric DVT is nonetheless recommended, with the choice of drug, dose and duration dependent on presentation and risk factors [31]. Thromboprophylaxis is not usually indicated in paediatric populations and, in the absence of additional risk factors, is not recommended even after isolated or polytrauma orthopaedic injury, so that clinical vigilance rather than routine prophylaxis is the mainstay in this group [31].

Prophylaxis integrates general, mechanical and pharmacological measures. General measures include limb elevation, avoidance of local compression, early mobilisation, active and passive exercises, calf muscle-pump exercises and adequate hydration and nutrition; mechanical methods include graduated compression stockings and intermittent pneumatic compression; and pharmacological agents include low-molecular-weight heparin, apixaban, dabigatran, fondaparinux, rivaroxaban and, in some cases, unfractionated heparin, dosed and timed according to the procedure [32]. Mechanical methods are less effective than pharmacological ones, but the best results are obtained by combining both, and even patients without apparent risk factors and with appropriately implemented prophylaxis can still experience a thromboembolic event, underscoring the need for vigilance [32]. Diagnostic pathways rely on compression ultrasonography and D-dimer for suspected DVT and on the Wells score to guide the evaluation of suspected PE, with individual pre-operative risk assessment, for example using the Caprini score, recommended for every patient [32]. For upper-limb procedures the

routine use of prophylaxis is not required in the absence of additional risk factors, but each elderly patient should be assessed individually [32].

11.2. Hydration, compression and behavioural strategies in endurance sport

Given the interaction between exercise, environment and individual risk, several preventive strategies specific to endurance sport have been proposed. The most fundamental is the maintenance of adequate hydration before, during and after prolonged exercise, which preserves plasma volume, reduces hemoconcentration and supports normal circulation; controlled oral rehydration during endurance events has been reported to reduce post-exercise fibrinolytic activation substantially [9]. Structured training progression and adequate recovery allow the cardiovascular and hemostatic systems to adapt and minimise cumulative endothelial stress and systemic inflammation, and compression garments may attenuate the rise in coagulation markers during marathon running [9]. Athletes travelling long distances to competition, particularly after prolonged exertion, may require specific measures such as maintaining hydration and periodic movement during travel, and those with prior VTE or confirmed hereditary thrombophilia may warrant individualised, temporary pharmacological prophylaxis around periods of prolonged immobility, major competition or travel, balancing thrombosis prevention against bleeding risk [9]. Education of athletes, coaches and sports-medicine staff about the symptoms of DVT and PE, including limb swelling, unexplained pain, chest discomfort and shortness of breath, is essential for early recognition [9].

11.3. Biomarker and physiological monitoring in athletes

Laboratory medicine has assumed an increasingly important role in monitoring the health of athletes, and it offers a potential avenue for identifying those at elevated thrombotic risk. Monitoring of hemostatic biomarkers such as D-dimer, thrombin-antithrombin complexes, platelet activation indicators and fibrinolytic proteins may help identify individuals who mount an exaggerated coagulation response to exercise; although routine screening of all athletes is not currently recommended, targeted monitoring may benefit those with known thrombophilia or a history of thrombotic events [9]. The integrated biochemical, haematological and molecular evaluation that uncovered essential thrombocythaemia and acquired von Willebrand disease in an elite basketball player illustrates how a multidisciplinary laboratory approach can clarify the mechanism of an otherwise unexplained thrombotic event and personalise subsequent management, while cautioning against the indiscriminate use of specialised assays such as thrombin generation in athletes without a suggestive history [8]. Newer whole-blood

viscoelastic techniques may in future help flag athletes with impaired clot lysis after extreme exertion, although normal reference ranges in exhausted but healthy athletes are not yet established and no risk threshold has been defined [10].

Physiological monitoring of athletes intersects with thrombotic risk in two ways. First, the low resting heart rate that characterises highly conditioned athletes, and that reflects favourable autonomic adaptation, is precisely the feature that can render clinical decision rules such as PERC unreliable, as discussed above, so that objective assessment of an athlete's baseline physiology is clinically valuable [33]. Autonomic adaptation to training is measurable; heart-rate variability, for example, is a widely used non-invasive index of autonomic nervous system activity and is influenced by training modality, and comprehensive functional-fitness assessment can characterise an athlete's cardiovascular and metabolic status across a training cycle, providing context for interpreting resting vital signs during acute illness [36,37]. Second, comprehensive monitoring of general health, including nutritional status, is relevant because deficiencies common in athletes may interact with overall health; vitamin D, for example, has recognised roles in musculoskeletal and cardiovascular health and its status should be monitored across the training year, particularly in indoor athletes and during winter, as part of the holistic surveillance of the athlete's physiology [38]. While these monitoring tools are not thrombosis-specific, they form part of the broader medical oversight within which thrombotic risk in athletes is best assessed and managed [36,37,38].

12. Discussion

The evidence reviewed here establishes that VTE, although classically a disease of the sedentary, elderly or acutely ill, is a real and clinically significant problem in athletes, and that its recognition demands a deliberate departure from the textbook profile of the at-risk patient. Several themes recur across the heterogeneous literature and merit synthesis.

The first is the centrality of Virchow's triad, reinterpreted through the lens of sport. Every arm of the triad is engaged by athletic activity: endothelial injury through repetitive microtrauma and direct contact, hypercoagulability through hemoconcentration, inflammation and, transiently, exercise itself, and venous stasis through travel, immobilisation and anatomical compression [8,33]. This mechanistic unity links entities as apparently disparate as the pitcher's subclavian effort thrombosis, the cyclist's iliac compression by a hypertrophied psoas, the skier's intramuscular calf-vein thrombosis and the marathon runner's dural sinus thrombosis, and it explains why the same predisposing physiology can express itself in the upper limb, the lower limb, the pelvis or the cerebral venous system [1,14,17]. The biphasic model of exercise-

induced hemostasis, in which acute strenuous effort transiently favours coagulation while chronic training confers protection, resolves the apparent paradox that fit individuals develop thrombosis and cautions that the protective phenotype does not abolish risk during and immediately after intense exertion, particularly when a second hit such as dehydration, travel or an inherited defect intervenes [9,10].

The second theme is the primacy of Paget-Schroetter syndrome as the paradigmatic athletic thrombosis and the relative maturity of its evidence base. The case literature is remarkably consistent regarding demographics, pathogenesis and the superiority of a multimodal approach: young overhead athletes and manual workers, mechanical compression at the costoclavicular junction compounded by repetitive microtrauma, and a treatment paradigm of anticoagulation plus catheter-directed thrombolysis plus first-rib resection that prevents recurrence in the great majority [5,6,11,20]. Where the evidence is less settled, the disagreements are illuminating. The timing and necessity of surgical decompression remain debated, with a clear trend toward an aggressive approach justified by the high rate of recurrence after conservative management and by the catastrophic potential of PE, tempered by a minority of well-documented cases in which anticoagulation alone sufficed in the absence of anatomical compression [13,20,26]. The management of subacute thrombi, formerly a gap, is increasingly addressed by mechanical and pharmacomechanical thrombectomy, and the reported cases suggest that the aggressive philosophy applied to acute disease should extend to the subacute phase [20,28,29]. The breadth of the case literature, encompassing waiters, baristas, bowlers, painters and firefighters as well as elite athletes, delivers a consistent and clinically important message: effort thrombosis should be suspected whenever spontaneous upper-limb venous thrombosis follows repetitive or sustained arm activity, irrespective of the intensity of that activity [12,21,24,25].

The third theme is the fragility of standard diagnostic tools in the athletic population and the corresponding need for athlete-adapted interpretation. Clinical decision rules such as PERC and the Wells score, and even the initial imaging, can mislead: PERC can be falsely reassuring because an athlete's relative tachycardia falls below the absolute threshold, the Wells score awards no points for athletic exposures, and duplex ultrasound can miss central subclavian or intramuscular calf thrombi [1,17,33]. The recurring narrative of the missed or delayed diagnosis, whether the pitcher whose effort thrombosis presented as PE, the endurance athlete whose leg symptoms were written off, or the skier whose intramuscular thrombosis was invisible on standard imaging, points to a single remedy: a high index of suspicion, informed by knowledge of the athlete's baseline physiology and of the sport-specific mechanisms of thrombosis [1,17,18,33]. The role of D-dimer is nuanced, elevated non-specifically by exercise,

surgery and inflammation yet clinically pivotal when ordered because of an athlete's status, as in the PERC-negative basketball player [33].

The fourth theme is the immaturity of the evidence on treatment individualisation and return to play. There are no VTE-specific or cardiology-specific return-to-play guidelines for athletes, and the available recommendations are extrapolated from adults and from first principles [14,34]. Intermittent anticoagulation to permit continued participation in contact sport is a promising but expert-opinion-based strategy whose long-term efficacy and safety are unknown, and the balance between thrombosis prevention and bleeding risk in contact sport must be negotiated through shared decision-making that weighs the athlete's individual risk profile and goals [14,34]. The psychosocial dimension, long neglected, is now recognised as substantial: VTE is an invisible disability that threatens athletic identity and precipitates depression, anxiety and fear, and its management requires a multidisciplinary team attentive to mental as well as physical health [35].

Several limitations of the underlying evidence must be acknowledged. The literature is dominated by single case reports and small series, which are prone to publication and selection bias and cannot establish incidence; the largest descriptive dataset in professional athletes was drawn from media reports rather than clinical records and almost certainly underestimates the true burden while over-representing high-profile athletes with access to advanced care [4]. Much of the mechanistic and epidemiological data on exercise and hemostasis derives from small, predominantly male cohorts, often without sex-specific analysis, limiting generalisability [9,10]. There is a near-total absence of randomised evidence specific to athletes for either treatment or return-to-play, and the reported outcomes of individual cases, while encouraging, cannot be pooled meaningfully. Finally, the causal contribution of some proposed risk factors, such as psychological stress and unregulated supplements, remains associative rather than proven in athletes [14,19]. These gaps define a clear research agenda: prospective registries to establish incidence and outcomes, sex-balanced studies of exercise hemostasis, validation of athlete-adapted diagnostic thresholds, and trials of return-to-play and intermittent anticoagulation strategies.

13. Conclusions

Venous thromboembolism in athletes is an uncommon but clinically important and potentially fatal condition that defies the stereotype of the sedentary at-risk patient. On the basis of the evidence reviewed, the following principal conclusions are offered.

First, athletes are exposed to all three components of Virchow's triad through repetitive microtrauma, hemoconcentration and inflammation, and venous stasis from travel and immobilisation, and this exposure frequently combines with inherited thrombophilia, hormonal therapy, occult malignancy, altitude, supplements or psychological stress to precipitate thrombosis; the protective effect of habitual training does not eliminate the transient hypercoagulability of acute intense exertion.

Second, Paget-Schroetter syndrome is the paradigmatic athletic thrombosis, arising in young overhead athletes and manual workers from mechanical compression at the costoclavicular junction and repetitive intimal microtrauma; it is best managed by a multimodal approach of anticoagulation, catheter-directed thrombolysis and first-rib resection, whereas anticoagulation alone carries high rates of residual obstruction, recurrence and post-thrombotic syndrome, and a proactive, aggressive strategy, extended where appropriate to subacute thrombi, optimises venous patency and return to sport.

Third, thrombosis in athletes is not confined to the upper limb: lower-extremity trauma-related and effort-induced DVT, iliac and internal iliac vein compression, intramuscular calf-vein thrombosis, postoperative DVT and pulmonary embolism all occur and share the same underlying physiology, and each demands a low threshold for investigation.

Fourth, standard diagnostic tools may mislead in athletes because their baseline physiology, particularly a low resting heart rate, and their high pain tolerance can render clinical decision rules falsely reassuring and delay diagnosis; a high index of suspicion, athlete-adapted interpretation of scores and vital signs, and appropriate use of D-dimer and imaging are essential.

Fifth, anticoagulation must be individualised to balance the prevention of recurrence against the bleeding risk of sport, with intermittent dosing an emerging but unproven option for contact athletes, and structured, graduated return-to-play planning is required in the absence of dedicated guidelines.

Sixth, VTE imposes a genuine psychosocial burden, functioning as an invisible disability that threatens athletic identity and precipitates mental-health difficulties, so that comprehensive care must integrate physical treatment, psychological support and multidisciplinary transition planning. The overarching priority for future research is the generation of athlete-specific evidence, through prospective registries and trials, to replace the current reliance on extrapolation from the general population and on the accumulated wisdom of case reports.

Table 1. Representative reports of venous thromboembolism in athletes and effort-related settings

Setting / sport	Ref.	Territory	Key clinical message
Professional US leagues (NHL, MLB, NBA, NFL)	[4]	UE/LE DVT, PE	55 VTE events (1999–2016); mean age 29.3 y; UE-DVT returned to play fastest; most events lacked a major identifiable risk factor.
Skier (recreational, active)	[1]	Intramuscular calf veins	Persistent post-traumatic calf swelling with normal MRI and initial Doppler; targeted scan revealed soleal/gastrocnemius vein thrombosis.
Amateur soccer / football	[11]	Posterior tibial vein	Blunt-trauma DVT unmasked combined heterozygosity for factor V Leiden and prothrombin G20210A.
Competitive swimmer	[22]	Axillary–subclavian (UE)	Classic Paget–Schroetter syndrome; CDT then first-rib resection.
Professional baseball pitcher	[18]	Subclavian (UE) + PE	Effort thrombosis presenting only as dizziness/dyspnoea from PE at altitude; silent arm.
Offensive lineman (non-dominant arm)	[26]	Subclavian (UE)	Effort thrombosis of the non-dominant arm treated successfully with anticoagulation alone (no compression).
Waiter / bowler / barista / painter / firefighter-runner	[12,21,24,25]	Axillary–subclavian (UE)	Effort thrombosis follows sustained or repetitive arm use across occupational and low-intensity settings.
Competitive cyclist	[14]	Iliofemoral (LE)	Iliac vein compression by hypertrophied psoas plus homozygous factor V Leiden.
Long-distance collegiate runner	[30]	Bilateral internal iliac / iliofemoral	Extensive proximal DVT on combined oral contraceptives;

			thrombolysis-free mechanical thrombectomy.
Sedentary person after 25-km walk	[27]	Anterior tibial / popliteal	Lower-extremity effort thrombosis after unaccustomed heavy exertion.
Adolescent basketball player (post-op)	[31]	Fibular / posterior tibial	Postoperative DVT after ORIF of tibial tuberosity; risk scores performed poorly.
Division I collegiate basketball player	[33]	Pulmonary embolism	PERC-negative PE; relative tachycardia (79 bpm vs resting 47–60) prompted D-dimer and CT diagnosis.
Marathon runner	[17]	Dural sinus + calf DVT	Multi-territory thrombosis; OCP, malignancy history and athletic exposures; Wells score underestimates risk.

CDT – catheter-directed thrombolysis; CT – computed tomography; DVT – deep vein thrombosis; LE – lower extremity; MRI – magnetic resonance imaging; OCP – oral contraceptive pill; ORIF – open reduction and internal fixation; PE – pulmonary embolism; PERC – Pulmonary Embolism Rule-Out Criteria; UE – upper extremity; VTE – venous thromboembolism.

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