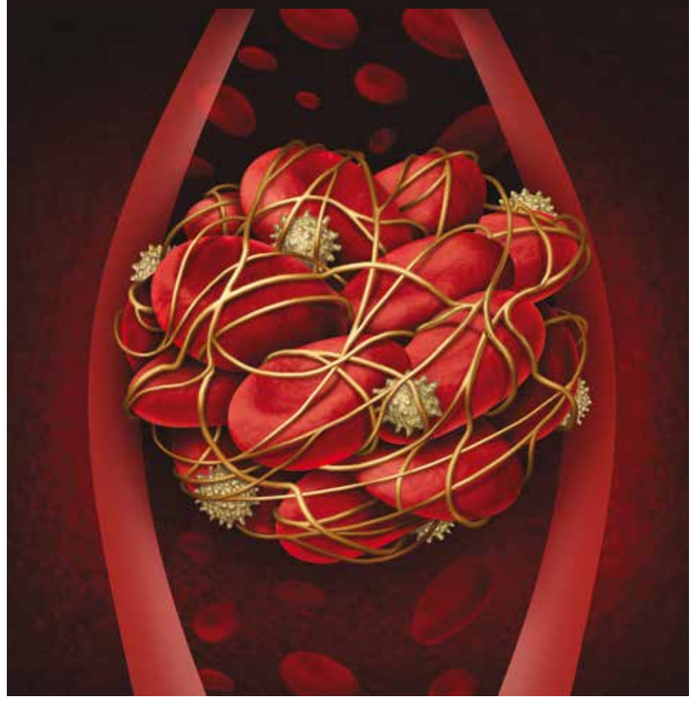
DODGING DEATH

Why you're at risk of a DVT

Specialist Vascular Surgeon, Dr Laura Redman, explains why triathletes are likely candidates for Deep Vein Thrombosis, and why awareness of this potentially fatal syndrome is key.



Deep Vein Thrombosis (DVT) usually provokes an element of fear in most because it is known to carry the risk of blocking the blood vessels to the heart and causing death. This is known as Pulmonary Embolus (PE) and is the main reason for treating DVT as an emergency. Importantly, this is not the only risk, but it may lead to lifelong problems in the leg if not treated timeously and adequately. These problems include swelling, pain, skin changes and wound formation known as "Post Thrombotic Syndrome". Thus, early treatment of deep vein thrombosis is absolutely essential.

DVT is typically thought to occur in bedridden, immobile, overweight patients, or in patients with cancer or post-surgery. However, we are seeing DVT more frequently in healthy athletes. Why? Hippocrates was one of the wisest doctors in medicine and said: "If we could give every individual the right amount of nourishment and exercise, not too little and not too much, we would have found the safest way to health." Although exercise far outweighs the benefits of no exercise, in today's competitive world, the amount required for competitive training can result in strain to the body. As an increased incidence of DVT in athletes is a fairly new awareness, we decided to explore further.

DVT explained

Veins are the blood vessels that take deoxygenated blood back to the heart to get more oxygen. If there is a change in flow, the blood may clot, causing a thrombosis. This prevents blood from leaving the leg, with resultant swelling. The clot may then propagate causing more extensive symptoms with the risk of a piece breaking off and going to the lung. Veins have valves within them as they carry blood against gravity. Thus, the clot may sit on these valves with the risk of damaging them permanently. There is a risk, in extreme cases, of stopping the arterial inflow to the leg with resultant massive swelling and the risk of gangrene and limb loss, although this is quite rare. Symptoms will depend on which veins are affected in the leg.

What will cause DVT?

Virchow's triad of statis, vessel wall injury and hypercoagulability has been known for a long time as the initiating factors for thrombus development. To prevent thrombosis, one needs good blood flow, in

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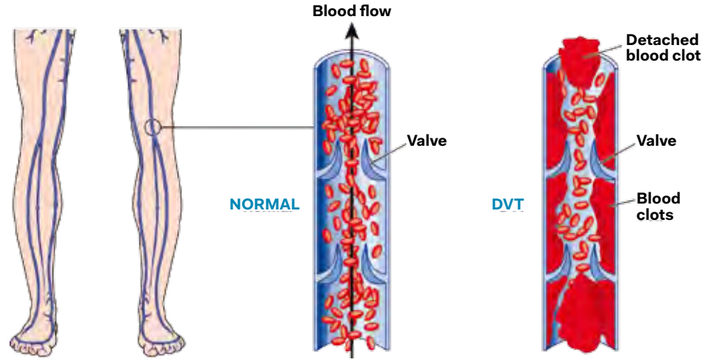
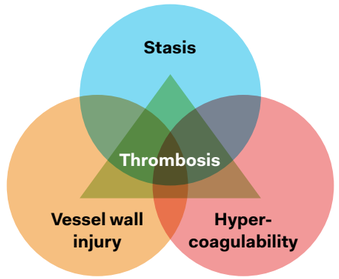


an adequate conduit (i.e. blood vessel), and correct blood consistency. If any of these are altered, there's a risk of thrombus.



Stasis refers to a change in the blood flow. Traditionally, this occurs in patients with prolonged bed rest, stroke patients or prolonged travel. So why would it occur in athletes? This may be related to change in the circulatory dynamics. Fit athletes have a low resting heart rate as well as low blood pressure. This results in "slower" blood flow through the vascular system which may predispose to thrombus. This is obviously not something that can be changed in athletes and is a physiological response to fitness, however, one will usually need another inciting event on top of the low heart rate, such as dehydration. There may, of course, be congenital anatomical structures, unrelated to exercise, that with training become exacerbated. This is not exclusive to athletes. Some patients have congenital anomalies which become obvious during pregnancy, or later in life. This can result in pelvic changes – shape and form from exercise position and muscle mass. There are known anatomical points in the pelvis where veins may become compressed. One may have initial swelling of a limb, varicose veins high up on the legs or pelvis, pain when exercising (venous claudication) or one may present with thrombosis that results in the entire leg being swollen and painful. We've seen the venous claudication and iliac vein thrombosis in athletes. This can be treated with a minimally invasive technique of stenting the vein to improve venous outflow. Should the patient present with thrombus, emergent pharmaco-mechanical thrombolysis can be performed to remove the clot (see below).

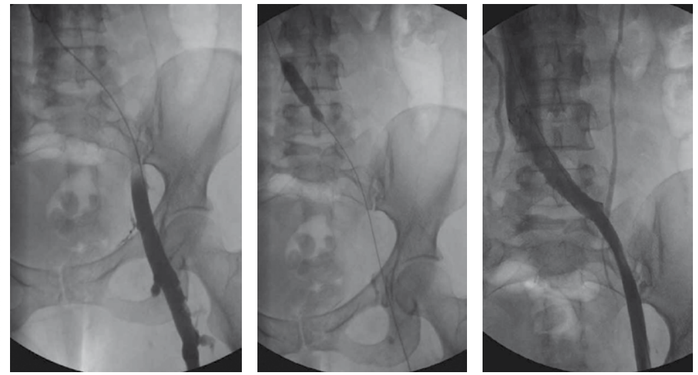
Vessel wall injury, or vascular damage, traditionally refers to injury to the vein wall by means of external trauma or placement of intravenous lines or some sort of inflammation. In athletes, this refers to physical trauma, strain or injury, as well as repetitive micro trauma.

Lastly, there is hypercoagulability, which may lead to thrombus. This refers to any cause which leads to an increased tendency to clot. There are again congenital clotting defects which may predispose one to clotting. However, often they do only predispose one, and another insult is necessary to cause the clot. Pregnancy, cancer, infection, trauma and some autoimmune diseases may cause a hypercoagulable state predisposing one to clotting. In athletes, additional causes of hypercoagulability can be dehydration. This is fairly common. If there is an underlying predisposing cause to thrombosis and one is not adequately hydrated, the risk of thrombus formation is high.

The elephant in the room

Drugs, legal and illegal, cannot be ignored. It is well known that oestrogen is hypercoagulopathic and thus any steroid formulation will have some clotting risk. The use of steroids for enhanced performance is not approved. The doses athletes may use are higher than those used medically, and formal studies of any kind cannot ethically be done. Not only do steroids increase blood clotting risk, but they affect the rest of the vascular tree, raising blood pressure, worsening cholesterol profile and affecting the heart. There are physiological changes that occur when one takes anabolic steroids that are known to increase clotting risk and it is believed that there is a connection between non-fatal heart attack and stroke in athletes using steroids. A post mortem of a 22-year-old athlete who died of a heart attack showed thrombus in the main coronaries.

Growth hormone is used by some athletes, and a direct relationship to



Emergent pharmaco-mechanical thrombolysis involves placing a catheter into the iliac vein and instilling a drug that breaks down the clot. Thereafter, a catheter can manually suck the clot out. This helps reduce the risk of developing post thrombotic syndrome.

clotting has been proven in mice. Growth hormones affect the clotting factors in the liver. While they increase the risk of thrombosis in both men and women, it is more relevant in men. It is known to affect the cardiovascular system in other ways – enlarging the heart, raising blood pressure, increasing the risk of diabetes and raising cholesterol.

Erythropoetin (EPO) is usually used to help patients with anaemia and some athletes have been using it to increase red blood cells to increase their oxygen carrying capacity. This, however, makes the blood thicker and more prone to clotting. EPO is known to increase thrombotic events of DVT, pulmonary embolus, heart attack and stroke.

Diuretics are used by some athletes to decrease weight for lighter weight classes or as a masking agent to hide drug usage during testing. Diuretics have many side effects such as electrolyte disturbance and potassium loss. Most importantly they also cause dehydration and decrease blood pressure, thus affecting the blood consistency and the flow of blood, making one very prone to clotting.

Creatinine is used as a supplement to help release muscle energy. There is no evidence that it enhances performance in aerobic or endurance sports. There is likely an indirect relationship of creatinine to thrombosis – case reports have been documented and it is thought this is most likely due to dehydration (fluid is drawn into the muscles).

Stimulants such as caffeine and amphetamines may have an indirect relationship to thrombosis in athletes through dehydration and abnormalities in heart rhythm as well as blood pressure. Stimulants have been associated with heart attack and stroke as well.

What is the treatment for DVT?

Blood thinners need to be given for all patients. The medications are chosen individually but include heparin, warfarin and the new oral anticoagulants. The problem for athletes is that once on blood thinners there is an increased risk of bleeding (with the concern being brain bleeding), and high-impact sports need to be stopped. Should one present with pelvic thrombosis (i.e. calf and thigh swelling) one would benefit from surgical intervention, as described above, in order to remove the clot burden and reduce the risk of PTS.

Thus it can be seen that athletes are at an increased risk of thrombosis from a physiological point of view, although the risk is low. Medications can definitely enhance this risk. Some athletes will have congenital predisposing risk factors and this should not be forgotten. Thrombosis in athletes is likely a combination of factors. Awareness is *very* important – one should keep hydrated, avoid drugs, seek medical help for limb pain and swelling, and a key for any concern is to seek early intervention.

THE DOS AND DON'TS

DO:
- Keep hydrated
- Seek medical attention early for pain or swelling in the limbs
- Seek immediate medical attention for any shortness of breath
- Train wisely, use correct foot gear to prevent injury swelling

DON'T:
- Take performance-enhancing drugs
- Train when injured, allow the body to heal
- Push through symptoms of pain, swelling or shortness of breath

IT'LL NEVER HAPPEN TO ME!

These triathletes all survived DVT. Here's what *did* happen to them.

SHAUN DIAMOND, Project Manager

"I woke one morning unable to put my left foot on the ground, with a sharp pain across the bottom of my left foot, very similar to Plantar Fasciitis, but with no sign of abating. On a scale of one to 10, the pain was a nine. Long story short, I had a DVT in a vein in my foot. Thanks to a very clued up physio for not treating, and sending me for an ultrasound."

Dr Redman: "A DVT of the foot is extremely unusual. However, in principle, the foot has veins with blood flowing and if Virchow's triad is altered, one may be predisposed to thrombosis. Sean likely had micro trauma to his foot, either directly from his sport (the pedal on the bicycle) or possibly footwear that caused some irritation and damage to the vein wall lining. Sean was also found to have a mild genetic mutation of one of the clotting factors. This may lead to a slight increase in DVT, however, this particular type of mutation is extremely common (with about 20% of the population). Thus, how much it contributed is uncertain. Coagulation defects and deficiencies are controversial. There are certain ones that definitely predispose to increased thrombosis risk, and there are others that wouldn't change any of our management if we detected them. There are many we cannot test for, thus we look at all the contributing factors."

BRITT HYLAND, Swim Coach

"With one week to go to the Double Century, my left leg had lost power and doubled in size. Severe lower back pain led me to believe that the swollen leg was related to the back pain – I was pretty sure that anti-inflammatories would get me through race day. Two days later I went for an ultrasound which revealed a blood clot from my knee to my belly button. I would never have survived the Double Century."

Dr Redman: "Britt had what we would classically label as 'May Thurners Syndrome' – compression of her left iliac vein in the pelvis from the right iliac artery as it crosses over the vein, and the spine which is behind the vein. This is an anatomical anomaly. Sport may have contributed, by either changing pelvic structure (from muscle bulk) and aggravating compression, or she may have chronically had the narrowing, and an additional factor such as dehydration caused the already altered blood flow to thrombose. If the narrowing was quite severe before, Britt would have had what we call 'venous claudication' – when she exercised, blood couldn't leave the leg and it would become painful and swollen (calf and thigh), finding relief with leg elevation and rest. When Britt got the thrombosis it was extensive and what we call 'Upper Segment DVT'. This requires more than medical therapy and surgery is needed to remove ('suck out') some of the thrombus load. This decreases the chance of future leg problems from damaged valves. In order to prevent it happening again, a stent was inserted through the iliac vein narrowing to open it up. She would need to be on anticoagulation for three months and take anticoagulation whenever there is a risk of thrombosis such as long-haul flight or surgery."

CANDICE MORRISON, Gynaecologist

"I had a pulmonary embolism in 2012, three days after 70.3 East London. I was working a 24-hour shift in labour ward when I started experiencing severe chest pain, I put it down to muscle aches from the race and even went to physio, but the next day the symptoms progressed and I became short of breath. A CT scan confirmed I had blood clots in my lungs. Further imaging showed I had multiple clots in my iliac vein up to the vena cava. I had a stent inserted into my iliac vein and a filter to prevent further clots moving from the clots to my lungs. Since then I had a second DVT and I've been on blood thinners. Looking back, there were warning signs that I ignored for months. I'd had a heaviness in my left leg since Double Century, and I was tired and struggling to keep up with the group I usually cycled with. During EL I struggled on the run, walking the hills, and I was surprised because I'd put in a lot more training than in previous years. I think my clots were a combination of a genetic predisposition, having a factor V Leiden mutation, and being on the birth control pill, but the dehydration during half IRONMAN and long hours of cycling also played a role. I made a complete recovery and I've done a full IRONMAN, two 70.3s and DC since then, but I'm very aware of staying hydrated and taking my blood thinner regularly. I'm now more careful about who I prescribe the contraceptive pill to – in athletes with any risk factors for blood clots I encourage them to rather have a mirena inserted or to use non-hormonal methods of contraception."

Dr Redman: "Candice likely had a similar anatomical picture as Britt with the iliac vein compression. Although aggravating factors caused this thrombosis, and she is correct – the genetic predisposition, oral contraceptive and dehydration are a combination bound to cause a clot on top of an anatomical anomaly – she would be predisposed to thrombosis even without iliac vein compression. Candice may not have ever been aware she had the compression of the iliac vein, if it weren't for the other factors, unless she was experiencing venous claudication (pain and swelling when exercising). Most importantly, she complicated with the ultimate fear – pulmonary embolus. This is the reason we treat DVT as an emergency, as although it may cause mild to moderate symptoms, it can be fatal as well if a large piece of clot lodges in the lungs."

Candice mentioned doing 24-hour medical shifts. It would be important to know what is going on with her more superficial (or saphenous) veins as this may aggravate deep vein issues, and she would benefit from class two compression especially when standing for long periods."

ANDREW VAN DEN HEEVER, Director, Cape Radiology

I had a work trip to the UK and was concerned it would interfere with my training for 70.3 Durban. In 2015 I'd managed a time of 05:09, and was wanting to go under five in 2016, but the swim was cancelled and the rest didn't go too well. This year I was well prepared to give it a go.

In London, despite feeling metabolically off, I ran a hard 20km the first day, did a weights session in the hotel gym the second day, and on my last day ran 10km. I felt weirdly sore but put it down to the training, flying, working and travelling. Before my flight home I spent about four hours in the airport lounge reading. My seat on the plane was in the middle of the middle four seats, next to a rather large woman and near the bassinettes with a few unhappy infants. My solution to this nightmare: 15mg of oral Midazolam. I took my sleeping tablet right away and the next thing I knew I was being woken up for breakfast as we approached Cape Town. What a bonus!

As a trained athlete I had a resting heart rate in the 40s. I had pro-inflammatory micro trauma to my leg muscles from running, I was dehydrated from the long flight and the protracted session in the airport, and I'd sedated myself enough to be immobile for 10 hours. A perfect storm of haematological coagulopathy. About 10 days later, just over a week out from the race, I decided to do a short but intense ride to keep the neuromuscular stimulus of my taper intact. I had normal power and could push hard, but I was disproportionately out of breath. This happened every time I pushed hard and was exacerbated by any incline. I brushed it off as a "bad day". At the coffee stop just past half way I started to get chest pain – a sharp pain worsened by any deep breaths. When I got home I took two puffs of a bronchodilator inhaler but the pain didn't abate. That night I could only lie on my right side, the side of the pain. Anything else was unbearable. The penny still didn't drop.

The next day at work I X-rayed myself and saw a patch of collapse (atelectasis) in the base of my lung, but nothing else. This can occur with chest pain alone due to the reflex inhibition of chest wall expansion in response to pain. It can, however, also be a marker of a pulmonary embolism. This now crossed my mind as a possibility. Around 11pm that night, when I still couldn't sleep, my wife Natalie said, "We're going for a CT scan!" My scan showed multiple bilateral pulmonary emboli. Ten minutes later I was in ICU, and 70.3 Durban was history.

I'm now seven months out, and off all medication. I continued to run, use a stationary bike and gym, and I've just started riding again. I feel 100% and have suffered no permanent ill effects after my near-death experience. If you have one whilst riding, like I did, your chances of a horrible outcome are higher. I got lucky, my wife saved my life (thanks babe!). As trained athletes we are healthy, but slow heart rates, muscle damage and dehydration put us at an increased risk for DVT and PE.