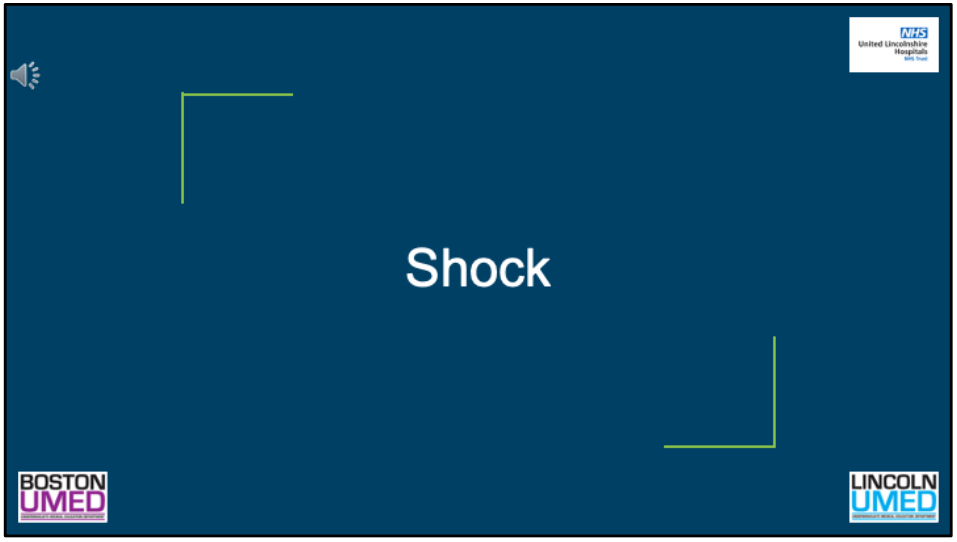
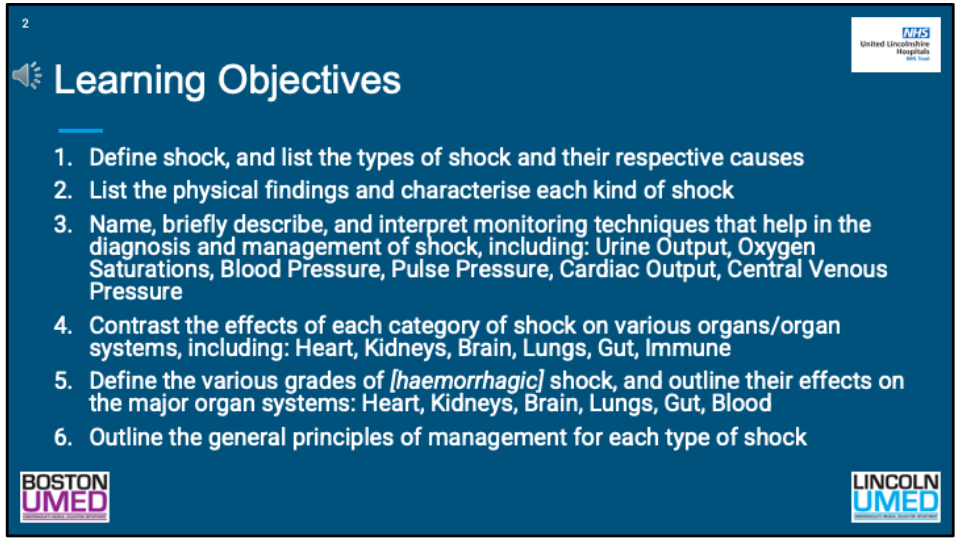


Your notes:

Hello, and welcome to your session on Shock. My name is Benjamin Gerrans, and I'm one of the clinical teaching fellows working at Lincoln and Boston. You can either watch the video for the session, or work through the powerpoint file, which has the narrations on the slide, and transcript in the notes.
Either way, I hope you find this session useful. This is a slightly reworked version of the session I produced for the CP1 online course, expanded to cover the additional objectives at a CP3 level. So if you've looked at that course, this will be quite familiar to you.



Your notes:

In this session, we will be covering the following learning objectives

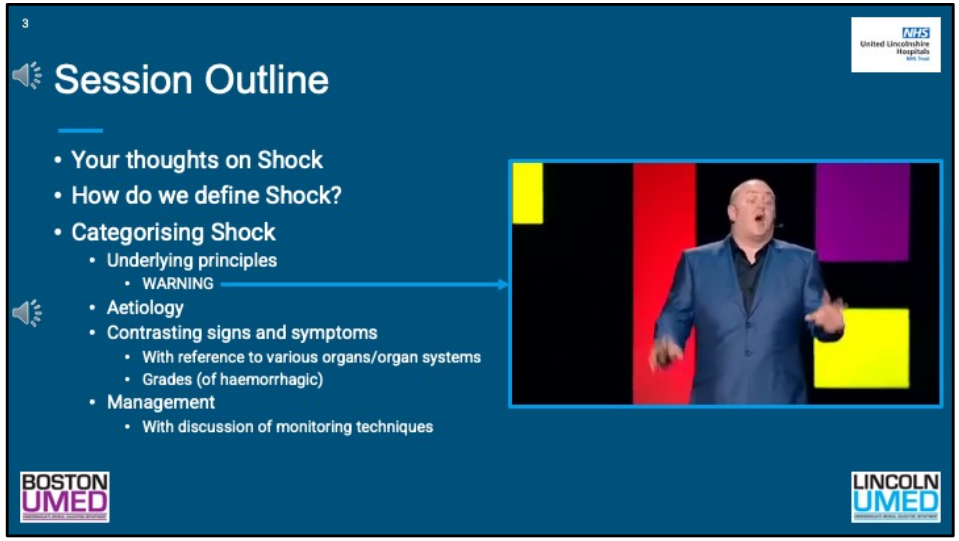
The first is that we should be able to define shock, and list the types of shock and their causes

The second is that we should be able to list the physical findings and characteristics of each kind of shock

Next, we should be able to name, describe, and interpret some monitoring techniques that help in our diagnosis and our management of shock

We should be able to contrast the effects of each type of shock, and differentiate between the effects of the various grades of shock, on the major organ systems

Finally, we should be able to outline the general principles of management for each type of shock.



Your notes:

So, thinking about how we're going to cover those objectives:

First of all, I'm going to ask you to have a think about what you already know about shock.

Then, I'll show you some of the comments or thoughts that I've had from students that I've previously taught on this topic, and we can see whether your thoughts are aligned with theirs.

Next, we'll discuss a definition of shock, and I'll do that with reference to those previous students' thoughts

After that, we'll talk about how we divide the concept of shock into various categories and sub-categories

In order to do this, we'll run through some of the underlying principles. Now at this point, there should be a fair warning on the upcoming content – and for this, I'll hand you over to Dara O'Briain…

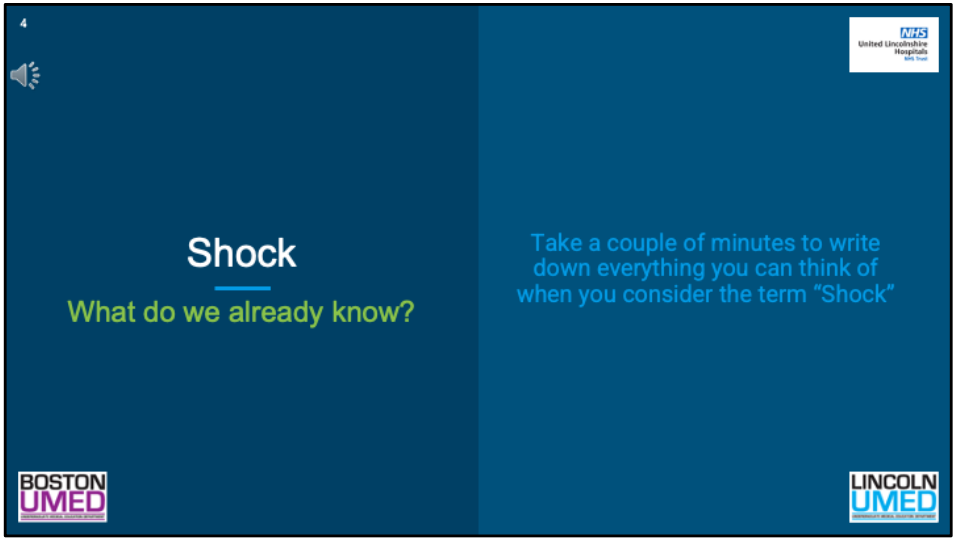
video plays

I couldn't have said it better myself, Dara…

Having discussed the underlying principles and outlined the various categories, we will discuss the aetiology and the causes of each of these with reference to the underlying principles previously covered.

We'll also outline how they differ in their presentation in terms of signs and symptoms, with particular reference to the various organs and organ systems; and also how, in haemorrhage, the patient's presentation can be used to judge the Grade, or severity, of shock.

And finally, we'll discuss the management of the various types of shock, and the monitoring that we use.



Your notes:

With that in mind, the first thing we'll do is to have a think about what we already know about shock.

I'm going to give you about 2 minutes here to have a think about that, during which time you should note down your thoughts.

To give you a timing guide, you'll see a green bar running across the bottom of the slide – the slide will move on when it reaches the right hand side.

On the next slide, I'll give you some examples of what students have previously said, so that you can compare your responses with theirs. Then we'll use those comments to put together a definition of shock.

Your notes:

I hope that's given you enough time to put down your thoughts.

Now compare what you've produced with what some of my previous students have suggested.

For lots of students, Blood Pressure, or Circulation come to mind when they think of Shock.

In a similar vein, some students mention perfusion and oxygen. Exploring this further, they sometimes talk specifically about oxygen delivery.

Sometimes, students talk about it being to do with organs, or even to do with tissues.

Another route of discussion tends to be the significance of Shock – people talk about it being a Failure, or that it is Life-Threatening.

Students also talk about the signs or symptoms of shock – common answers here include reduced Urine Output, prolonged Capillary Refill Time, or altered Consciousness.

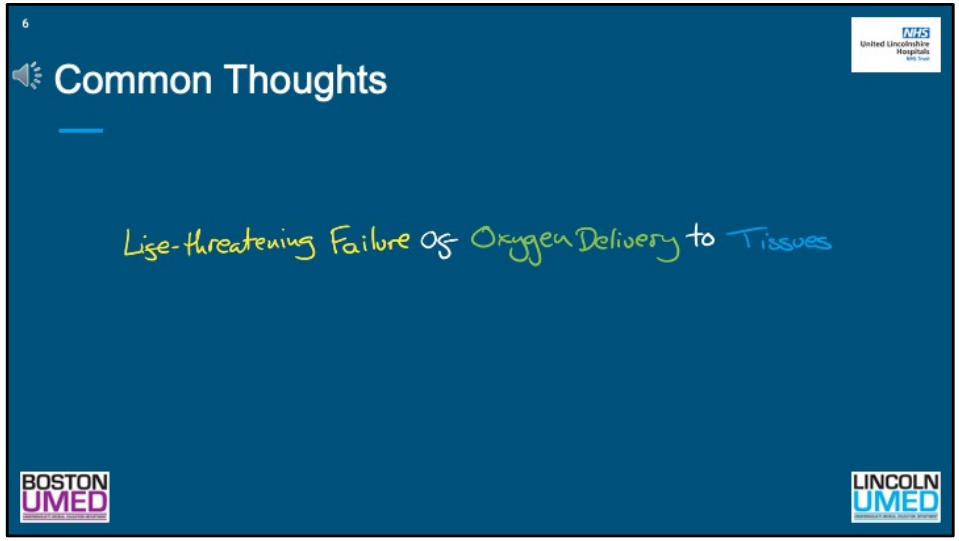
Occasionally, students talk about some causes of Shock – most commonly, they mention Blood Loss, or Sepsis.

Some comment that we look at the Lactate – can you think why this may be? We'll discuss this in a little more detail later on.

Finally, a large number of students talk about needing to give fluids. Again, we'll discuss this in more detail later on.

All of these comments are relevant to shock, although this forms by no means an exhaustive list, so any other things you've thought of may well be equally as valid as any of the comments included on this slide. How exactly each of these comments is relevant to shock should become clear through the course of this session.

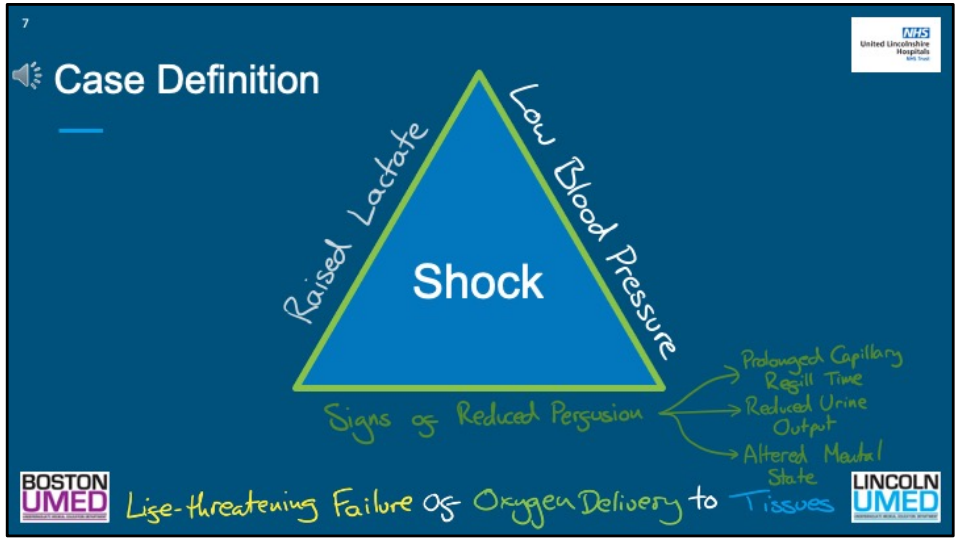
However, which of these comments do you think are the most important in constructing a definition of Shock? I'll give you about a minute to have a think about that and come up with a definition of your own.



Your notes:

So, what did you come up with as a definition of Shock?
As ever in medicine, a number of different definitions have been proposed. Personally, I use the following definition:

"The life-threatening failure of oxygen delivery to tissues"



Your notes:

Whilst keeping that definition in mind, we'll now discuss how this can be used to develop some criteria for identifying Shock.

The case definition for Shock consists of a triad of signs.

The first of these occurs due to tissue oxygen starvation. Normally, tissues respire aerobically, however when starved of oxygen they have to respire anaerobically. The process of anaerobic respiration has a by-product, which builds up in the blood. What is this?

Anaerobic respiration produces lactate, which accumulates in the blood, causing a hyperlactataemia, or a raised lactate.

Now oxygen delivery to tissues is reliant on perfusion, and perfusion is reliant on the circulation, and in particular on Blood pressure. A reduced blood pressure is the second part of this triad.

The third part of the triad is evidence, or signs, of that reduced perfusion. We look for this in three organ systems where reduced perfusion has a clear and easily visible clinical manifestation. Can you think of which organ systems they are?

Those organ systems are:

- The Skin – where hypoperfusion manifests as a prolonged capillary refill time
- The Kidneys – where it manifests as a reduced urine output level
- The Brain – where we may see an altered mental state, such as confusion, or reduced consciousness

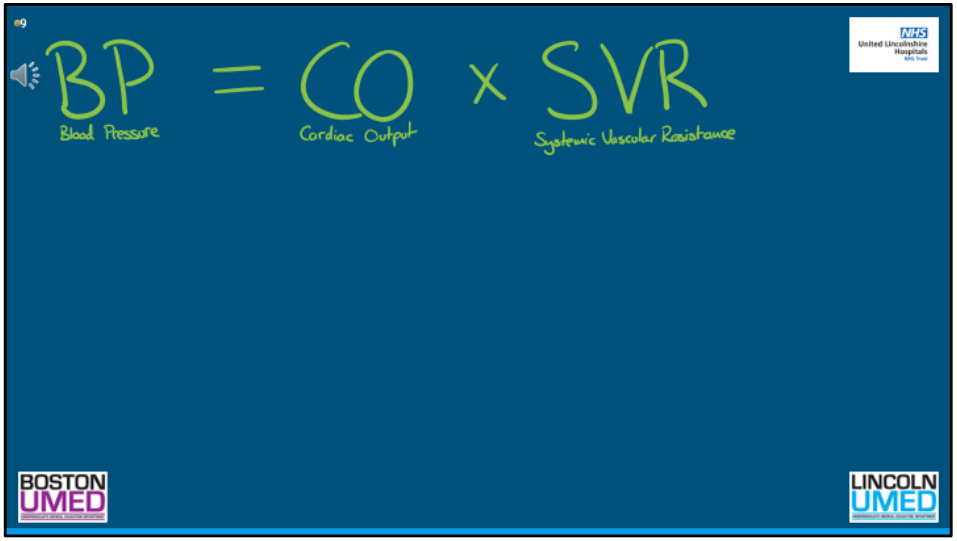
Now it should be noted that Shock isn't in itself a diagnosis, but is merely a pathophysiological state, which has a number of underlying causes.

Your notes:

Having outlined our definition of shock as a life-threatening failure of oxygen delivery to tissues, we then discussed how this oxygen delivery is reliant on perfusion, which is in turn primarily determined by Blood Pressure. As such it is clear that Blood Pressure is of critical concern in Shock.

So what do you think determines a patient's blood pressure?

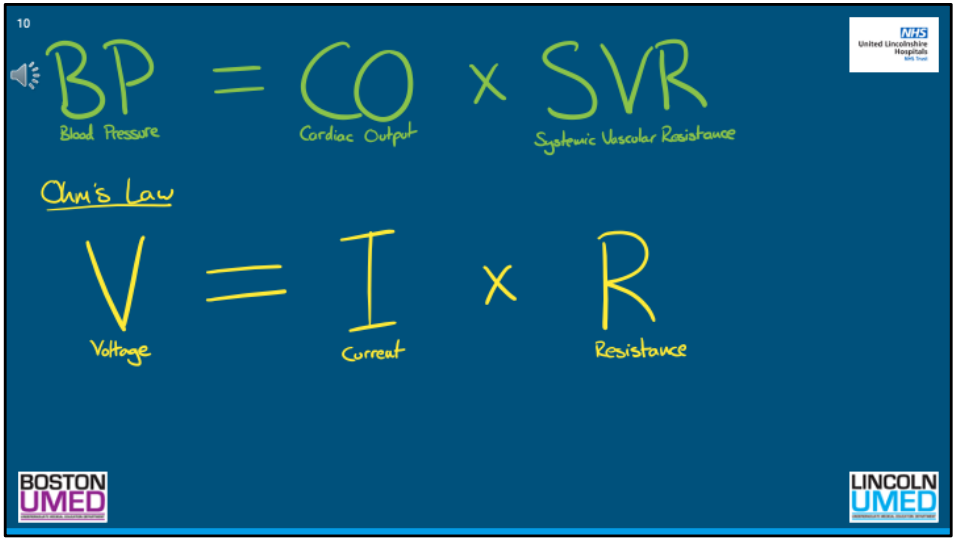
I'll give you a minute to have a think about it.



Your notes:

So what were you able to come up with?

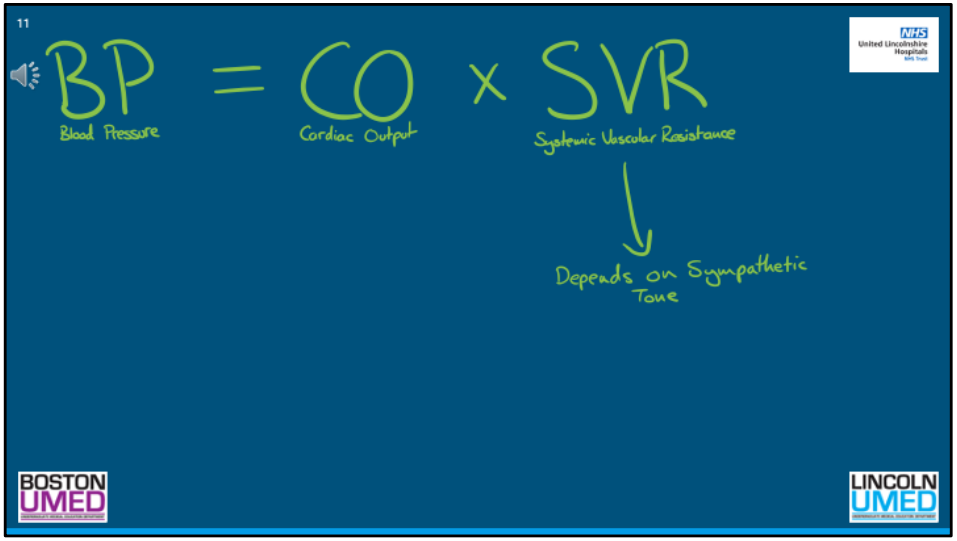
Blood Pressure is a factor of the Cardiac Output, and the Systemic Vascular Resistance.
If you think back to secondary school physics, this equation has a parallel in electronics, where it is the mathematical representation of a Law. Can you think what this Law is?



Your notes:

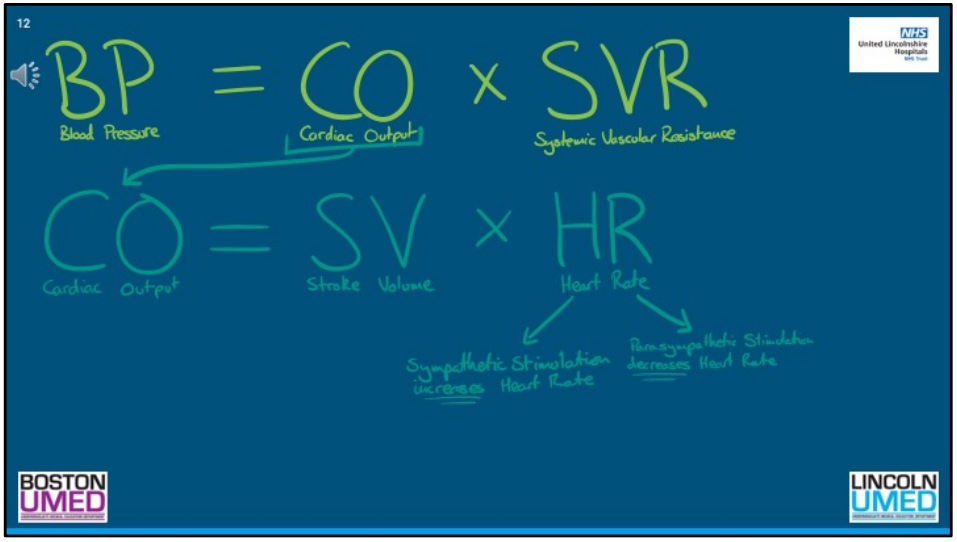
This is Ohm's Law, which in electronics governs the relationship between Current, Resistance, and Voltage.

In our circulatory system, the Blood Pressure is like the Voltage (V), the Cardiac Output is equivalent to the Current (I), and the Systemic Vascular Resistance is equivalent to the resistance in a circuit (R). All these, put together, form the equation V = IR, or Voltage equals Current times Resistance. The heart, working as a pump, is like the battery in this circuit.



Your notes:

Your Systemic Vascular Resistance is dependant on regulation by the autonomic nervous system, via sympathetic tone. Sympathetic stimulation triggers vasoconstriction, and therefore an increase in systemic vascular resistance.



Your notes:

Your cardiac output can be subdivided further.

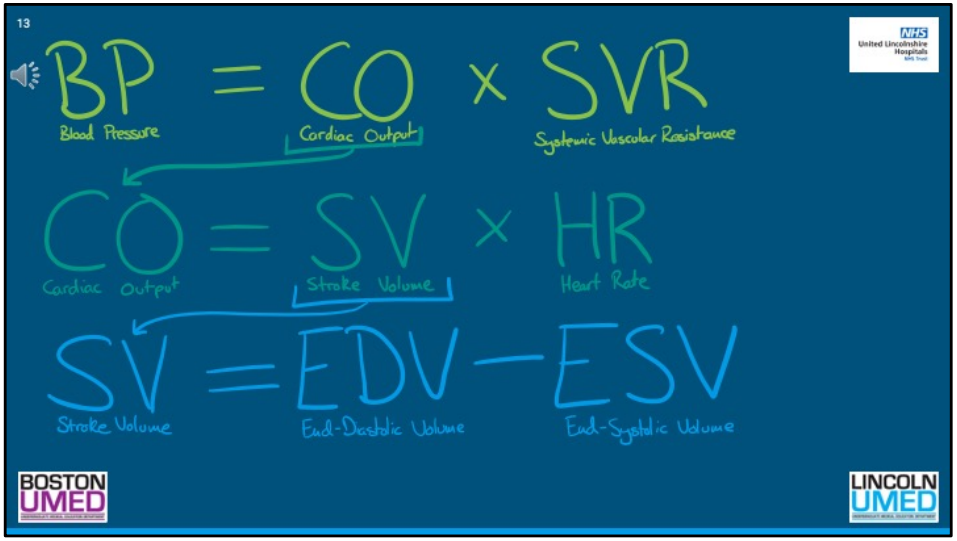
Cardiac output is the total volume of blood that is pumped out of the heart in one minute. Can you think of another thing related to the heart that we measure by the minute?

The answer I'm looking for here is Heart Rate, which is the total number of beats the heart makes in a minute.

Now, if you take the total volume of blood that the heart pumps out in a minute, and divide it by the number of heart beats in a minute, you get the average volume of each individual beat, which is known as the Stroke Volume.

If you rearrange that equation, you can see that Cardiac Output is equal to the Stroke Volume times the Heart Rate.

Similarly to the Systemic Vascular Resistance, your Heart Rate is determined by your autonomic nervous system, with sympathetic stimulation increasing the heart rate, and parasympathetic stimulation reducing the heart rate.



Your notes:

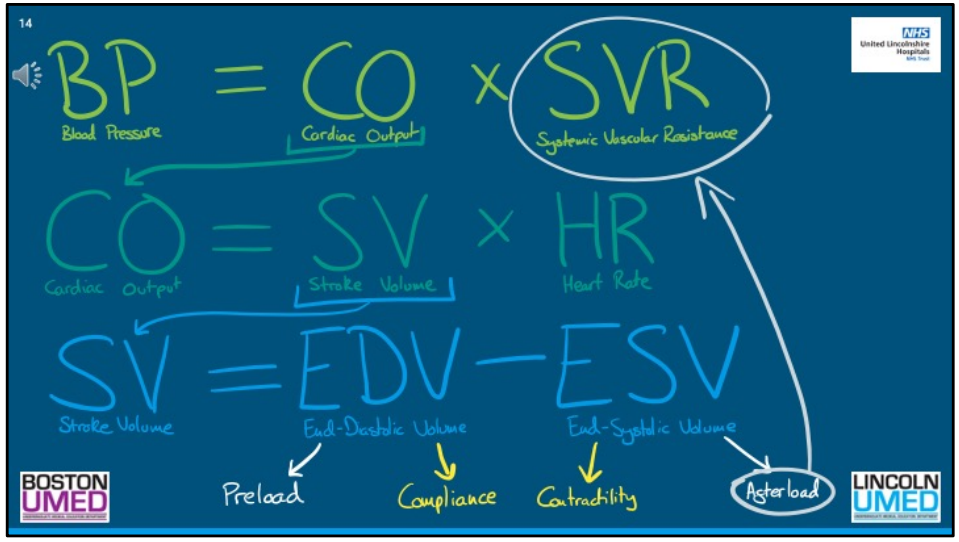
Once again, we need to look at something in greater depth – this time, the **stroke volume**.

Now, in order to think about how we **measure** the stroke volume, imagine you're trying to measure how much water you've poured from a jug down the drain. We can't measure the water we've poured away, but we can work out how much has gone by looking at two other measurements from the jug.

Before pouring, we can measure how much water is in the jug when it's full.
Then, having poured some of the water out of the jug, we can measure how much is left in it.
By **subtracting** the post-pour volume from the pre-pour volume, we find out the amount we poured away.

The same applies to the heart. We can measure the volume of the heart, and specifically the left ventricle, at different points in the cardiac cycle (by befriending someone that's actually able to perform an ECHO).
In terms of the jug analogy, the pre-pour volume in the cardiac cycle is the **End-Diastolic Volume (EDV)** – the volume at the end of filling.
Equally, the post-pour volume in the cardiac cycle is the **End-Systolic Volume (ESV)** – the volume at the end of emptying.

The **End-Diastolic Volume** is dependent on how well the heart is able to fill, whilst the **End-Systolic Volume** is dependent on how well the heart is able to empty. Each of the End-Diastolic Volume and the End-Systolic Volume are, broadly, determined by two factors. For each, one factor is to do with the heart muscle itself, whilst the other is external to the heart. Can you name the factors that influence filling and emptying of the heart? I'll give you about a minute to think about **this.**



Your notes:

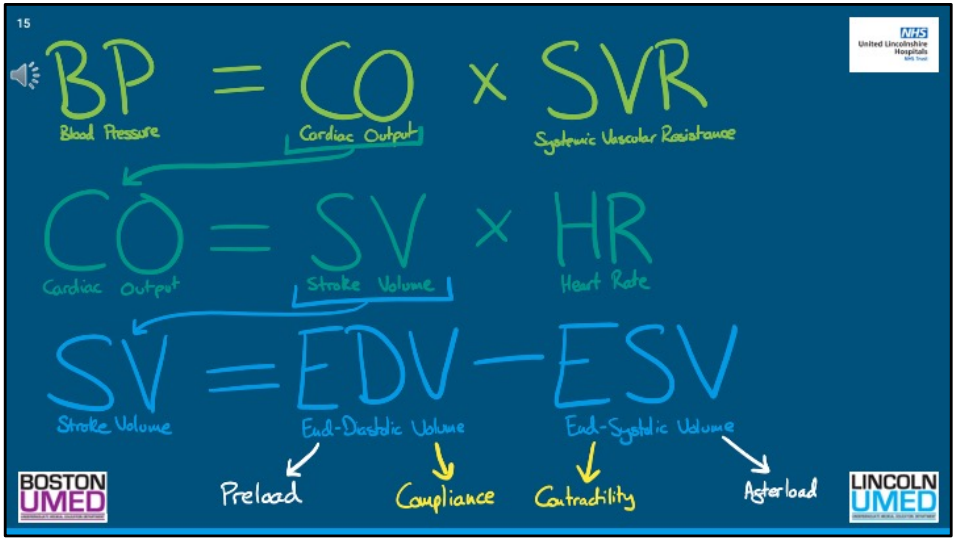
The factors that influence filling, and therefore End-Diastolic Volume, are: Externally, Preload; and Internally, Compliance.

- There are a number of definitions and conceptualisations of Preload, but it can be viewed most simply as the "filling pressure" – often this is dependent on the patient's fluid status, being reduced if the patient is dry)
- Compliance describes the stretchiness, or relaxation, of the heart muscle

You can conceptualise this as like trying to blow up a balloon. The preload is how hard you are blowing, whereas the compliance is how stretchy the balloon is. Hopefully your heart is more like a party balloon than the modelling balloons that clowns use, which need a pump to inflate!

The factors that influence emptying, and therefore End-Systolic Volume, are: Internally, Contractility; and Externally, Afterload.

- Contractility, describes the force of contraction of the heart muscle
- Afterload, refers to what the heart is pushing against
    - This is, in essence, the same as Systemic Vascular Resistance (SVR), however I keep these conceptually separate, as disorders of systemic vascular resistance leading to shock are due to a lack of resistance, whereas disorders of afterload leading to shock are due to there being too much resistance. There's a Goldilocks zone there, somewhere in the middle.



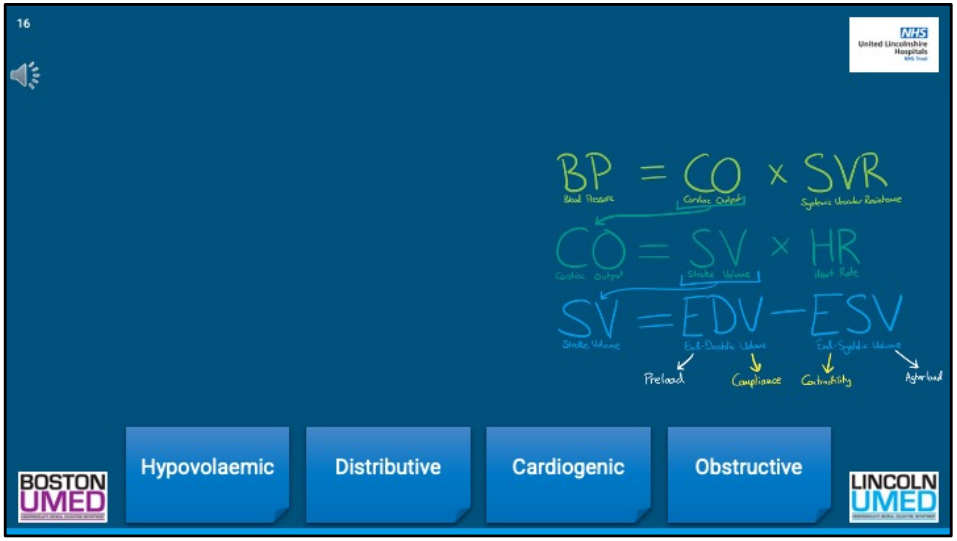
Your notes:

So, we have seen that Blood Pressure is dependent on a number of factors, from the resistance in blood vessels which is determined by the sympathetic tone, to the heart rate and the various factors that affect our stroke volume, which together determine our cardiac output.

This means that there are many areas where something can go wrong, but also for each of those a number of ways the body can compensate for (or try to correct) this problem. For instance, an abnormality of cardiac output can be compensated for by vasoconstricting, which increases systemic vascular resistance. Whereas, if there is an abnormality of Systemic Vascular Resistance, the body might attempt to raise the cardiac output to compensate by increasing the heart rate.

As a result, each abnormality is likely to display different signs and also require different management.

This leads us on to talk about the categories of shock. What categories of shock are you able to name? Once again, I'll give you about a minute to write down as many as you can come up with.



Your notes:

This is another area where you'll hear as many different answers and schemes as there are doctors in the world, so when you hear someone use a different classification to me, don't be surprised. However, there is still a lot of commonality between different people's conceptualisations of these categories – they're just different ways of representing the same information.

Personally, I divide shock into four main categories:

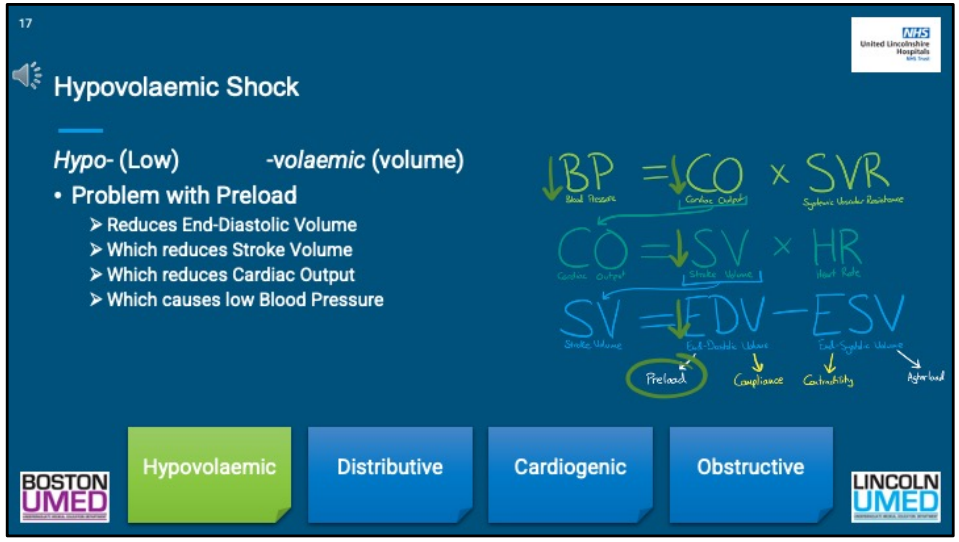
- Hypovolaemic
- Distributive
- Cardiogenic
- Obstructive

I then place other commonly-mentioned types of shock as sub-types of these four main categories. Those sub-categories include:

- Haemorrhagic
- Septic
- Anaphylactic
- Neurogenic

We'll discuss later just how these subcategories fit into the picture, but for now we'll look at each of the four main types in turn.

First, let's look at Hypovolaemic shock

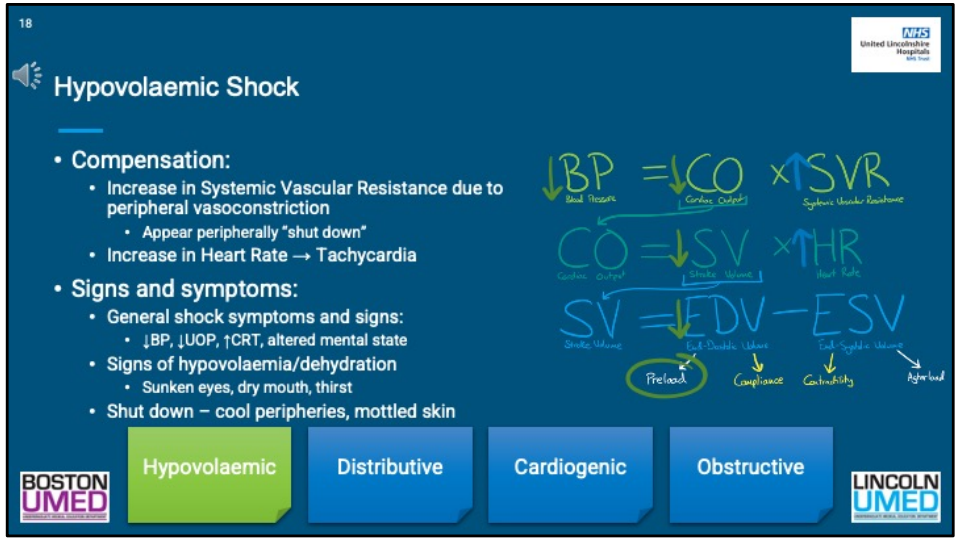


Your notes:

Hypovolaemic shock occurs when there is an insufficient volume of blood in the intravascular compartment.

In the equations on the right, where does this cause problems, and how does it cause a low blood pressure?

A low intravascular volume reduces the preload, or filling pressure, in the heart. This, in turn, reduces your End-Diastolic Volume (EDV), which reduces your Stroke Volume (SV), which reduces your Cardiac Output, and thus causes a reduction in blood pressure.

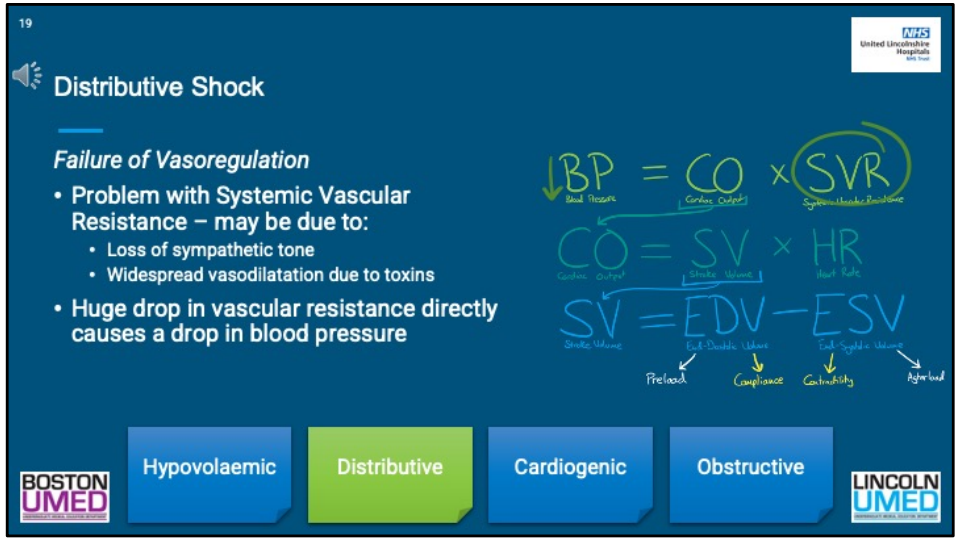


Your notes:

In Hypovolaemic shock, the body attempts to compensate for this low blood pressure.
It does this in a number of ways:

- Compensates for decreased cardiac output with peripheral vasoconstriction, which increases the systemic vascular resistance.
  - This vasoconstriction is targeted to the least critical organs, such as the skin, the gut, and the kidneys in order to maintain perfusion of the vital organs such as the heart, lungs, and brain.
- Compensates for a reduced Stroke Volume by increasing the Heart Rate, producing a tachycardia
- There may also be an increase in the heart's contractility and compliance, in order to optimise the stroke volume, although this is harder to observe.

In terms of signs and symptoms, we will have the general symptoms of shock, as outlined in the triad earlier on, with Hypotension, and signs of reduced perfusion.
We also need to perform a fluid status assessment, which will show signs of hypovolemia or dehydration such as sunken eyes, a dry mouth and thirst. You might also assess the Jugular Venous Pressure and skin turgor.
As a result of the patient's compensation, you are likely to find the patient is tachycardic. It's important to manually palpate the pulse: not only because this gives further information such as the character, volume, and regularity of the pulse; but also because the readouts of heart rate on monitoring are incredibly unreliable, particularly where the patient's heart rate is irregular.
You are also likely to find signs of peripheral "shut down", where the patient's peripheries are cool, and their skin mottled, as a result of the peripheral vasoconstriction that occurs.

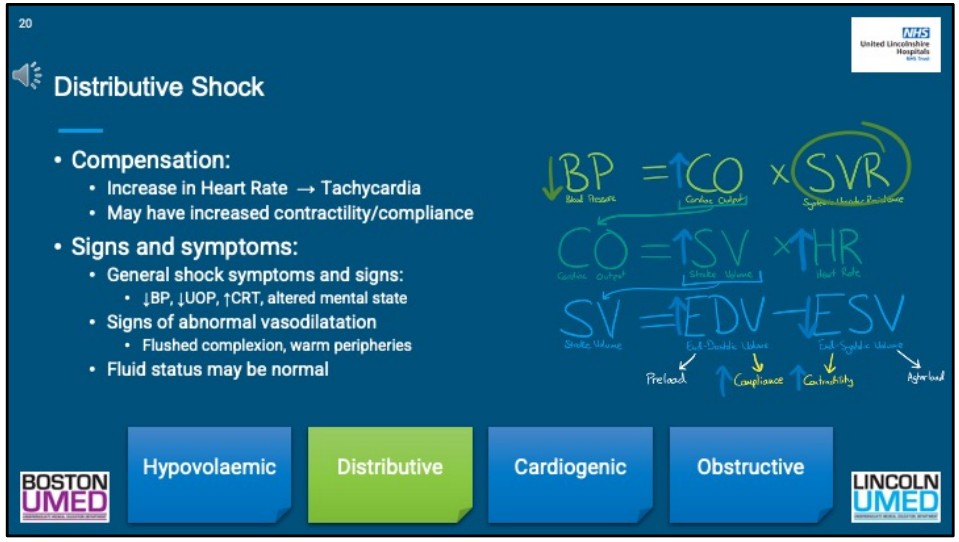


Your notes:

Distributive shock occurs when there is an abnormal, widespread vasodilatation, and can also be described as a failure of Vasoregulation.

In the equations on the right, where does this cause problems, and how does it cause a low blood pressure?
This causes a drop in systemic vascular resistance, which causes blood pressure to fall

Based on what we've discussed previously, can you think why this might happen?
As systemic vascular resistance depends on sympathetic tone, anything that causes a loss of sympathetic tone can cause Distributive Shock.
Certain toxins can also cause this widespread vasodilatation.



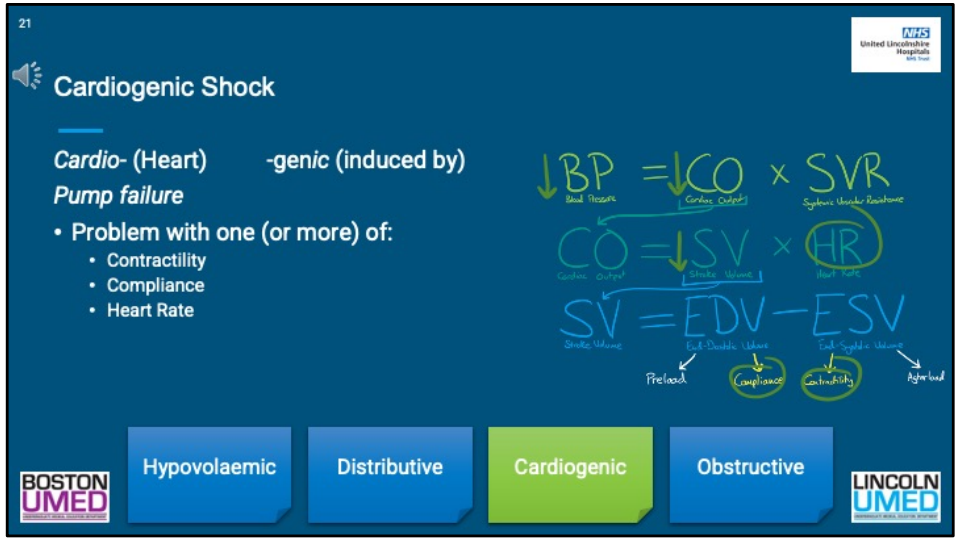
Your notes:

In Distributive shock, the body usually attempts to compensate for the lack of systemic vascular resistance by increasing the heart rate, and can also increase the contractility and compliance of the heart in order to optimise the stroke volume. These actions together optimise the cardiac output, in order to make up for the lack of resistance.

In terms of signs and symptoms, again we will have the general symptoms of shock, as outlined in the triad earlier on, with Hypotension, and signs of reduced perfusion.

In addition, we will see signs of that abnormal vasodilatation, which allows blood to reach the peripheries despite the low blood pressure. We may see that the patient is flushed, looking very red, and that their peripheries are unexpectedly warm to the touch. This is in contrast with every other category of shock.

In this case, the fluid status may be normal, overloaded, or hypovolaemic. But please bear in mind that these types of shock can and do coexist, which adds a layer of complexity to their recognition and management.



Your notes:

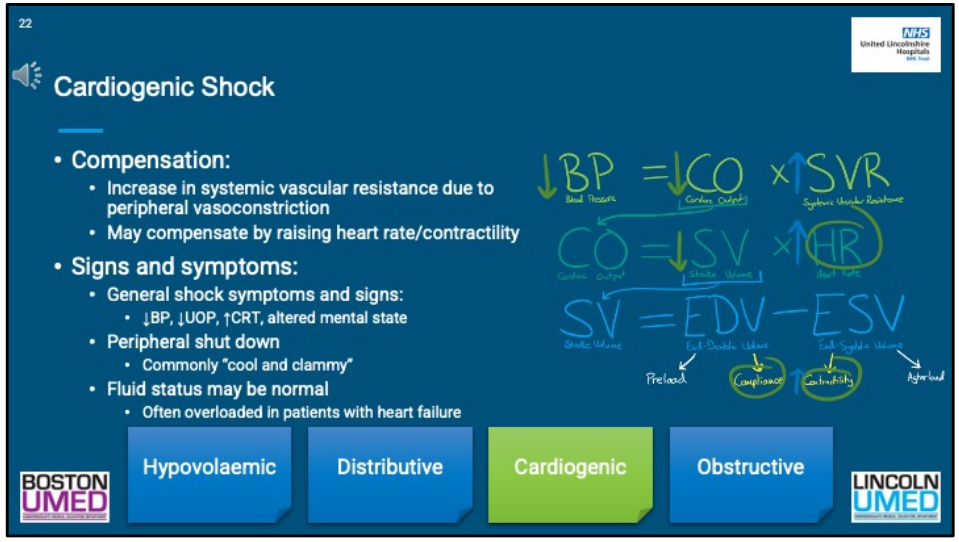
Cardiogenic shock occurs when the heart stops pumping effectively. Hence, it is also commonly called “Pump Failure”

In the equations on the right, where can this cause problems, and how does it cause a low blood pressure?

There are a number of problems that can cause pump failure.

- If this is due to a Myocardial Infarction, the muscle itself will be unable to contract with enough, or possibly any, force, causing problems with contractility. MIs can also impair myocardial relaxation, causing problems with compliance.
- Arrhythmias of the heart include bradyarrhythmias that reduce the cardiac output and tachycardias and/or arrhythmias that prevent the ventricles from filling properly.

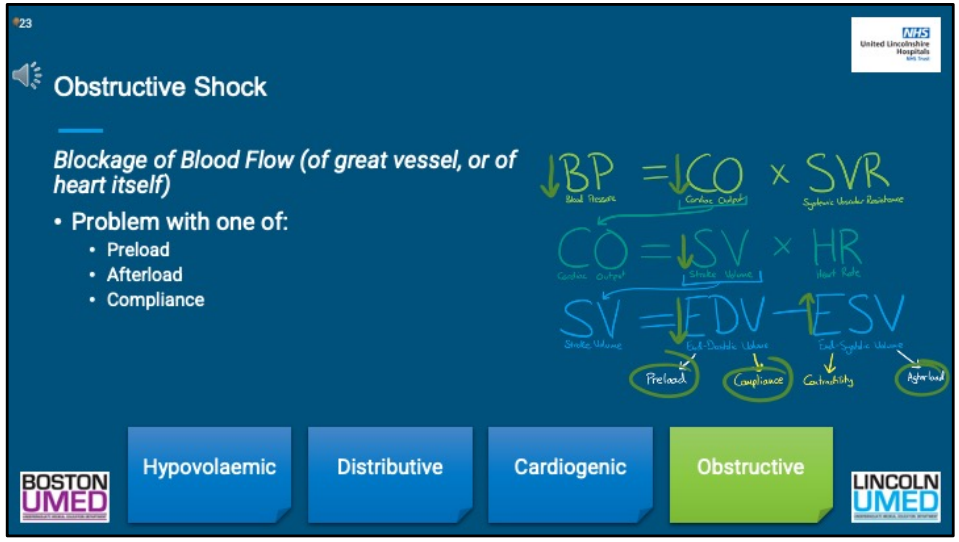
Other than the Bradyarrhythmias, which are simply an insufficient heart rate to maintain our cardiac output, each of these problems affects the stroke volume and, by extension, cardiac output and the blood pressure



Your notes:

In Cardiogenic shock, the compensation depends on the specific cause, however universal to all is likely to be a peripheral vasoconstriction, which increases the systemic vascular resistance.
If the problem lies with the compliance or contractility of the heart, then the heart rate can rise to compensate.
If the problem lies in the heart rate, there may be some compensatory increase in myocardial contractility, for instance.

In terms of signs and symptoms, we will have the general symptoms of shock, as outlined in the triad earlier on, with the Hypotension, and signs of reduced perfusion.
Commonly these patients are "cool and clammy" – a result of that peripheral vasoconstriction, which diverts blood away from the skin.
Their fluid status varies, although the patient will commonly be overloaded if they are in heart failure
They may have abnormal heart rhythms, whether tachycardic (either as the primary problem, or as a compensatory mechanism), or bradycardic.



Your notes:

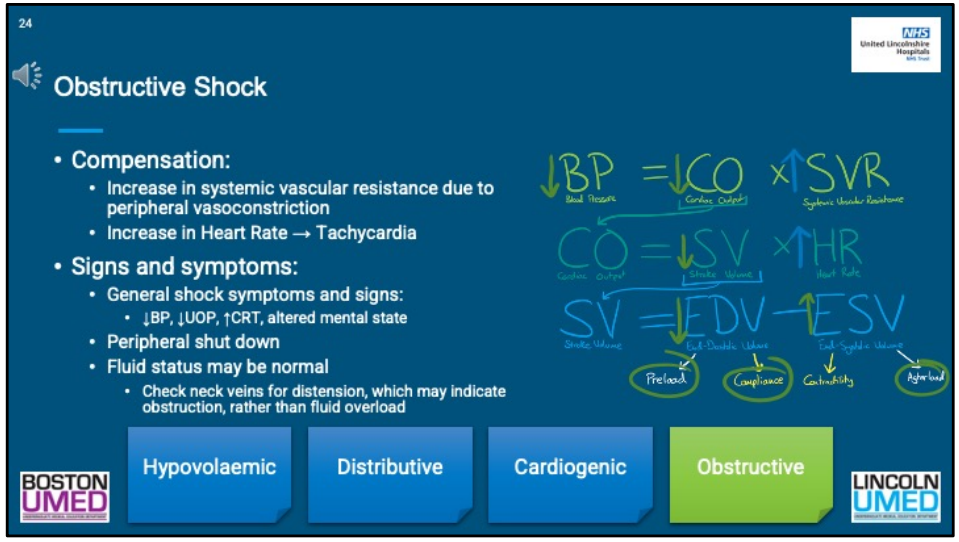
Obstructive shock occurs when there is a blockage of Blood Flow, either in one of the great vessels, or a blockage of the heart itself.

In the equations on the right, where does this cause problems, and how does it cause a low blood pressure?

These blockages can affect the Preload (vena cava/pulmonary circulation), Afterload (Aorta), or the Compliance (by compressing the heart itself). The abnormalities that can cause each of these are:

- Vena cava – compression due to mediastinal shift in Tension Pneumothorax
- Pulmonary circulation – massive pulmonary embolism can cause such severe obstruction of blood flow through the pulmonary circulation that pulmonary venous return to the left atrium is affected
- Aorta – in aortic dissection at the aortic root or arch, blood starts to be pumped directly into the dissection flap, rapidly expanding the dissection and blocking the aortic lumen, resulting in a huge increase in afterload
- Compression of the heart – in cardiac tamponade, blood accumulated in the confined space of the pericardial sac, preventing left ventricular filling, and affecting compliance – imagine trying to blow up a balloon that is in a bottle full of water!

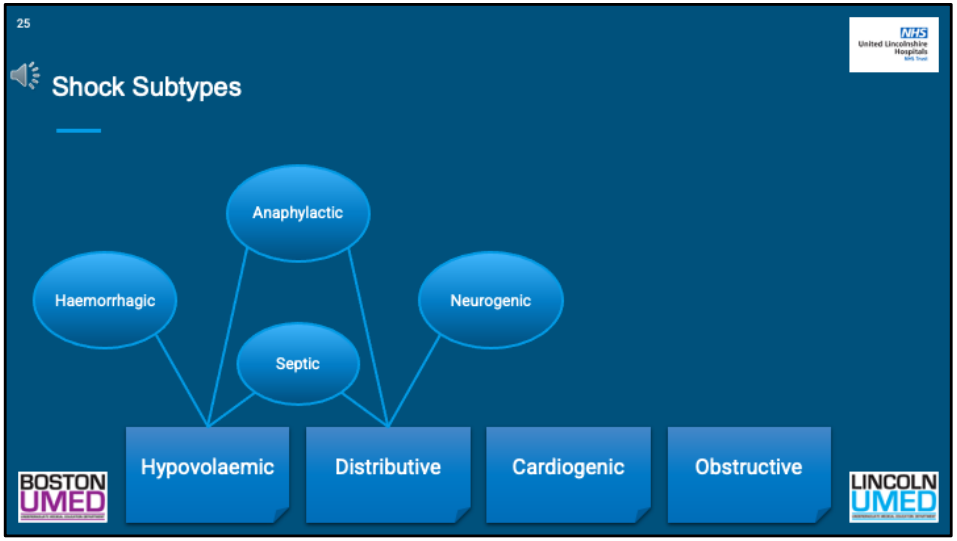
Problems with Preload and Compliance reduce the End-Diastolic Volume, whereas problems with Afterload increase the End-Systolic Volume, however in each of these cases the stroke volume is impaired, leading to a reduction in cardiac output and therefore blood pressure



Your notes:

In obstructive shock, the body will once again compensate by vasoconstricting (which increases systemic vascular resistance), and also by increasing the heart rate.

In terms of signs and symptoms, we will again have the general symptoms of shock as outlined in the triad earlier on, with Hypotension, and signs of reduced perfusion.
The patient is likely to be peripherally shut down, with cool peripheries and a mottled appearance, and also have a tachycardia. Remember – feel the pulse manually: there's a wealth of information to be had by doing so! For example, in the case of a patient with an aortic arch dissection, by feeling both radial pulses simultaneously you may be able to observe a radial-radial delay.
Again, the fluid status may be normal. It is important, in this case, to check the neck veins – yes, for the JVP – but also for a gross distension that may indicate an obstruction to blood flow causing obstructive shock (contrasting with an elevated JVP indicating fluid overload).

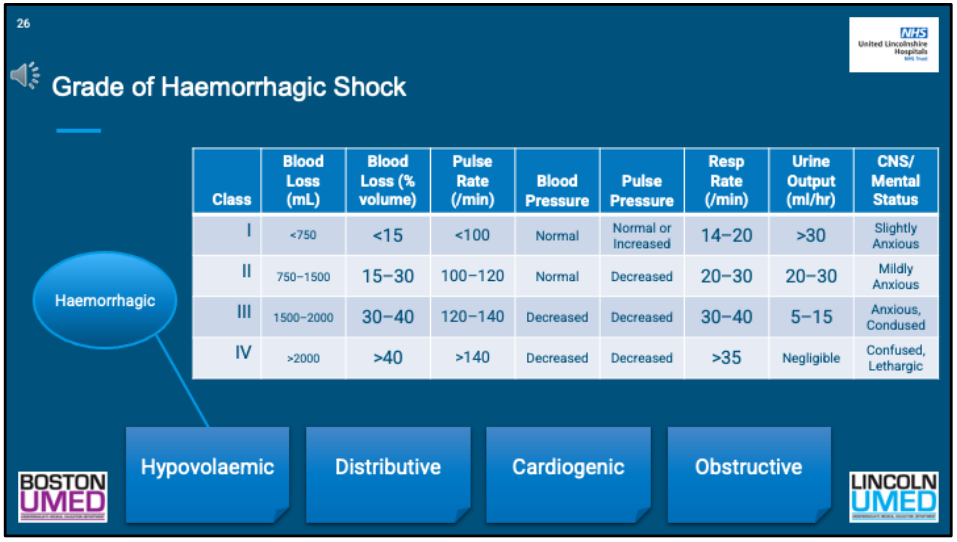


Your notes:

That rounds up my four main categories of shock. However, earlier, I mentioned the types of shock I use as subcategories – these were:

- Haemorrhagic
- Anaphylactic
- Septic
- Neurogenic

In haemorrhagic shock, we have an acute loss of intravascular volume, so this is a subcategory of Hypovolaemic shock. It is a special case within hypovolaemic shock as it is important to consider how and with what we are replacing this volume. We've lost not just the fluid component of blood, but also its contents. Particularly important are the red cells, without which we aren't able to effectively deliver oxygen to tissues, and also the platelets and clotting factors, without which we will be unable to clot and stem the bleeding. This also raises the important point in haemorrhagic shock that the absolute imperative is to stop the bleeding. There's no point turning on the taps if you're not going to put the plug in as well.

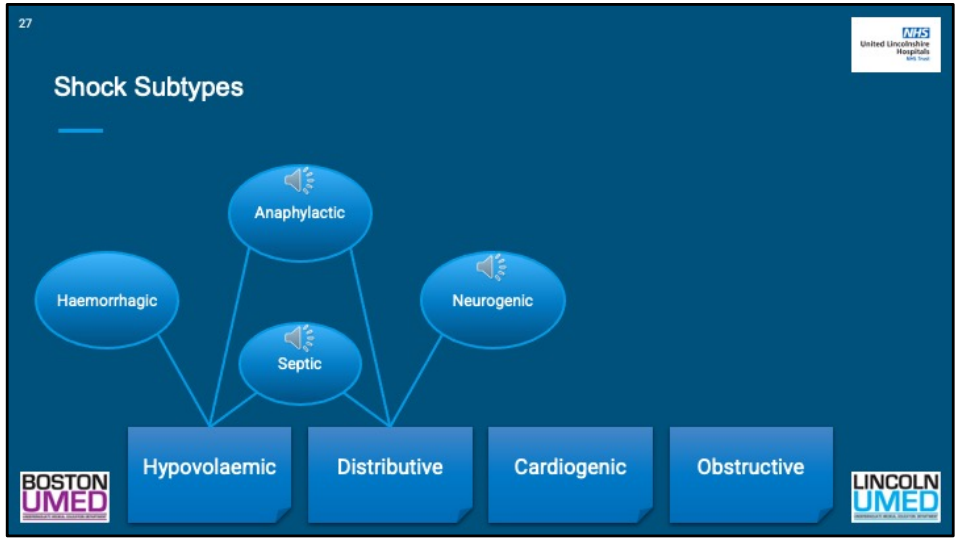


Class	Blood Loss (mL)	Blood Loss (% volume)	Pulse Rate (/min)	Blood Pressure	Pulse Pressure	Resp Rate (/min)	Urine Output (ml/hr)	CNS/ Mental Status
I	<750	<15	<100	Normal	Normal or Increased	14–20	>30	Slightly Anxious
II	750–1500	15–30	100–120	Normal	Decreased	20–30	20–30	Mildly Anxious
III	1500–2000	30–40	120–140	Decreased	Decreased	30–40	5–15	Anxious, Condused
IV	>2000	>40	>140	Decreased	Decreased	>35	Negligible	Confused, Lethargic

Your notes:

In Haemorrhagic shock, there is a relationship between the volume of blood lost and the signs and symptoms that are observed.
This allows us to divide haemorrhagic shock into four grades, or classes, each with an expected set of signs, and consistent with certain volumes lost.
The benefit of this is that it allows us to estimate the volume of blood our patient has lost based on their clinical signs and symptoms, which can direct our management.
It should be noted that this classification system is not perfect, and serves only as a guide rather than as gospel.

You'll note that as we progress through the classes of shock we see a progression in tachycardia, with this and the respiratory rate both abnormal before the blood pressure even begins to drop. This is as a result of those compensatory mechanisms that we have discussed, which aim to preserve the blood pressure and perfusion of vital organs, for as long as possible. We also see the urine output start to drop quite early on, and one of our earliest signs is an alteration in the patient's mental state



Your notes:

Anaphylactic shock is a mixed picture. The aetiology here is that widespread histamine release has a profound vasodilatory effect which causes a distributive shock. However, another effect of Histamine is that it increases the permeability of the vessels, leading to excessive loss of fluid into the interstitial space (commonly called "third-space losses"). So in these cases, it is of course of critical importance to remove the trigger in the first instance, and then administer adrenaline and antihistamines. Adrenaline acts on adrenergic receptors in the sympathetic nervous system to reverse the systemic vasodilation, acting as a vasopressor. It also decreases vessel permeability, which helps to prevent third-space fluid losses. However, fluid loss will have already occurred, and will not immediately resolve, so there is a role for correcting hypovolaemia by giving fluid boluses. Administration of steroids (usually hydrocortisone) is also important in anaphylaxis, however the theory is that this reduces the likelihood of a biphasic anaphylaxis (i.e. a second event), rather than having any role in correcting the current abnormalities.

Septic shock is also a mixed picture. In this case, bacterial endotoxins have a similar effect to histamine: that of widespread vasodilatation (causing distributive shock), and of increasing vessel permeability (causing hypovolaemia through third-space losses). Again, critical to management here is treatment of the trigger, so we administer antibiotics urgently. The mainstay of initial management in sepsis is administration of fluid boluses (to correct hypovolaemia), however in cases that are refractory (i.e. do not respond) to fluid boluses, referral to HDU or ITU is indicated to administer infusions of vasopressors in order to reverse the systemic vasodilatation.

Neurogenic shock occurs when there is an injury to the central nervous system above the T6 level. This causes a total loss of sympathetic tone whilst leaving vagal stimulation unopposed. One effect of this is profound vasodilatation causing a distributive shock. However where you might expect to see a compensatory rise in the heart rate in other forms of distributive shock, in neurogenic shock this might not occur due to the loss of sympathetic stimulation and unopposed vagal activity on the sinoatrial node. So, where you have a patient who appears to be in distributive shock, who has a paradoxical bradycardia, consider whether there has been an injury to the brain or spinal cord resulting in neurogenic shock. Management of Neurogenic shock may include a combination of any or all of:

- Fluid resuscitation is important, despite no overall loss of total body water volume, as an expansion of intravascular volume will help to fill the larger intravascular space that has resulted from the vasodilatation (correcting *relative hypovolaemia*)
- Vasopressors (to reverse systemic vasodilatation), such as vasopressin (ADH), adrenaline,

and noradrenaline amongst others
- Inotropes (to increase myocardial contractility), such as dopamine, isoprenaline, adrenaline, and noradrenaline amongst others
- Chronotropes (to correct the Bradycardia), such as atropine, adrenaline, and dopamine, again amongst others

You'll note that many of the medications I've listed come under multiple categories, as they each have a different profile of Vasopressor, Inotropic and Chronotropic effects, but most have an overlap between these effects.

Bear in mind that Neurogenic shock occurs in around half of people who suffer a spinal cord injury, and it normally takes 1-3 weeks to resolve, requiring prolonged supportive measures.

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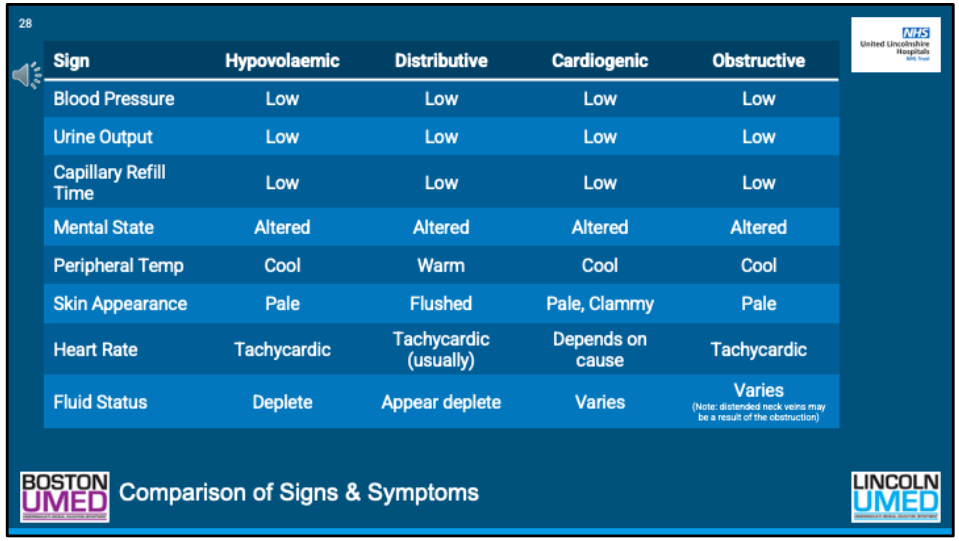
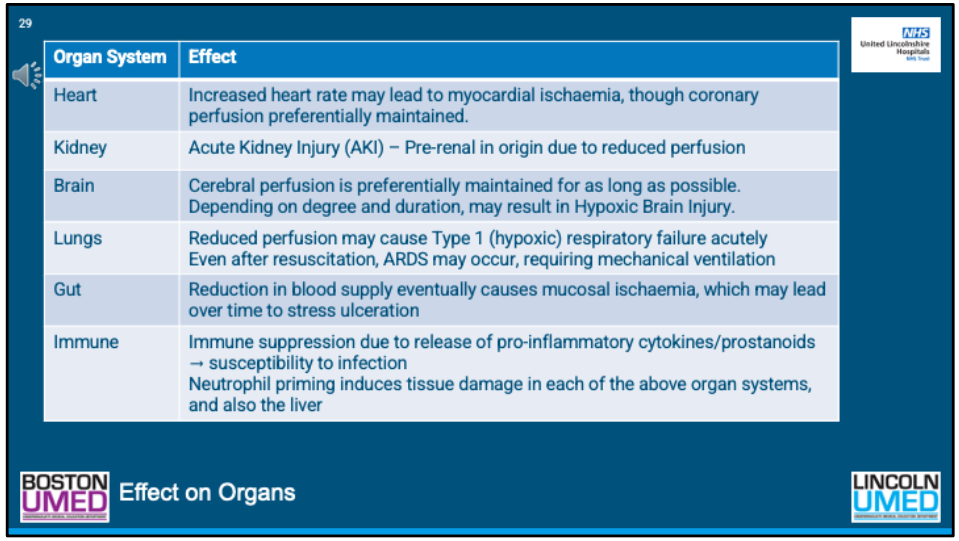


Your notes:

This slide is a table that compares the signs and symptoms you might expect to observe in each of the four broad categories of shock.
You'll very quickly notice that the top four rows are the same across all categories – these are the defining features of circulatory shock, and are therefore expected in every case. You would also expect to see a raised lactate.

Hypovolaemic shock is notable due to the signs of fluid depletion such as sunken eyes, dry mouth, thirst, invisible JVP, reduced skin turgor.
Distributive shock stands out from the others, due to the relative warmth, and flushed appearance of the skin that results from the abnormal vasodilatation. These patients may appear volume deplete either due to the coexistence of hypovolaemia, or the relative hypovolaemia due to vasodilatation. They are usually tachycardic in order to compensate, however in Neurogenic shock this may not be the case – a paradoxical bradycardia occurs.
Cardiogenic shock tends to give cool, clammy and pale peripheries. You may also note abnormal heart rhythms (including bradycardias), signs of fluid overload (such as ankle oedema or bibasal crepitations), and possibly chest pain (depending on the cause).
Obstructive shock tends also to cause cool and pale peripheries, induce tachycardia by way of compensation, and can occur in any fluid status. Here, it is important to observe the neck veins. Obstruction will result in backup of blood, which will manifest in distension of the neck veins. In these cases, consider whether any of the causes of obstructive shock may have occurred, and ensure that they are treated rapidly.

Finally, I'll reiterate that whilst we are able to clearly divide shock into these categories on paper, in patients in real life the types of shock often coexist, with one leading to another. So whilst the signs and symptoms can be clearly laid out on paper, remember that the reality is rarely so clear-cut and simple.



Your notes:

The fourth objective for this session asks for you to contrast the effect of each type of shock on each of these organ systems. However, the effects are pretty consistent across all types of shock, so I have just listed some potential effects for each.

In the case of the heart, although coronary perfusion is preferentially maintained, ischaemia can occur. Remember that coronary blood flow occurs predominantly during diastole. In shock, patients usually develop a tachycardia in order to maintain blood pressure, which will result in a reduction in the amount of time spent in diastole. This can cause a relative reduction in coronary blood flow at a time of high demand, leading to ischaemia.

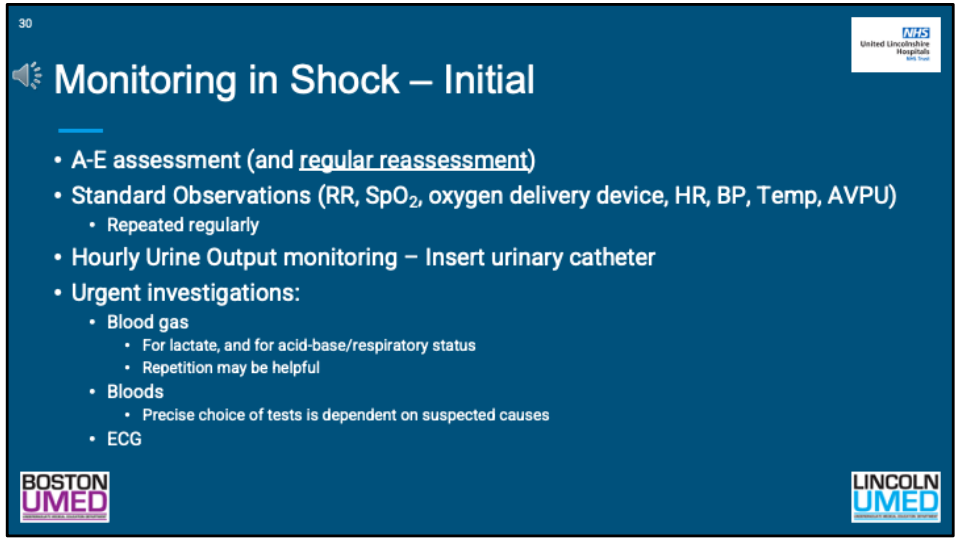
The kidneys experience a reduction in perfusion in shock. This can rapidly lead to an Acute Kidney Injury that is pre-renal in origin.

The brain, like the heart, has its perfusion preferentially maintained. However, in profound and prolonged shock, cerebral perfusion will be impaired. Remember that the brain is unable to maintain sufficient energy production through anaerobic respiration, so is highly susceptible to injury when perfusion falls. This results in Hypoxic Brain Injury.

Whilst the initial effect of poor perfusion in the lungs may be a reduction in oxygen exchange and a resultant type 1 respiratory failure, the insult of shock predisposes to subsequently developing Acute Respiratory Distress Syndrome (ARDS), which requires mechanical ventilation and is associated with short-term mortality and long-term morbidity.

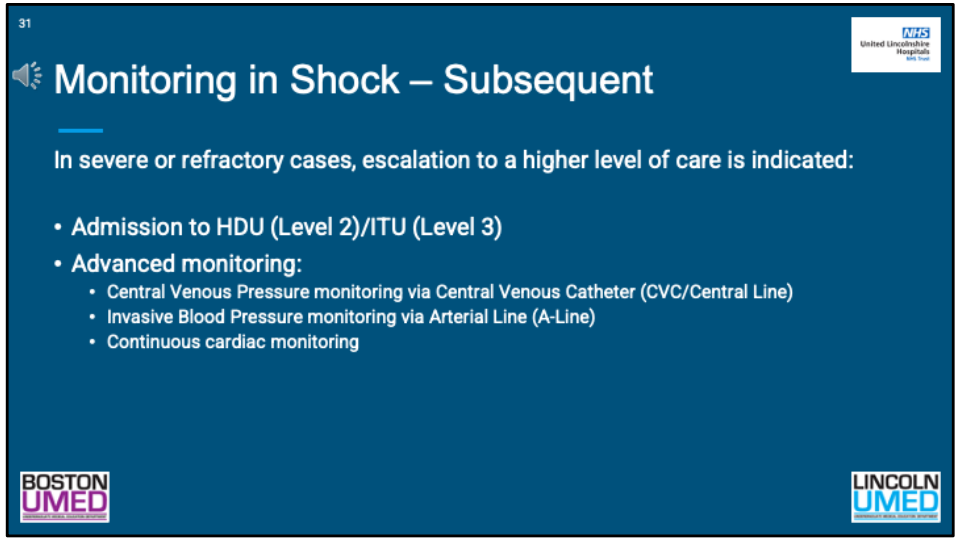
Blood flow is diverted away from the gut in shock (as the gut isn't particularly critical to our ability to flee a tiger…), so the gut mucosa becomes ischaemic, and this may over time result in stress ulceration and severe haemorrhage.

The release of pro-inflammatory cytokines and prostanoids results in immune modulation and suppression. This causes a susceptibility to infection. Meanwhile, neutrophil priming induces tissue damage in the heart, kidney, lungs, gut and liver, causing further morbidity in the patient with shock.



Your notes:

Monitoring in shock is undertaken initially through an A-E assessment, with at least one set of standard observations recorded. It is very important that the patient is regularly reassessed to monitor their response to treatment, and that observations are also repeated frequently.
Urine output monitoring gives us an idea of renal perfusion, and this gives us a good guide as to the degree of shock, and whether the patient is improving.
Within our A-E assessment, we will want to start some urgent investigations. A blood gas is important, as this will give us the Lactate – one of the signs of that reduced perfusion. It also allows us to monitor the patient's respiratory status, and whether there is any acid-base derangement. The nature of any acid-base derangement or respiratory failure may help us to identify the cause of the shock. Repetition of blood gases can allow us to monitor for any improvement or further deterioration.
A set of blood tests, tailored to your patient, will also help us to identify the cause of the shock. Common tests here include Full Blood Count, Urea & Electrolytes, CRP, Coagulation screen, Blood Cultures, and Cardiac markers such as Troponin or BNP. In the case of suspected anaphylaxis, you may wish to request a Mast cell tryptase in order to confirm the diagnosis. If you suspect haemorrhage, it is important to send off a sample of blood for Group & Save/Crossmatch.
An ECG is a quick, inexpensive, and non-invasive bedside test that can identify abnormal heart rhythms, and look for signs of myocardial injury. This is important to perform in any acutely unwell patient.



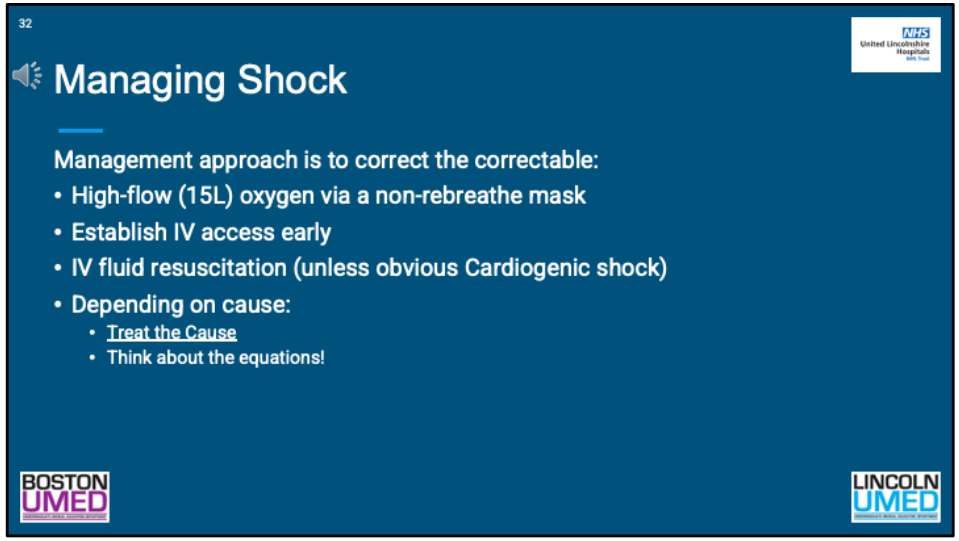
Your notes:

In cases where the patient doesn't respond sufficiently to initial treatment with fluid resuscitation, they usually require escalation to Level 2 or Level 3 care, where advanced monitoring can be carried out.
Such monitoring methods include Central Venous Pressure monitoring, Invasive Blood Pressure monitoring, and Continuous cardiac monitoring.
In this environment, we are also able to provide a much wider range of supportive interventions.

Central venous pressure monitoring can be used as a guide to the right atrial filling pressure, which is determined in part by fluid volume. So if this is low, it suggests that fluid replacement with boluses may be helpful in management. If this is raised, it may indicate obstructive shock, such as is caused by a massive PE, or Tension Pneumothorax.

Invasive blood pressure monitoring gives us moment-by-moment readings of blood pressure, without the time delay between recordings when cycling Non-invasive Blood Pressure measurements. This is particularly useful in unstable patients, where deterioration can be rapidly identified and treated with this continuous monitoring.

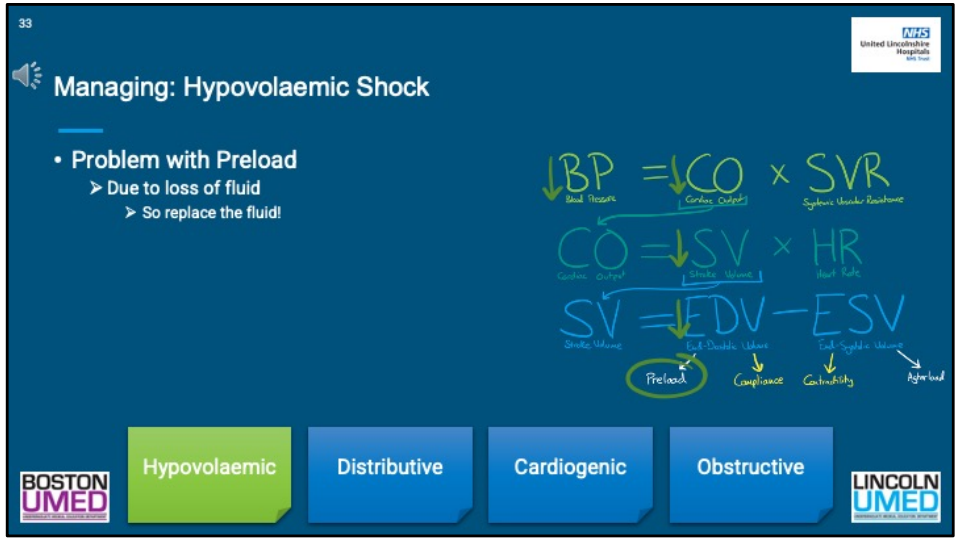
Continuous cardiac monitoring has similar benefits to invasive blood pressure monitoring, allowing us to identify arrhythmias immediately, rather than having to wait to attach electrodes and leads, and record the rhythm. This can reduce the delay to commencing management of any arrhythmia.



Your notes:

As ever, with A-E assessment, your job is to correct the correctable.
We discussed how the definition of shock is a life-threatening failure of oxygen delivery to tissues, and we can help to optimise oxygen delivery by providing high-flow oxygen at 15L/minute via a non-rebreathe mask.
We need to establish good and secure IV access early in our assessment, in order to commence IV fluid resuscitation (although if the patient is obviously in cardiogenic shock, IV fluid resuscitation can, and probably should, be avoided). We can also take blood when establishing IV access, in order to send this off for investigation

The remaining management is to identify and treat the cause, and support the patient's blood pressure in the ways you identify as necessary – you'll need to think about the equations here!



Your notes:

We discussed how, in hypovolaemic shock, the problem rests with preload. Simply put, the issue here is to do with intravascular fluid volume.

So, by extension, the management of this type of shock is to replace the fluid.

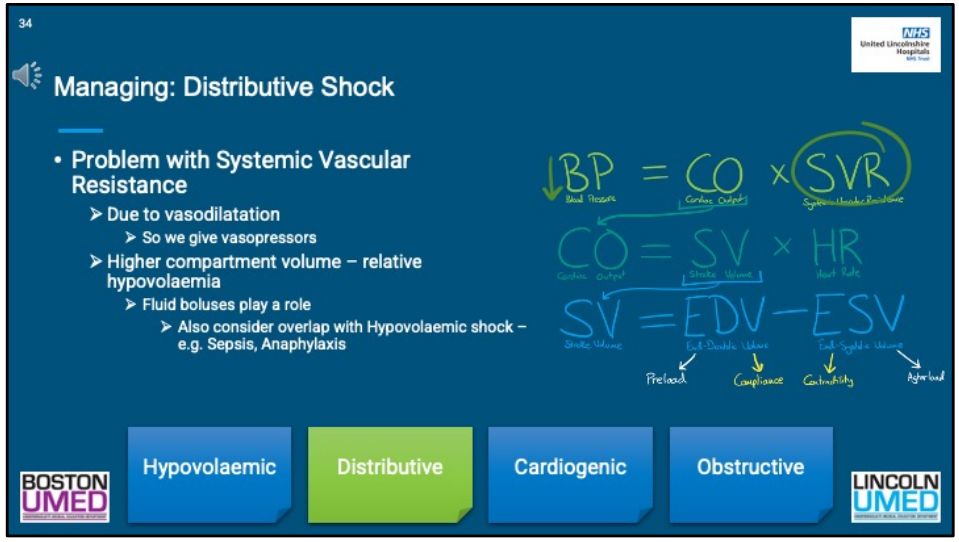
With fluid replacement, we're aiming for like-for-like.

So if the patient is bleeding, we need to replace this loss with blood products!

Most of the rest of the time, however, (0.9%) Normal saline boluses will suffice – my general rule of thumb is to give 500ml boluses, unless the patient is at risk of fluid overload, at which point I'd halve the bolus to 250ml.

Those at risk of fluid overload may include the frail, those in renal failure, or heart failure, but there are no hard-and-fast rules, so this depends on clinical judgement.

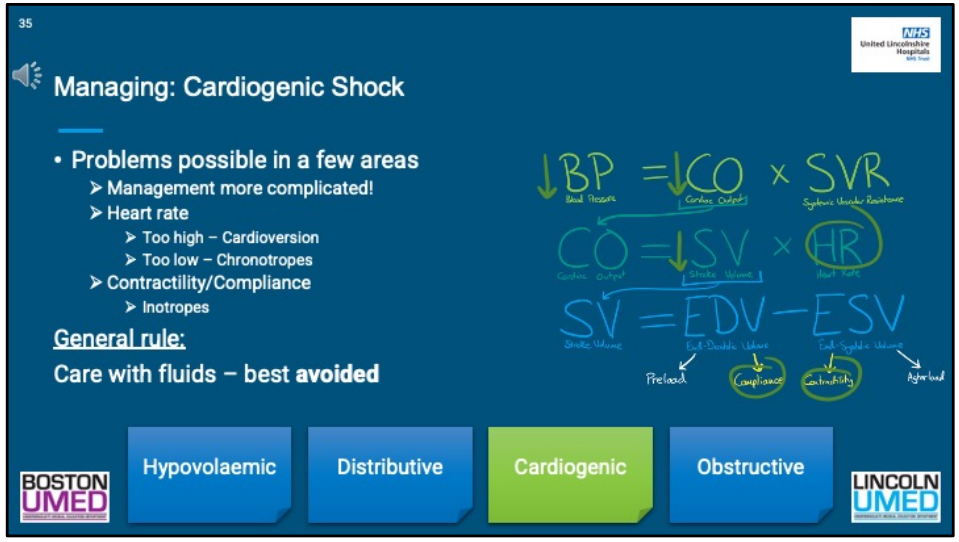
The most critical step with fluid boluses is to **reassess**. Has the bolus helped? If so, has it helped enough? (consider further boluses if not). If it hasn't helped, consider ongoing losses (and how to "put the plug in"), or whether there is a role for fluids (is this an obstructive shock? Do we need to relieve the obstruction?). Finally, if the patient has deteriorated, have they gone into fluid overload (stop the fluids!), and/or are they in cardiogenic shock (think of other courses of management, and stop the fluids!).



Your notes:

In distributive shock, we have abnormal vasodilatation causing a reduction in our Systemic Vascular Resistance.
The main treatment for this, therefore, is to reverse the vasodilatation, by administering vasopressors.
There is, however, due to an increased volume of the intravascular compartment, a relative hypovolaemia.
So, fluid boluses still play a role.

Also bear in mind the significant overlap here with hypovolaemic shock, in the subcategories of anaphylactic and septic shocks.

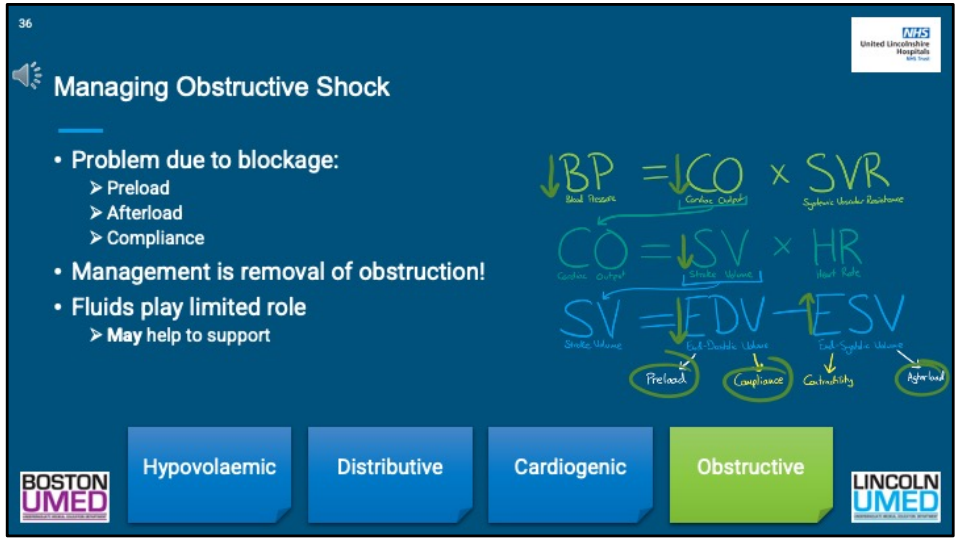


Your notes:

In cardiogenic shock, management gets somewhat more complicated, as there are a number of areas where the problem can occur.
If the issue is to do with the heart rate, we need to normalise this.
If it is too fast, we can cardiovert, whether chemically or electrically.
If it is too slow, we can administer chronotropic medications to increase the heart rate

If the problem rests with the contractility or compliance of the heart, Inotropic medicines can help to correct the abnormality.

A general rule with cardiogenic shock is that we should be careful with fluids, as they can worsen the patient's condition, and they are really best avoided in these cases

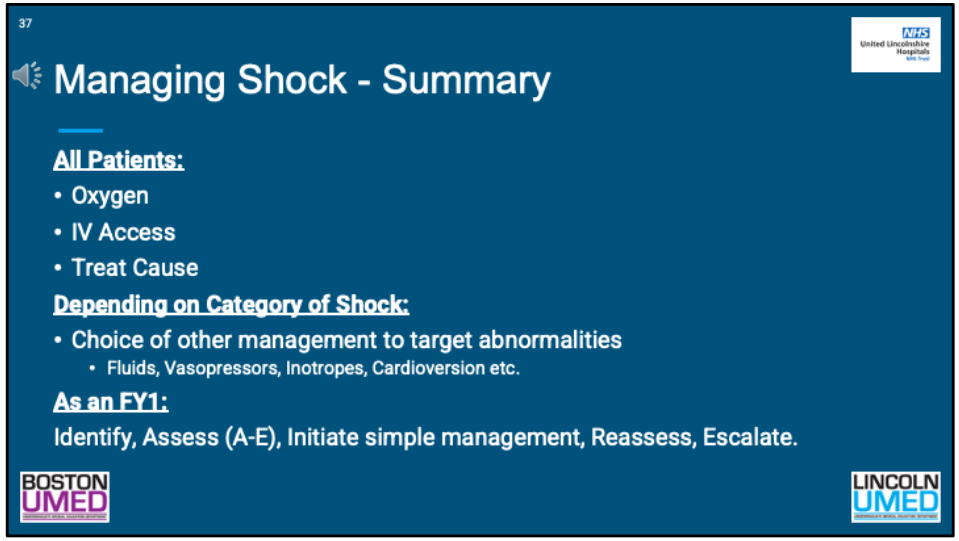


Your notes:

In obstructive shock, things are rather more simple, despite the range of potential causes in our equations.
There's an obstruction, or blockage, and it needs to be removed.
Fluids will play a limited role – they might help to support blood pressure (despite the patient being euvolaemic), and are unlikely to harm the patient if given.

In terms of removing the obstruction:

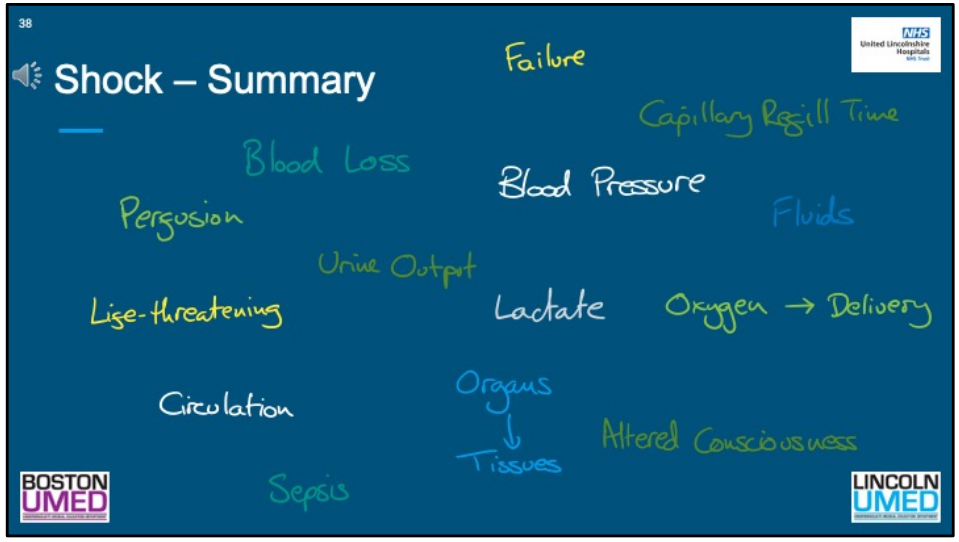
- Tension Pneumothorax – Needle decompression followed by chest drain insertion
- Massive PE – Thrombolysis
- Aortic dissection – Vascular surgery
- Cardiac Tamponade - Pericardiocentesis



Your notes:

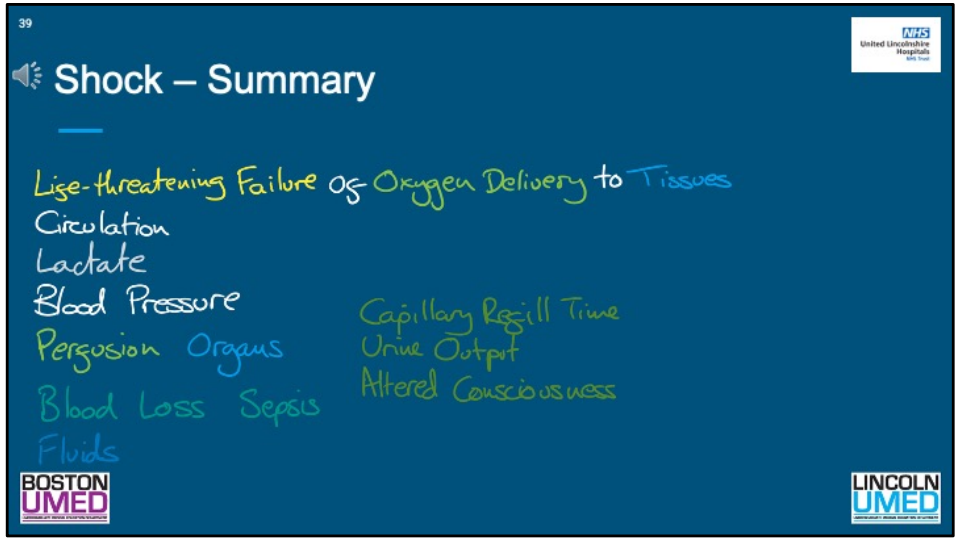
Our management, overall, is to give oxygen to optimise its delivery to tissues, gain IV access, and identify and treat the underlying cause.
Then, depending on the category of shock, we may provide other forms of management, such as fluids (most common), and more rarely vasopressors, inotropes, chronotropes or cardioversion. Bear in mind that these more rare interventions (i.e. those beyond fluid boluses) require escalation to a higher level of care.

So, your role as an F1 will be to identify the unwell shocked patient, undertake a comprehensive A-E assessment of them, and commence initial management such as Oxygen, Fluids, and simple treatments of the cause such as antibiotics. You'll need to reassess the patient to monitor for response to your treatment. It's also particularly important to escalate these patients to your seniors and, in the case of those that aren't responding to initial management, or those continuing to deteriorate, you may need to escalate to critical care.



Your notes:

So, in summary, we'll look back at the list of terms my previous students have mentioned, and see how they each fit into the picture.



Your notes:

I outlined how Shock is defined as the life-threatening failure of oxygen delivery to tissues.

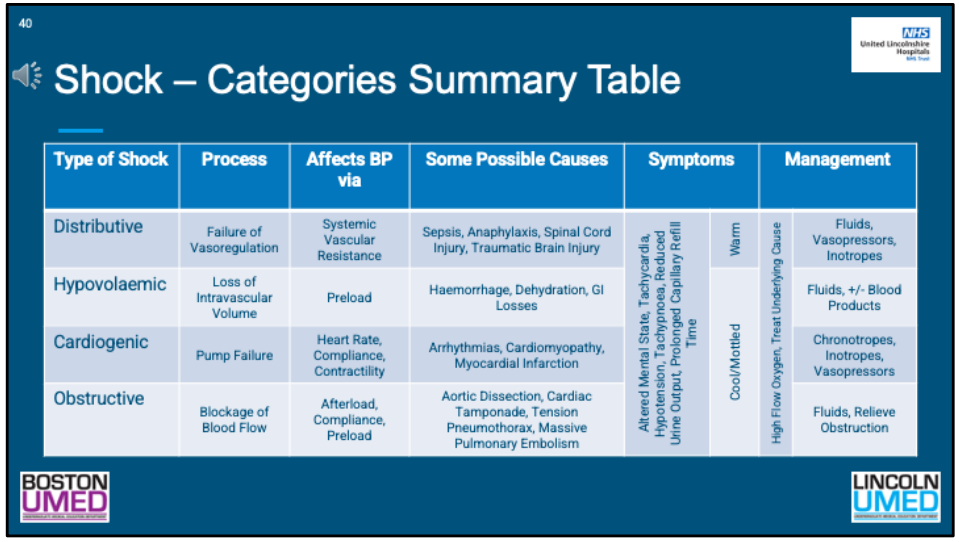
I also mentioned that this oxygen delivery is reliant on the circulation.

I outlined the triad that forms the case definition of shock, which consists of:

- A raised lactate – the by-product of the anaerobic respiration that occurs in the absence of sufficient oxygen supply
- A reduced Blood Pressure
- Evidence of reduced perfusion, visible in three organs:
    - Skin – Prolonged capillary refill time
    - Kidneys – Reduced Urine Output
    - Brain – Altered consciousness, or mental state

I've outlined the various types of shock, including subcategories and causes such as blood loss and sepsis. I discussed their differing aetiology (with fair warning in advance about the nerd-out here) and their differing presentations. On the next slide I'll include a summary table of the types of shock, for your reference.
Finally we have discussed the monitoring and management, which often includes fluid resuscitation but also includes other measures such as vasopressor, inotropic and chronotropic support, and most importantly the treatment of the underlying cause.

As ever, don't forget to escalate, and don't forget to reassess the patient!



Your notes:

This final slide is the promised summary table of each of the four main categories of shock. Feel free to use this as an aide-memoire.

I hope you've found this session useful. On a side note, feel free to let me know if you've spotted a couple of "Easter Eggs" hidden throughout the session.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Your notes: