

The USCOM and Haemodynamics

A Guide for Junior Medical and Nursing Staff



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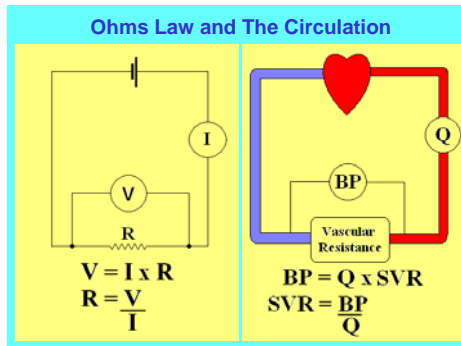
Introduction to the USCOM.

The USCOM is an ultrasonic cardiac output monitor which has recently become available in clinical practice. In essence, the USCOM works by measuring the velocity of the aortic or pulmonary blood flow as it leaves the heart. Validated internal algorithms calculate the diameter of the aortic and pulmonary valves based upon the patient's height and weight. If we know how big the "hole" is and how fast the blood is flowing through it and for how long, then we know how much blood flows per minute, which is, by definition, the cardiac output. Ultrasound is incredibly accurate at measuring blood flow velocities and the validated nomogram has also been shown to give accurate predicted values for the diameters of the heart valves. In the hands of an experienced user, the USCOM provides readings of cardiac output which are in the order of 95-98% accurate. It is the most accurate method of measuring cardiac output in clinical practice and is far superior in this regard to other methods such as pulmonary artery catheterisation (Swan-Ganz) which is around 80-85% accurate or PiCCO, which is about 65-70% accurate. The USCOM is also totally non-invasive.

The USCOM is much more than simply a method for measuring cardiac output however. This brief book outlines some of the many features that the USCOM brings to clinical practice and particularly its role in the optimization of haemodynamics. This booklet is not intended to be the definitive work on haemodynamics, but rather a quick look-up guide to what the numbers produced by the USCOM mean in simple terms and what they mean for your patient. There are many complex clinical entities covered by the global term of haemodynamics which are beyond the scope of this book, but a sound understanding of haemodynamics and what the USCOM can tell you, coupled to your medical knowledge and a little interpolation, should help you diagnose and treat even the most complex cardiovascular and pulmonary problems. If this guide seems basic in parts I make no apology; haemodynamics is not rocket science, but simply good basic physiology.

So what is haemodynamics? Essentially, it is the study of blood flow to the body tissues. All the tissues of the body need an adequate blood flow to deliver nutrients and oxygen and remove the products of metabolism. "Any circulatory disturbance leading to inadequate perfusion and inadequate oxygenation of the tissues" is as good a definition of shock as any, but cardiac failure, hypertension and hypotension without inadequate perfusion all fall under the umbrella of haemodynamics. Is the patient's hypoxia due to a pulmonary or cardiovascular cause? Haemodynamics and the USCOM will tell you. Is the patient's hypotension due to cardiogenic causes or vascular collapse? Again, haemodynamics and the USCOM will tell you.

Still interested? Then let's start with a very basic analogy between Ohm's Law and the circulation.



In the example on the left, we have a battery and a few wires coupled to a resistor. For a given current flow I , the voltage that is generated across the resistance is given by $I \times R$. This is Ohm's Law. In the example on the right, for any given blood flow, Q , the blood pressure that will be generated by this flow through the vascular resistance is given by the same formula, in this case $BP = Q \times SVR$. In effect, Ohm's Law works just as well for the circulation as it does for our simple circuit.

Did you notice that I said that blood pressure is generated by the blood flow, not the other way round? The heart is a volume pump not a pressure pump. Blood is a liquid and is therefore incompressible. As it tries to force its way through the peripheral resistance, being pushed by the heart, it generates a back pressure which is the blood pressure. This is the result of flow through the vascular resistance, it is not what made the blood flow in the first place. Once you get your head around this concept then the rest of haemodynamics becomes a lot easier to understand.

Blood pressure.

There are only two things that can go wrong with blood pressure, it can be too high, hypertension, or too low, hypotension. From our simple analogy with Ohm's Law we know that if the blood pressure is too low then it can only mean that the cardiac output is too low, the systemic vascular resistance is too low, or both are too low. Similarly, if the blood pressure is too high then either the cardiac output or systemic vascular resistance or both are too high. Simple.

$$BP = CO \times SVR$$

Cardiac Output.

The cardiac output is the product of the stroke volume and the heart rate, so we could rewrite our simple formula as **BP = SV x HR x SVR**

Now we know that the normal mean blood pressure is around 90 mmHg, a typical heart rate would be around 75 bpm, but how do we know the stroke volume and how do we calculate systemic vascular resistance?

The systemic vascular resistance can be calculated from the simple formula **SVR = BP/CO**. The problem of course is that we need to know the cardiac output in order to calculate the SVR, and to know the cardiac output we have to know the stroke volume. The USCOM measures cardiac output by first measuring stroke volume, which it does by measuring the ejection velocity of blood flow through the aortic or pulmonary valve and multiplying this by the cross-sectional area of the orifice. It then displays the stroke volume directly on-screen. By calculating the interval between successive pulses the heart rate can be measured. Multiplying the stroke volume by the heart rate gives us the cardiac output. If we input the patient's blood pressure then the USCOM can calculate the SVR.

Returning to the simple model of hypotension and hypertension, we can now say that if the BP is too high then it could be because the heart rate is too high, the stroke volume is too high, or the SVR is too high, or a combination of these. Similarly for hypotension, the heart rate, the stroke volume or the vascular resistance is too low, or a combination of these. Let's examine some clinical examples of this, starting with hypotension. In the first category the BP is low because the cardiac output is low because the stroke volume is low.

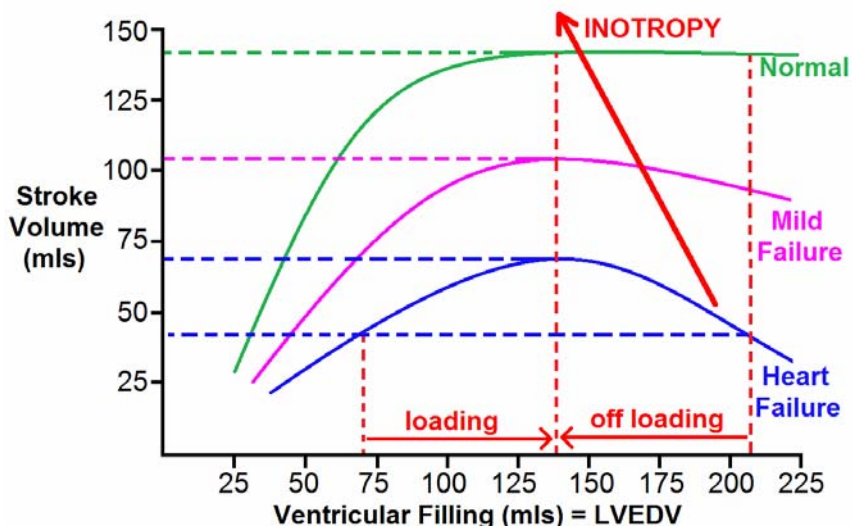
BP low, CO low, SV low

- **Myocardial contractility low**
- **Conduction defects**
- **Valvular heart disease**
- **Mechanical inefficiency**
- **“Toxic myocardium”**
- **Metabolic upsets**
- **Drugs, anaesthetics**
- **Arrhythmias**
- **Sub-optimal preload**

Most of these you will be familiar with, but what exactly do we mean by "sub-optimal preload"?

Preload and stroke volume.

To understand this we need to look at the classic work of Frank and Starling, who looked at the stroke volume that resulted from any given degree of ventricular preloading.



The ventricular preload is essentially the volume of blood in the ventricle immediately prior to systole. They found that very distinct patterns emerge for the normal heart and the failing heart. The diagram above shows three different preloadings, 1ml/kg, 2ml/kg and 3ml/kg, and three levels of heart function, normal, mild failure and established ventricular failure. If we look at the failing heart (blue line), we find that the stroke volume is critically dependent upon the preload. With an optimal preload of 2ml/kg the output stroke volume is almost double that of the underloaded heart at 1ml/kg or the overloaded heart at 3ml/kg. In effect, we can almost double the stroke volume simply by correcting the preload from the under or over loaded state to an optimum value. If the preload is too low, as in haemorrhage or dehydration, then this will respond to volume expansion, or loading. If the preload is too high, as may occur in congestive cardiac failure, then reducing it by using vasodilators or frusemide can produce a dramatic improvement in stroke volume and cardiac output.

Stroke volume is therefore critically dependent on the volume of blood in the left ventricle at the end of diastole, the end diastolic volume or LVEDV (or RVEDV in the case of the right ventricle). There is no simple way to measure this, but from the Frank Starling curve we know that the optimum value of end diastolic volume must be when the stroke volume is maximal. If we think the patient may be hypovolaemic or underfilled then try volume expansion, perhaps 250 to 500 ml of iv fluid and measure the stroke volume again. If we're sure that the patient is overloaded, then try a vasodilator such as GTN. Did the stroke volume and cardiac output increase as we expected? If the answer is yes then we are going the right way. Carry on doing what you're doing until the stroke volume reaches a peak and just begins to fall. You have now found the peak of the Frank Starling curve.

But what if we don't know if the ventricular preload is too high or too low? Do you try giving fluid and risk further overloading an already overloaded ventricle? What if the ventricle is not yet optimally loaded but we give furosemide or vasodilators, will we make things worse? *Primum non nocere*, first do no harm.

Can the USCOM tell us which way to go in this situation? The answer is yes, and very easily. First, with the patient lying supine, measure the stroke volume. Then elevate the legs (not tip the whole patient, just lift the legs). Note that you or an assistant must do this, not the patient. This will auto-transfuse a few hundred ml of blood into the central circulation. Did the SV increase or decrease? If the SV increased then the patient is under loaded. Simple volume expansion is called for. Did the SV fall? No problem, the ventricle is already overloaded. Put the legs down again, and this puts us back to where we started with no harm done. Now let's off-load the patient with a diuretic, vasodilator or whatever, just so long as it reduces the preload. How much vasodilation or diuretic do we need? Well, that dose that maximises the stroke volume - simple titration of preload against stroke volume. We can repeat the passive leg raising test as often as we need to.

Myocardial Contractility (Inotropy).

OK, so now we know how to hit the peak of the Frank Starling curve, but what if that is still not enough cardiac output? Here again, the Frank Starling curve and the USCOM tell us which way to go - increase inotropy. Now we know about inotropes, but which one and how much should be used? To answer this we need to know three things. What is the cardiac output now and what do we want it to be? What is the SVR now and what should it be? Is there a single inotrope which can do this, and if there is no single inotrope which can solve both problems then which combination of drugs do we use?

As we saw from our simple Ohm's Law analogy, SVR is equivalent to the resistance in the circuit. If it is too low then the voltage, which is analogous to blood pressure, will be too low. What should the SVR be? The section on typical values at the end of this booklet gives the figures for each age group, but a figure

of 1,000-1,600 is a good general guide. Does the patient need an increased or decreased SVR? The table below gives an indication of which drug to choose.

If we need increased inotropy and increased SVR then dopamine fits the bill, as would ephedrine if a lesser response would be adequate. If the SVR is already too high but we still need inotropy, then dobutamine would be a good choice.

Drug	Inotropy	Heart Rate	SVR
Adrenaline	↑↑	↑↑	↑↑
Noradrenaline	↑	↑ - ↓	↑↑
Dopamine	↑↑	↑ - ↑↑	↑ - ↑↑
Dobutamine	↑↑	↑	↓
Isoprenaline	0 - ↑	↑↑	↓
Phenylephrine	0 - ↓	↓ - 0	↑↑
Ephedrine	↑	↑	↑

If SVR is about right but we need a high degree of inotropy than a combination of dobutamine and dopamine would be appropriate. Other combinations can be "blended" to achieve the effect that we want, after all, we can easily measure both CO and SVR and see what progress we are making and then modify the treatment regimen accordingly.

If cardiac output is adequate but the low BP is due to low SVR i.e. excessive vasodilation, then a pure vasoconstrictor would be ideal. Which would you choose? Here, phenylephrine, or possibly noradrenaline, would seem reasonable.

Whatever you choose, the USCOM provides immediate haemodynamic feedback to allow you to fine-tune your treatment.

One question arises however. What should the cardiac output be? How do we judge this in a neonate, a child, an expectant mother or indeed a prop forward? This is where we need to know the cardiac index. The cardiac index is the cardiac output divided by the body surface area. For each square metre of body surface area we need a cardiac output of at least 2.4 litre/min/metre², the figure being higher in children, pregnancy and some disease states such as anaemia. The section on typical values gives some guidance as to the figures appropriate to each patient, but as a general rule 2.8 to 3.4 is a good ballpark figure in adults, whilst 3.2 to 4.4 is more typical in children.

So, we have now in effect defined a low cardiac output as a cardiac index that is below normal for that patient. By doing this we already know which way to go to treat cardiac failure, but the USCOM gives us even more help.

Preload - inotropy - afterload.

This simple triad is composed of the three elements which determine cardiac output. The preload, which equates to ventricular filling, can be too high or too low as we have seen. We know how to increase inotropy or myocardial contractility, but how can we measure it? Stroke volume gives us some clues, but is there anything else we can use? And what, after all this, is afterload?

One of the figures displayed by the USCOM is the Vpk, which is the peak velocity of ventricular ejection. Imagine you wanted to throw a ball into the air. The stronger the muscles in your arm, then the faster (and higher) you could throw the ball. The speed of the ball is therefore a good indication of the strength of your arm, just ask any cricket fielder or baseball pitcher. In the same way, the more powerful the ventricle, then the faster the blood will be ejected. Vpk tells us in a very real way how powerful the ventricle is. Vpk for the left ventricle is around 1.1 – 1.5 m/s in healthy patients. In patients with cardiac failure or low contractility/inotropy this figure might well be only 0.6 or 0.7 m/s or even less. For the right ventricle the figure would be 0.7 to 1.2 in healthy patients - see “typical values”. (In a later booklet, “The USCOM and Inotropy” you will read about a much more sophisticated way of measuring inotropy, but let’s stick with simple things for the time being.)

So far so good, but what is afterload and how do we measure it? In essence, afterload is simply the work that the heart has to do to push blood into the aorta and around the body. Imagine pushing a wheelbarrow full of sand. Is it easier to push it up hill or on the level? Pushing it up a gradient is hard work and gets harder the steeper the gradient. The blood pressure in the root of the aorta (or pulmonary artery for the right ventricle) is like the gradient of the hill. A high BP means that the ventricle is pushing uphill!

But what about pushing our wheelbarrow on the flat, but first over smooth concrete and then across a muddy field? The viscosity of the blood and the degree of vasodilation of the vascular tree have the same effect. High viscosity and vasoconstriction mean hard work for the ventricle.

Haemodynamics and the USCOM take the guesswork out of estimating afterload. From our analogy to Ohm’s Law, if $V = I \times R$ then $R = V/I$. For the circulation this is $BP = CO \times SVR$ and $SVR = BP/CO$.

The USCOM tells us how much resistance there is to the flow of blood and a sphygmomanometer can tell us the blood pressure, but the USCOM tells us something more, the minute distance.

Minute Distance.

The minute distance is how far the red blood cells travel in one minute, which might seem a strange thing to want to know until you realise that this is the mean flow rate in the aorta (or pulmonary artery). Just picture a river flowing. If the river is silting up or obstructed by fallen trees or abandoned cars, then the river flows slowly or almost stagnates. A healthy river without obstruction flows swiftly. The flow rate tells us how patent the peripheral circulation is, and also how easily the blood can flow through it which, as was mentioned above, depends to a large extent on viscosity. Just picture the same river, but instead of water it was mud flowing. How fast would the flow be then?

The normal flow rate for the aorta (aortic minute distance or AMD) is 14-22 m/min (10-16 m/min for the pulmonary artery, PMD). Suddenly the concept of hyperdynamic and hypodynamic circulation becomes clear. An aortic flow rate of 10 m/min is too slow, it is hypodynamic. An AMD of 28 m/min on the other hand is too fast and represents a clearly hyperdynamic circulation.

So now we know how to optimise preload, gauge myocardial contractility, and judge afterload, and we can measure stroke volume and cardiac output. Along with the blood pressure, we now have all the tools we need to optimize haemodynamics in the hypotensive patient. Take a look at the figures below generated by the USCOM for a 30 year old 60Kg female with a BP of 70/40. What is your diagnosis?

		V	ΔV	Avg
Exam Time: 16/08/2005 - 1:27:51 PM				
Transducer: 2.2MHz Mode: AV				
1				
2	Vpk (m/s)	2.7	-0.11	2.8
	vti (cm)	53	-2.6	53
3	HR (bpm)	79	4.9	77
	MD (m/min)	41	0.61	41
	ET% (%)	43	-1	44
	SV (cm ³)	153	-7.7	156
	CO (l/min)	12	0.18	12
	CI (l/min/m ²)	6.9	0.1	6.9
	SVR (d s cm ⁻⁵)	424	-6.3	427

If we scan down the values for Vpk, heart rate, MD, SV, CO, CI and SVR then it is obvious we are dealing with an apparently powerfully contracting heart which is generating a hyperdynamic circulation (AMD = 41 m/min) with an SV of around 2.5mls/kg, about double the value we would normally expect. This is generating a high CO of 12 L/min with a cardiac index of 6.9 L/min/m². Clearly the hypotension is not due to a low cardiac output! The answer is immediately apparent when we look at SVR, it is just about one third of normal. We are looking at marked peripheral vasodilation, vascular collapse if you like. This is a high-output, hyperdynamic state as a result of septicaemia. The heart is working almost maximally to increase the CO to try to raise the BP, but with an SVR this low, the heart just can't generate enough CO to compensate. The treatment? Raise the SVR to a value closer to 1200, and you know how to do that from what's gone before. Easy as painting by numbers! A word of caution though; when the SVR is this low any old heart could pump out a good output! There is very little opposition to ejection so stroke volume and ejection velocity are bound to be high. Does this have to mean that the ventricle is healthy? We'll return to this in later booklets.

But the USCOM isn't just about acute medical conditions. What about hypertension and chronic heart failure? Can the USCOM guide our therapy here? You bet it can!

Hypertension.

$$\text{BP} = \text{CO} \times \text{SVR}$$

Again this simple formula derived from Ohm's Law tells us that if the blood pressure is high then it can only be due either to increased cardiac output, raised vascular resistance, or a combination of both. The treatment of hypertension then becomes entirely straightforward. If the cardiac output is high and the SVR is normal then we need to reduce the cardiac output. This is where diuretics and β -blockers come into their own. If the cardiac output is normal but the vascular resistance is too high, then we need to use something that lowers the peripheral vascular resistance. This can be any one of the vasodilator drugs such as ACE inhibitors, Angiotensin Receptor Blockers (ARB's), calcium antagonists or whatever. If the elevated blood pressure is due to a combination of both increased cardiac output and increased SVR then we need a combination of the two types of treatment. But how much of each? Again the answer is simple - that amount of each drug which reduces the cardiac output and the vascular resistance to normal. If both CO and SVR are normal then blood pressure cannot be anything other than normal.

Compare this with the way that the majority of hypertension is treated both in primary care and even in hospital outpatients. You're just as likely to end up on one particular drug as any other. Indeed it is highly likely that the drug that you

will be prescribed is the one written on the side of the doctor's (free) ballpoint pen! In the past this is about as logical as prescribing in hypertension has been. With the USCOM we now have the ability to prescribe medication in hypertension in an entirely rational way based on the patient's underlying haemodynamics.

		V	ΔV	Avg
1				
17/05/1932				
Exam Time: 2/11/2004 - 5:53:07 PM				
Transducer: 2.2MHz				Mode: AV
2	Vpk (m/s)	1.2	0.00	0.98
	vti (cm)	14	0.00	15
	HR (bpm)	85	0.00	77
	MD (m/min)	12	0.00	11
	ET% (%)	44	0.00	45
	SV (cm ³)	37	0.00	43
	CO (l/min)	3.1	0.00	3.3
	CI (l/min/m ²)	2.1	0.00	2.2
	SVR (d s cm ⁻⁵)	2930	0.00	2930

These are the readings for a 72 year old female with hypertension (170/110), and angina, being treated with atenolol 50mg daily. What are your thoughts?

Clearly, her cardiac index is too low, whilst her SVR is sky high! Her AMD is only 11-12. It's not surprising that her BP is high or that her heart is working hard against that sort of afterload. This patient is crying out for a reduction in her SVR which would both reduce her BP and her angina, and probably allow her CI to increase to a more normal value. It's easy to add a vasodilator and re-evaluate her haemodynamics. Maybe she still needs a small dose of β -blocker as well as an ACE inhibitor or calcium antagonist, time will tell, but we can monitor exactly how she responds to medication and fine-tune our therapy now. No more guesswork.

As a parting thought, have you ever considered how many hundreds of clinical trials of anti-hypertensive medication have been carried out involving probably tens of thousands of patients? How many of them ever considered the underlying pathophysiology or haemodynamics? Given this, how much can we rely on this vast and almost unchallenged body of work? Can we take morbidity and mortality studies that were performed as if all hypertension were the same and apply this to any individual patient whose hypertension is due to say, a high cardiac output? Is their risk the same as the patient who has a raised SVR and a normal or low CO?

The answer is that we simply don't know and a vast amount of work lies waiting to be revisited, revalidated and completely re-evaluated!

Chronic heart failure.

Chronic heart failure is, by definition, a cardiac index of less than 2.4 litres per minute per square metre of body surface area, or 2.4 L/min/m^2 assuming a normal haemoglobin level and saturation. The aim of treatment in cardiac failure is to produce a cardiac index equal to or greater than 2.4 L/min/m^2 . We know from the previous pages that cardiac output depends on preload, inotropy (myocardial contractility), and afterload. The USCOM can guide us in manipulating each of these parameters individually, as well as showing us the overall outcome - the CO and CI. What's more, the USCOM can do this for both the right and the left ventricles independently, allowing us to optimize haemodynamics whichever side of the heart is the limiting factor.

Again, if we consider the management of cardiac failure in primary care and even hospital practice, it is very often done by guesswork. The patient simply describes how they feel since they commenced upon the new medication, be it digoxin, a diuretic, an ACE inhibitor or β -blocker. From the patient's description of their lifestyle, we try to guess whether their cardiac output has increased as a result of our therapy. If we were to treat hypertension without ever measuring the patient's blood pressure we would probably be regarded as being negligent, yet we treat patients with cardiac failure on a daily basis without ever measuring the outcome of our treatment.

OK, echocardiography can give some guidance, but how practical is it to perform echocardiography several times over a couple of months whilst we tailor our therapy to the patient's needs? Even then, echocardiography doesn't tell us the one thing we really need to know, the cardiac index. Now echocardiography can do this, but it takes an experienced echocardiographer anything up to 20 minutes or so to do this, so they generally don't bother unless specifically asked! In general they just "eyeball" the degree of ventricular contraction and quote a figure for "ejection fraction"(EF). Problem is, EF depends totally on preload and afterload as we saw above, and it even varies with heart rate. Can we do better?

The USCOM now brings a rational basis to prescribing in chronic cardiac failure. It takes all the guesswork out of the clinical situation. Simply measure the patient's cardiac index, modify the medication, remeasure the cardiac index. Did the change in medication help? If not then review it again. Haemodynamics is not rocket science.

Oxygen Delivery - DO₂

The prime function of the circulation is to deliver oxygen and nutrients to the tissues and to remove waste products from them. If we know the cardiac output and the patient's haemoglobin level and oxygen saturation, then we can calculate one further critical value in haemodynamics - the oxygen delivery to the body or DO₂.

One gramme of haemoglobin can carry 1.34 ml of oxygen as oxyhaemoglobin. If we know how many grammes of haemoglobin the patient has per litre of blood, what proportion of the haemoglobin is saturated (carrying oxygen), and how much blood the heart pumps each minute, then we can easily calculate the oxygen delivery to the tissues. (The small amount of oxygen carried in solution in plasma can be ignored as this represents only about 2% of the total.)

$$DO_2 = 1.34 \times \text{Hb conc.} \times \text{Cardiac Output} \times \frac{SaO_2}{100}$$

SaO₂ should strictly be measured from an arterial blood sample, but pulse oximetry gives us SpO₂ which is an acceptable surrogate for SaO₂. We can then revise our formula to:

$$DpO_2 = 1.34 \times \text{Hb conc.} \times \text{Cardiac Output} \times \frac{SpO_2}{100}$$

It probably won't come as a surprise to learn that the USCOM can have it's own plug-in pulse oximeter probe, with a data input screen to allow the user to enter the patient's haemoglobin concentration. The USCOM then performs the calculation above and voila - DO₂! (Well OK, DpO₂ to be pedantic.)

So what kind of values do we get for DO₂ in normal patients? If we take a normal haemoglobin level as 150g/L and a normal SpO₂ as 98% then if the CO is 5.5L/min, the calculation is

$$DO_2 = 1.34 \times 150 \times 5.5 \times \frac{98}{100} = 1,083 \text{ ml / minute}$$

This is a typical value for an adult, but what figures should we use for our neonate or indeed our linebacker or prop forward? In just the same way that we used cardiac index instead of total cardiac output to compare patients of differing sizes, so we can substitute cardiac index in place of cardiac output in the equation above, to give us oxygen delivery index or DO₂I. Taking a typical cardiac index as 2.4 – 3.2 L/min/m² and inserting this into the calculation we get

$$\text{DO}_2\text{I} = 1.34 \times 150 \times (2.4 \text{ to } 3.2) \times \frac{98}{100} = 473 \text{ to } 630 \text{ ml / minute / m}^2$$

From the above we can say that a DO_2I of 500 – 600 ml/min/m² is where we should be aiming in an adult. The comparable figure for a child, due to their high CI values, would be more like 700 - 850 ml/min/m².

If you want to scare yourself, try doing the calculation for someone who is anaemic, with a low CO or CI and then add in a reduced oxyhaemoglobin saturation. Suddenly we see why haemodynamics matters in this most fundamental of the circulatory functions. Haemodynamics and DO_2 are quite literally vital. Not only does the USCOM ensure that we can keep the haemodynamics in the correct range and keep the patient vital (i.e. alive!), but it takes the guesswork out of a critical area of medicine where guesswork is no longer acceptable.

In the later booklets in this series we'll go into much greater depth about just how precise circulatory control and manipulation can be with the USCOM, but I suspect you can already see that our old clinical methods are looking a little shaky!

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This document is not written in stone! Any advice or suggestions you may have are most welcome and may be incorporated in future versions of this guide.

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Appendix 1 - Normal USCOM Values - Adult Aortic

Age	Type	Vpk	Pmn	vti	MD	FT	FTc	SV	SVI	CO	CI	MAP	SVR	SVRI	SVV	SW	CPO	SMII	PKR	D02	D02I
16 to 25	Mean	1.4	3.7	28	20	314	346	80	49	5.9	3.6	85	1221	2027	20	902	1.1	1.84	26	1121	681
	Low	1.2	2.5	23	16	286	314	64	40	4.6	2.8	74	942	1507	12	698	0.8	1.40	17	886	533
	High	1.7	4.9	33	25	343	378	96	58	7.1	4.3	96	1501	2546	27	1106	1.4	2.30	36	1356	829
26 to 35	Mean	1.2	2.7	26	18	343	365	76	43	5.8	3.5	94	1216	2110	21	924	1.1	1.62	31	1105	665
	Low	1.0	1.7	22	15	304	320	63	35	4.8	2.9	89	848	1454	12	779	0.8	1.30	16	911	546
	High	1.4	3.7	30	21	383	410	89	50	6.8	4.2	99	1583	2767	30	1069	1.3	2.00	46	1299	783
36 to 45	Mean	1.2	2.8	27	20	347	385	78	45	5.7	3.3	89	1291	2247	20	911	1.1	1.59	35	1087	624
	Low	1.1	2.0	23	16	311	345	65	38	4.7	2.7	84	1060	1842	11	771	0.9	1.30	24	891	518
	High	1.4	3.6	31	23	383	425	91	51	6.7	3.8	94	1523	2651	30	1051	1.3	1.80	45	1283	730
46 to 55	Mean	1.2	2.8	26	18	336	383	72	44	5.1	3.1	82	1336	2239	19	772	0.9	1.48	36	972	591
	Low	1.0	2.0	23	15	302	346	63	36	4.2	2.4	77	1084	1712	11	680	0.8	1.20	25	811	466
	High	1.4	3.7	30	22	370	420	81	51	5.9	3.7	87	1587	2766	26	865	1.1	1.80	47	1134	717
> 55	Mean	1.0	2.1	24	16	354	370	63	40	4.2	2.7	82	1425	2221	21	604	0.7	1.13	37	795	509
	Low	0.9	1.6	21	13	325	347	55	35	3.5	2.2	78	1205	1876	12	509	0.5	1.00	28	667	430
	High	1.2	2.5	27	18	384	393	71	46	4.8	3.1	86	1646	2565	30	700	0.8	1.30	46	923	589
		m/s	mmHg	cm	m/min	ms	ms	ml	ml/m ²	l/min	l/min/m ²	mmHg	d.s.cm ⁻⁵	d.s.cm ⁻⁵ m ²	%	mJ	W	W/m ²		ml/min	ml/min/m ²

These values are supplied as a guide only. The generalisability of these values to all subjects has not been confirmed. The author recommends that the normal values and ranges for any particular demographic group should be established locally.

Appendix 2 - Normal USCOM Values - Adult Pulmonary

Age	Type	Vpk	Pmn	vti	MD	FT	FTc	SV	SVI	CO	CI	MAP	SVR	SVRI	SVV	SW	CPO	SMII	PKR	D02	D02I
16 to 25	Mean	1.1	2.1	23	17	340	374	80	49	5.9	3.6	85	1221	2027	20	902	1.1	1.84	26	1121	681
	Low	0.9	1.4	19	14	309	339	64	40	4.6	2.8	74	942	1507	12	698	0.8	1.40	17	886	533
	High	1.3	2.8	27	20	370	408	96	58	7.1	4.3	96	1501	2546	27	1106	1.4	2.30	36	1356	829
26 to 35	Mean	0.9	1.6	21	15	371	394	76	43	5.8	3.5	94	1216	2110	21	924	1.1	1.62	31	1105	665
	Low	0.8	1.0	18	12	329	346	63	35	4.8	2.9	89	848	1454	12	779	0.8	1.30	16	911	546
	High	1.1	2.2	25	18	413	443	89	50	6.8	4.2	99	1583	2767	30	1069	1.3	2.00	46	1299	783
36 to 45	Mean	1.0	1.6	22	16	375	416	78	45	5.7	3.3	89	1291	2247	20	911	1.1	1.59	35	1087	624
	Low	0.8	1.2	19	13	336	373	65	38	4.7	2.7	84	1060	1842	11	771	0.9	1.30	24	891	518
	High	1.1	2.1	26	19	413	459	91	51	6.7	3.8	94	1523	2651	30	1051	1.3	1.80	45	1283	730
46 to 55	Mean	1.0	1.7	22	15	363	414	72	44	5.1	3.1	82	1336	2239	19	772	0.9	1.48	36	972	591
	Low	0.8	1.2	19	12	326	374	63	36	4.2	2.4	77	1084	1712	11	680	0.8	1.20	25	811	466
	High	1.1	2.1	25	18	400	454	81	51	5.9	3.7	87	1587	2766	26	865	1.1	1.80	47	1134	717
> 55	Mean	0.8	1.2	20	13	382	400	63	40	4.2	2.7	82	1425	2221	21	604	0.7	1.13	37	795	509
	Low	0.7	0.9	17	11	350	375	55	35	3.5	2.2	78	1205	1876	12	509	0.5	1.00	28	667	430
	High	0.9	1.5	22	15	414	424	71	46	4.8	3.1	86	1646	2565	30	700	0.8	1.30	46	923	589
		m/s	mmHg	cm	m/min	ms	ms	ml	ml/m ²	l/min	l/min/m ²	mmHg	d.s.cm ⁻⁵	d.s.cm ⁻⁵ m ²	%	mJ	W	W/m ²		ml/min	ml/min/m ²

These values are supplied as a guide only. The generalisability of these values to all subjects has not been confirmed. The author recommends that the normal values and ranges for any particular demographic group should be established locally.

Appendix 3 - Normal USCOM Values - Paediatric Aortic – Neonate to 6 years

Age	Type	BSA	Vpk	vti	HR	MD	FT	FTc	SV	SVI	CO	CI	Hb	D02	D02I	SBP	DBP	MAP	SVR	SVRI	SMII	PKR
1 to 30 days	Mean	0.22	1.13	16.4	125	17.9	239	355	5.5	25	0.78	3.5	155	162	736	73	39	50	5068	1405	0.71	33
	Low	0.18	0.96	14.2	115	16.0	214	326	4.2	20	0.62	3.1	142	129	637	64	29	41	3679	1204	0.60	27
	High	0.26	1.30	18.6	135	19.8	264	384	6.8	30	0.94	4.0	168	195	836	83	50	59	6457	1606	0.82	38
1 to 12 mths	Mean	0.41	1.31	20.5	124	25.4	255	363	14.8	36	1.83	4.4	125	306	740	85	52	63	2889	1191	1.24	23
	Low	0.35	1.12	18.4	103	20.9	224	339	12.9	31	1.49	3.7	103	250	623	68	37	50	2111	919	1.08	15
	High	0.48	1.50	22.6	145	29.9	285	386	16.6	40	2.16	5.1	147	362	858	102	68	76	3666	1464	1.40	32
1	Mean	0.50	1.39	21.8	119	25.6	259	362	19.8	39	2.32	4.6	118	365	732	90	50	64	2256	1125	1.45	21
	Low	0.42	1.16	19.2	110	22.6	232	326	16.5	34	1.99	4.1	96	314	646	73	34	49	1790	904	1.03	14
	High	0.58	1.62	24.3	128	28.7	285	398	23.1	44	2.65	5.2	139	417	818	107	67	78	2722	1345	1.88	28
2	Mean	0.60	1.38	26.2	104	26.8	305	398	29.1	49	2.96	5.0	117	464	777	96	53	67	1879	1120	1.50	22
	Low	0.49	1.18	21.8	90	22.3	277	371	23.0	40	2.46	4.1	94	386	647	76	35	50	1486	884	1.23	15
	High	0.70	1.59	30.6	118	31.3	333	425	35.2	57	3.46	5.8	140	543	907	116	72	85	2273	1356	1.78	30
3	Mean	0.68	1.49	27.9	99	27.4	303	387	35.3	52	3.45	5.1	114	528	774	102	55	71	1713	1166	1.70	20
	Low	0.54	1.27	23.6	86	22.6	270	345	28.4	43	2.78	4.1	93	425	622	80	37	54	1290	876	1.37	13
	High	0.82	1.71	32.2	112	32.2	336	429	42.2	61	4.13	6.1	135	631	926	124	73	87	2136	1457	2.03	27
4	Mean	0.74	1.54	29.1	95	27.6	312	390	40.4	55	3.82	5.2	115	589	794	102	53	69	1504	1107	1.72	18
	Low	0.57	1.33	25.4	81	22.4	281	350	33.5	47	3.02	4.1	94	465	631	81	33	52	1204	890	1.37	13
	High	0.91	1.74	32.9	109	32.8	342	430	47.3	63	4.62	6.2	136	712	956	122	72	85	1805	1323	2.07	24
5	Mean	0.80	1.47	29.1	89	25.6	322	390	44.7	56	3.93	4.9	117	616	768	103	54	70	1477	1176	1.71	20
	Low	0.64	1.27	25.3	78	21.4	298	356	37.4	48	3.18	4.1	98	499	641	79	35	52	1166	947	1.41	15
	High	0.97	1.68	33.0	100	29.9	347	423	52.0	64	4.67	5.7	136	733	895	126	73	88	1787	1405	2.01	26
6	Mean	0.88	1.48	29.6	85	25.1	323	383	49.3	56	4.16	4.8	116	647	739	107	56	73	1459	1269	1.80	21
	Low	0.67	1.27	25.6	73	20.7	301	353	40.6	49	3.35	3.9	95	520	605	82	35	54	1148	1014	1.44	12
	High	1.08	1.69	33.7	97	29.4	346	413	58.0	64	4.98	5.6	137	774	874	132	77	93	1771	1525	2.17	30
		m ²	m/s	cm	bpm	m/min	ms	ms	ml	ml/m ²	l/min	l/min/m ²	g/l	ml/min	ml/min/m ²	mmHg	mmHg	mmHg	d.s.cm ⁻⁵	d.s.cm ⁻⁵ m ²	W/m ²	

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Appendix 4 - Normal USCOM Values - Paediatric Aortic – 7 to 16 years

Age	Type	BSA	Vpk	vti	HR	MD	FT	FTc	SV	SVI	CO	CI	Hb	D02	D02I	SBP	DBP	MAP	SVR	SVRI	SMII	PKR
7	Mean	0.94	1.52	30.2	84	25.3	322	379	53.8	58	4.48	4.8	115	691	736	111	58	76	1393	1290	1.91	20
	Low	0.71	1.32	26.3	71	21.1	298	349	43.6	49	3.60	4.0	93	555	606	87	42	59	1141	1073	1.56	15
	High	1.17	1.72	34.1	97	29.5	346	409	63.9	66	5.36	5.7	137	826	867	135	74	93	1645	1507	2.26	26
8	Mean	1.03	1.50	30.4	84	25.2	328	384	59.1	58	4.90	4.8	116	761	741	114	60	78	1323	1343	1.94	22
	Low	0.74	1.25	25.7	71	20.4	302	353	48.0	49	3.86	3.9	91	600	592	90	44	61	1058	1078	1.56	15
	High	1.31	1.74	35.1	96	30.1	353	415	70.2	67	5.94	5.8	141	923	889	137	76	95	1589	1607	2.32	28
9	Mean	1.12	1.45	30.0	83	24.8	332	387	62.3	57	5.17	4.7	118	817	731	113	60	78	1268	1373	1.88	23
	Low	0.80	1.21	25.7	70	19.4	305	356	51.2	49	3.86	3.8	96	610	587	90	44	61	1004	1121	1.48	16
	High	1.43	1.69	34.4	96	30.3	358	418	73.5	65	6.47	5.6	140	1023	875	136	76	95	1531	1625	2.29	29
10	Mean	1.22	1.53	31.4	77	24.0	331	372	70.0	58	5.36	4.5	120	861	706	115	61	79	1245	1491	1.96	21
	Low	0.86	1.29	26.7	65	19.2	306	344	56.2	48	4.07	3.5	97	654	553	92	47	63	949	1116	1.56	15
	High	1.58	1.76	36.1	89	28.8	357	401	83.9	68	6.64	5.4	143	1068	859	139	76	95	1541	1867	2.37	27
11	Mean	1.29	1.51	31.1	78	24.0	330	374	73.8	57	5.71	4.5	120	918	709	117	62	80	1174	1498	1.97	21
	Low	0.96	1.32	26.8	66	19.8	305	340	60.6	49	4.49	3.6	99	723	572	94	46	64	917	1181	1.60	16
	High	1.63	1.71	35.3	90	28.3	355	408	87.1	65	6.93	5.3	141	1114	846	140	79	97	1430	1815	2.33	27
12	Mean	1.35	1.74	34.9	81	28.2	331	382	86.0	64	6.92	5.1	120	1113	823	122	63	83	988	1323	2.29	17
	Low	0.99	1.45	30.6	68	23.0	308	355	71.3	57	5.55	4.3	98	892	687	106	42	65	805	1090	1.84	12
	High	1.72	2.04	39.3	94	33.4	353	409	100.6	70	8.29	6.0	142	1333	959	139	84	101	1171	1556	2.73	22
13 to 16	Mean	1.49	1.78	35.8	79	25.2	333	376	92.3	62	6.88	4.6	124	1143	767	124	65	85	991	1476	2.17	22
	Low	1.17	1.57	31.5	67	20.5	310	344	79.4	53	5.61	3.7	99	939	622	103	47	67	740	1102	1.74	17
	High	1.81	1.99	40.1	92	29.9	356	408	105.2	71	8.15	5.6	149	1347	912	145	83	103	1242	1850	2.60	28
		m ²	m/s	cm	bpm	m/min	ms	ms	ml	ml/m ²	l/min	l/min/m ²	g/l	ml/min	ml/min/m ²	mmHg	mmHg	mmHg	d.s.cm ⁻⁵	d.s.cm ⁻⁵ m ²	W/m ²	

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Appendix 5 - Normal USCOM Values - Paediatric Pulmonary – Neonate to 6 years

Age	Type	BSA	Vpk	vti	HR	MD	FT	FTc	SV	SVI	CO	CI	Hb	D02	D02I	SBP	DBP	MAP	SVR	SVRI	SMII	PKR
1 to 30 days	Mean	0.22	0.86	13.5	125	14.8	258	383	5.50	25	0.78	3.5	155	162	736	73	39	50	5068	1405	0.71	33
	Low	0.18	0.73	11.8	115	13.2	231	352	4.20	20	0.62	3.1	142	129	637	64	29	41	3679	1204	0.60	27
	High	0.26	0.99	15.3	135	16.4	285	414	6.80	30	0.94	4.0	168	195	836	83	50	59	6457	1606	0.82	38
1 to 12 mths	Mean	0.41	0.99	16.9	124	21.0	275	392	14.8	36	1.83	4.4	125	306	740	85	52	63	2889	1191	1.24	23
	Low	0.35	0.85	15.2	103	17.2	242	366	12.9	31	1.49	3.7	103	250	623	68	37	50	2111	919	1.08	15
	High	0.48	1.14	18.6	145	24.7	308	417	16.6	40	2.16	5.1	147	362	858	102	68	76	3666	1464	1.40	32
1	Mean	0.50	1.06	18.0	119	21.2	279	391	19.8	39	2.32	4.6	118	365	732	90	50	64	2256	1125	1.45	21
	Low	0.42	0.88	15.8	110	18.7	251	352	16.5	34	1.99	4.1	96	314	646	73	34	49	1790	904	1.03	14
	High	0.58	1.23	20.1	128	23.7	308	429	23.1	44	2.65	5.2	139	417	818	107	67	78	2722	1345	1.88	28
2	Mean	0.60	1.05	21.6	104	22.1	330	430	29.1	49	2.96	5.0	117	464	777	96	53	67	1879	1120	1.50	22
	Low	0.49	0.90	18.0	90	18.4	300	401	23.0	40	2.46	4.1	94	386	647	76	35	50	1486	884	1.23	15
	High	0.70	1.21	25.3	118	25.9	360	459	35.2	57	3.46	5.8	140	543	907	116	72	85	2273	1356	1.78	30
3	Mean	0.68	1.13	23.0	99	22.7	327	418	35.3	52	3.45	5.1	114	528	774	102	55	71	1713	1166	1.70	20
	Low	0.54	0.97	19.5	86	18.7	292	373	28.4	43	2.78	4.1	93	425	622	80	37	54	1290	876	1.37	13
	High	0.82	1.30	26.6	112	26.6	363	464	42.2	61	4.13	6.1	135	631	926	124	73	87	2136	1457	2.03	27
4	Mean	0.74	1.17	24.1	95	22.8	337	421	40.4	55	3.82	5.2	115	589	794	102	53	69	1504	1107	1.72	18
	Low	0.57	1.01	20.9	81	18.5	303	378	33.5	47	3.02	4.1	94	465	631	81	33	52	1204	890	1.37	13
	High	0.91	1.33	27.2	109	27.1	370	464	47.3	63	4.62	6.2	136	712	956	122	72	85	1805	1323	2.07	24
5	Mean	0.80	1.12	24.1	89	21.2	348	421	44.7	56	3.93	4.9	117	616	768	103	54	70	1477	1176	1.71	20
	Low	0.64	0.96	20.9	78	17.7	322	385	37.4	48	3.18	4.1	98	499	641	79	35	52	1166	947	1.41	15
	High	0.97	1.27	27.3	100	24.7	374	457	52.0	64	4.67	5.7	136	733	895	126	73	88	1787	1405	2.01	26
6	Mean	0.88	1.13	24.5	85	20.7	349	414	49.3	56	4.16	4.8	116	647	739	107	56	73	1459	1269	1.80	21
	Low	0.67	0.97	21.2	73	17.1	325	382	40.6	49	3.35	3.9	95	520	605	82	35	54	1148	1014	1.44	12
	High	1.08	1.29	27.8	97	24.3	373	446	58.0	64	4.98	5.6	137	774	874	132	77	93	1771	1525	2.17	30
		m ²	m/s	cm	bpm	m/min	ms	ms	ml	ml/m ²	l/min	l/min/m ²	g/l	ml/min	ml/min/m ²	mmHg	mmHg	mmHg	d.s.cm ⁻⁵	d.s.cm ⁻⁵ m ²	W/m ²	

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Appendix 6 - Normal USCOM Values - Paediatric Pulmonary – 7 to 16 years

Age	Type	BSA	Vpk	vti	HR	MD	FT	FTc	SV	SVI	CO	CI	Hb	D02	D02I	SBP	DBP	MAP	SVR	SVRI	SMII	PKR
7	Mean	0.94	1.16	25.0	84	20.9	348	409	53.8	58	4.48	4.8	115	691	736	111	58	76	1393	1290	1.91	20
	Low	0.71	1.00	21.7	71	17.4	322	377	43.6	49	3.60	4.0	93	555	606	87	42	59	1141	1073	1.56	15
	High	1.17	1.31	28.2	97	24.3	374	442	63.9	66	5.36	5.7	137	826	867	135	74	93	1645	1507	2.26	26
8	Mean	1.03	1.14	25.1	84	20.8	354	415	59.1	58	4.90	4.8	116	761	741	114	60	78	1323	1343	1.94	22
	Low	0.74	0.95	21.2	71	16.9	326	381	48.0	49	3.86	3.9	91	600	592	90	44	61	1058	1078	1.56	15
	High	1.31	1.33	29.0	96	24.8	381	449	70.2	67	5.94	5.8	141	923	889	137	76	95	1589	1607	2.32	28
9	Mean	1.12	1.10	24.8	83	20.5	358	418	62.3	57	5.17	4.7	118	817	731	113	60	78	1268	1373	1.88	23
	Low	0.80	0.92	21.2	70	16.0	329	385	51.2	49	3.86	3.8	96	610	587	90	44	61	1004	1121	1.48	16
	High	1.43	1.28	28.4	96	25.0	387	452	73.5	65	6.47	5.6	140	1023	875	136	76	95	1531	1625	2.29	29
10	Mean	1.22	1.16	25.9	77	19.8	358	402	70.0	58	5.36	4.5	120	861	706	115	61	79	1245	1491	1.96	21
	Low	0.86	0.98	22.0	65	15.8	331	371	56.2	48	4.07	3.5	97	654	553	92	47	63	949	1116	1.56	15
	High	1.58	1.34	29.8	89	23.8	385	433	83.9	68	6.64	5.4	143	1068	859	139	76	95	1541	1867	2.37	27
11	Mean	1.29	1.15	25.7	78	19.9	356	404	73.8	57	5.71	4.5	120	918	709	117	62	80	1174	1498	1.97	21
	Low	0.96	1.00	22.2	66	16.3	329	367	60.6	49	4.49	3.6	99	723	572	94	46	64	917	1181	1.60	16
	High	1.63	1.30	29.2	90	23.4	384	441	87.1	65	6.93	5.3	141	1114	846	140	79	97	1430	1815	2.33	27
12	Mean	1.35	1.32	28.9	81	23.3	357	413	86.0	64	6.92	5.1	120	1113	823	122	63	83	988	1323	2.29	17
	Low	0.99	1.10	25.3	68	19.0	333	384	71.3	57	5.55	4.3	98	892	687	106	42	65	805	1090	1.84	12
	High	1.72	1.55	32.5	94	27.6	381	441	100.6	70	8.29	6.0	142	1333	959	139	84	101	1171	1556	2.73	22
13 to 16	Mean	1.49	1.35	29.6	79	20.8	360	406	92.3	62	6.88	4.6	124	1143	767	124	65	85	991	1476	2.17	22
	Low	1.17	1.19	26.0	67	16.9	335	372	79.4	53	5.61	3.7	99	939	622	103	47	67	740	1102	1.74	17
	High	1.81	1.51	33.1	92	24.7	384	441	105.2	71	8.15	5.6	149	1347	912	145	83	103	1242	1850	2.60	28
		m ²	m/s	cm	bpm	m/min	ms	ms	ml	ml/m ²	l/min	l/min/m ²	g/l	ml/min	ml/min/m ²	mmHg	mmHg	mmHg	d.s.cm ⁻⁵	d.s.cm ⁻⁵ m ²	W/m ²	

These values are supplied as a guide only. The generalisability of these values to all subjects has not been confirmed. The author recommends that the normal values and ranges for any particular demographic group should be established locally.