3.
The USCOM and Inotropy
A Guide for Junior Medical and Nursing Staff

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Introduction.

So what exactly is inotropy? In a very real sense, inotropy is the power of the heart. In the same way that we talk about the strength of a muscle like the biceps, so we can talk about the strength of a muscle like the heart. In short, when it contracts how powerful is that contraction? Although not strictly the same thing, most clinicians now use the terms inotropy and myocardial contractility interchangeably to mean the power of contraction of the heart.

Inotropy is vitally important in haemodynamics. Cardiac output depends on stroke volume and heart rate. Stroke volume depends on three factors; preload which is the degree of ventricular filling at the start of systole, afterload which is the load the ventricle has to work against and is basically the mean aortic blood pressure, and inotropy. The heart has to ***respond*** to changes in preload and afterload to maintain a normal stroke volume and it does this through variations in inotropy. If preload increases, i.e. if more venous blood comes to the heart, then the ventricular fibres are stretched and respond with increased force of contraction and increased stroke volume, so more blood is pumped out of the heart! Similar responses occur if afterload changes. If the SVR rises then the heart has to contract more forcibly to continue ejecting a stable stroke volume. Simple, but crucial.

If the heart could not respond to arterial vasoconstriction for example, then the ventricle would dilate and fail. This is often seen in septicaemia where a very low SVR and blood pressure is treated by a simple vasoconstrictor. The cardiac output may initially be high and ventricular emptying may also be high with a stroke volume of over 75% of the resting end-diastolic volume (ejection fraction > 75%). As the arterial tree is very vasodilated, it is easy for the left ventricle to empty into the aorta. Very little effort is needed if the blood pressure is only 70/40 and the SVR 200! On an echocardiogram, the ejection fraction and cardiac movements may look great and can give a false sense of security.

However, the myocardium is often severely depressed in septicaemia and cannot overcome the increased afterload which follows the use of a vasoconstrictor. The ventricle now has to work much harder to eject the same stroke volume. If it cannot produce that extra power then it fails, sometimes very abruptly. We have to ensure that the ventricle has sufficient inotropy to cope with the afterload increase. Problem is, how do we know what power it has to start with and how do we know we have

given it sufficient power through the use of inotropes to cope with the arterial pressure we aim to expose it to? That's a tough one!

Similarly, not increasing stroke volume in response to increased preload leads to a backlog of blood in the venous system, increased venous pressure and oedema. This is bad enough in right heart failure with systemic oedema, but even worse in left ventricular failure with pulmonary oedema.

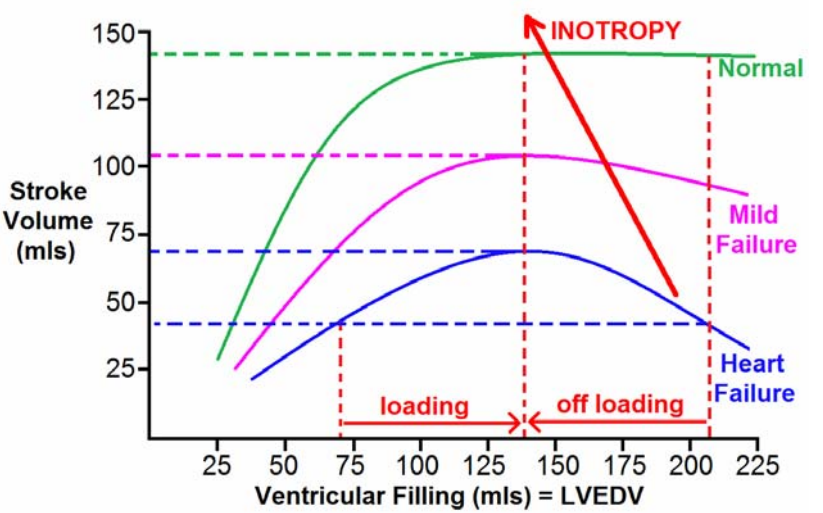
We really have very limited choices. We could reduce the preload by vasodilation, as with nitrates or frusemide (which reduces preload by vasodilation long before we see the first drop of urine!). We could reduce the circulating volume by diuresis, dialysis, fluid and salt restriction or even blood-letting, depending on the urgency. Alternatively, we could reduce the afterload so that the ventricle can empty more easily, so it can take more blood from the venous system and keep it pushed out into the body, an increase in forward flow. This is one of the actions of ACE inhibitors for example, and the reason why they are used in chronic LVF.

But what if the problem is just that the myocardium has insufficient "oomph" to cope at all? Our only choice is to increase inotropy and power the heart up to a more normal level. Problem is, as before, how do we know what power it has to start with and how do we know we have given it sufficient power through the use of inotropes to cope with the demands placed on it?

We have all seen inotropes used clinically, but how do we know when to use them? Which one do we use? How much should we use? What are our clinical targets or therapeutic goals? How do we know when we've reached them? How much easier life would be if only we could measure inotropy quickly and easily. Life without guesswork! We could replace questions with logical answers.

The USCOM and Inotropy.

Can we measure inotropy using the USCOM? You bet we can! And it's surprisingly simple and quick to do. Let's start with some very basic physiology (don't I always?)



Preload is otherwise known as ventricular filling, or more specifically, that volume that is in the ventricle immediately before the onset of systole. This is the ventricular end-diastolic volume or VEDV, and can be right or left depending on the ventricle involved, giving us RVEDV and LVEDV. In the main, preload normally refers to LVEDV (and no, they are not always the same volumes on both sides!). The Frank-Starling curve shows that for any given preload, the stroke volume depends critically on inotropy. In ventricular failure where inotropy is low, stroke volume will also be low. Cardiac output and cardiac index will consequently be low unless the heart rate can increase considerably. In the three curves above, a threefold increase in heart rate would be required to maintain CO and CI as inotropy falls from the highest level to the lowest, as CO = Stroke Volume x Heart Rate. Stroke volume is at the very centre of haemodynamics, and inotropy is at the heart of stroke volume. Inotropy is therefore central to haemodynamics.

In adults with septicaemia the cardiac index is often high, being greater than 5 L/min/m^2 during the phase when SVR is low. In the Bathurst Base Hospital series, the highest value we have seen was a cardiac index of 9.1 L/min/m^2 in a patient with an SVR of 181 dyne.s.cm^{-5}. As SVR increases in response to vasopressors, the CI can fall dramatically, and can fall to below 2.5 L/min/m^2 well before the SVR reaches normal (around 800-1000). In short, vasoconstriction is precipitating cardiac failure. The ventricle has insufficient inotropy to maintain a normal stroke volume in the face of anything approaching a normal afterload.

The degree of myocardial depression in septicaemia ranges from around 25 to 60% in adult patients, but myocardial depression may not seem obvious when the cardiac output is 15 L/min! In these patients the ejection fraction can be 90%, but it does not mean that inotropy is high, far from it.

This shows the misleading nature of ejection fraction as an index of inotropy, implying that vasodilators have a positive inotropic action as stroke volume and ejection fraction increase with their use, while noradrenaline, which often leads to a lower stroke volume and ejection fraction, would appear to be a negative inotrope! Clearly we need to find a better way to assess inotropy.

Sophisticated echocardiographic techniques can give us some guidance and wall tension/stress and maximum acceleration indices are often used, but this requires considerable skill at echocardiography and is not practical in the acute setting of the ED or ICU, let alone out-patients or the doctor's office. In a way, it is rather like analyzing the movements of the pistons and other components in the motor of a car to estimate the vehicle's on-road performance. If you're a Ferrari race engineer then it is possible, but very, very difficult.

We can turn this around though and say that if we measure the vehicle's on-road performance we can deduce a lot about the power of the motor. How fast can it go on the flat? What speed can it maintain on a gradient? These are all direct functions of power. The power generated by the motor determines how fast a vehicle can climb a hill or its top speed on the flat. The power generated by the motor appears as **potential energy** and **kinetic energy** in the vehicle.

Basic Science.

Consider this photograph…

Every time this child moves the pump handle through a full sweep she will generate one stroke volume of the pump. She will produce this stroke volume at a hydrostatic pressure and at a flow velocity which is determined by the force on the pump handle. But what if her father were to operate the pump instead? He would still produce the same stroke volume, but he would produce it in a much shorter time and with a higher pressure and flow velocity than his daughter could manage. Clearly the difference is due to the power that he possesses relative to his daughter.

(Photograph by kind permission of Oxfam – Donations can be made online!)

Haemodynamics is just the same. We can deduce the power of the heart by measuring the same variables as in the pump example here. We need to know stroke volume, hydrostatic pressure developed, flow velocity, fluid density (to calculate the mass of liquid pumped) and time taken to eject one stroke volume – the systolic flow time. Contraction of the myocardium follows the "all or nothing rule", it will contract with all the power that it has at that moment in time, and that depends on its **inotropy**.

We developed a formula derived from basic haemodynamic theory to calculate the external work done by the heart in a single beat. Power is the capacity to do work and is defined as the work produced divided by time taken, or work per unit time.

To calculate myocardial power or inotropy we need to measure the potential and kinetic energy developed by the heart, which is the external cardiac work, and then divide this by the flow time, the time taken to do this much work. Potential Energy is the energy used to produce blood ***pressure***. Kinetic Energy is the energy used to produce blood ***flow***.

Potential Energy is the product of change of pressure and the change of volume or $\mathbf{PE = \Delta P \times \Delta V}$. The change in pressure, **ΔP**, is the mean blood pressure – CVP, or the pressure of the blood coming out of the heart minus the pressure that the blood came in to the heart.

Kinetic Energy for any moving mass is given by the formula $\mathbf{KE = \frac{1}{2}mV^2}$, where m is the mass of the object and V its velocity.

Applying this to the heart, where the mass of blood is SV x Density, we get

Inotropy (Watts) = P.E./Flow Time + K.E./Flow Time

$$= \frac{\mathbf{BPm \times SV \times 10^{-3}}}{\mathbf{7.5 \times FT}} + \frac{\mathbf{D \times SV \times 10^{-6} \times Vm^2}}{\mathbf{2 \times FT}}$$ **(The Smith-Madigan formula)**

Where BPm = (mean arterial pressure – central venous pressure) in mmHg, SV = stroke volume in ml, D = density, Vm = mean velocity, FT = systolic flow time. The factors 7.5, 10^{-3} and 10^{-6} are required to convert mmHg and ml to kPa and m^3 to conform to SI values. The unit of inotropy is therefore the Watt, the SI unit of power.

Except for BP and blood density which is calculated from the haemoglobin concentration, other variables are measured directly by the USCOM. The USCOM measures the velocity of blood flow every 10 milliseconds during systole. In a typical ejection time of 380ms we have 38 measures of velocity from which we can derive the mean flow velocity. (In fact the

USCOM uses mean velocity to calculate Pmn, the pressure gradient across the valve, from the formula Pmn = 4 x V^2.)

Inotropy can be calculated using the USCOM data and the "Inotropy 2009" computer program. The program simply plugs the data into the formula above to calculate inotropy. It also derives the Smith-Madigan Inotropy Index (SMII) by dividing the total inotropy value by the body surface area of the subject (which is also calculated by the USCOM) just as we do with cardiac index. The most recent software updates for the USCOM do all this for you – talk to your dealer if yours doesn't do it yet.

Clinical studies.

So much for the theory, does it actually work? We looked at both normal healthy patients and patients with LVF to see. Normal patients have an SMII of 1.6 – 2.2W/m^2. Patients with cardiac failure have SMII values ranging between 0.4 and 1.0W/m^2. This should come as no surprise. When we looked at the Frank-Starling curve above, the difference in stroke volume between the normal curve and the heart failure curve was about three-fold. Severe heart failure patients have an SMII of about one third of normal, at around 0.6W/m^2.

Age v Inotropy.

In healthy subjects, younger patients have higher SMII values. Kids between 3 and 15 years of age have an average SMII of 1.92 W/m^2. In subjects between 16 and 35 the average SMII was 1.87 W/m^2 whilst for subjects over 50 years the figure was 1.68 W/m2. So guess what? Your heart gets weaker as you get older!

It seems that the approach of assessing cardiac power by "looking backwards" from the point of view of the circulation is valid. What's more, the circulation doesn't know or care why the heart is not delivering enough PE and KE to it. It matters nothing whether the problem is myocardial infarction, valvular disease, arrhythmias or anything else.

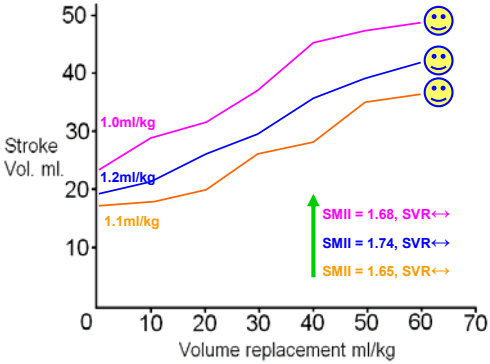
The circulation is a bit like a young child sitting at the dinner table. He doesn't care if the farmer didn't plant the crop, the supermarket was shut, the car broke down or mom forgot to go shopping. All he knows is that he's hungry and will make his displeasure known in an all too obvious way! Whatever the problem, if the heart can't deliver enough PE (blood pressure)

and KE (blood flow) to the circulation then the heart has failed, period. SMII shows us the magnitude of the problem.

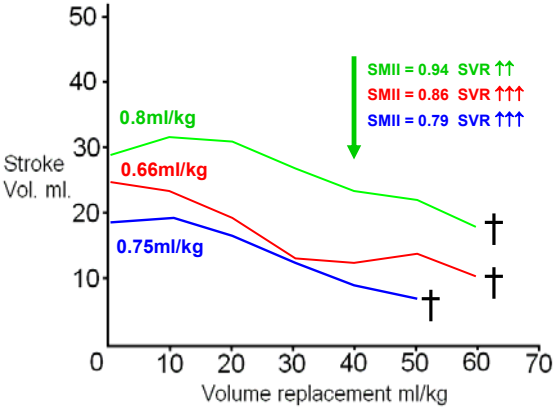
Use of Inotropes.

OK, so now we can measure inotropy, can we use it to treat patients appropriately? Well yes, we can. Let's look at some examples of septic shock in children.

The figure below shows the stroke volume (expressed as ml/kg) and the SMII after 40ml/kg volume resuscitation, for three patients who were typical "fluid responders".

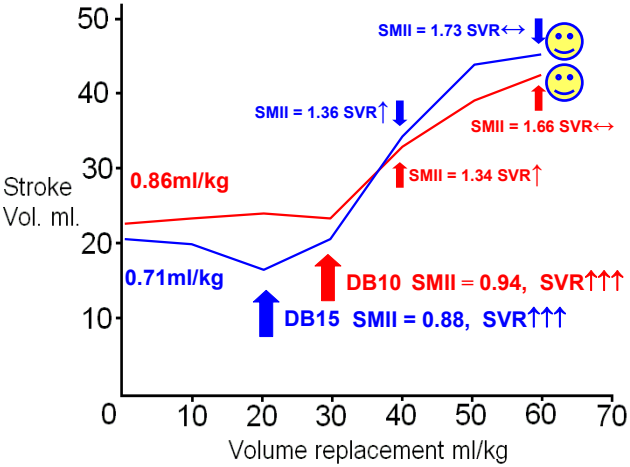


In contrast, three children failed to show clinical or haemodynamic improvement with volume resuscitation. Their traces are shown below.



These patients had stroke volumes at presentation below 1ml/kg and had an SMII of less than 1.0W/m^2 after 40ml/kg of fluid. Both SV and SMII are therefore showing clear evidence of heart failure. The CI for each patient was 2.3 L/min/m^2 (green), 2.1 L/min/m^2 (red) and 1.9 L/min/m^2 (blue). At 60 ml/kg, the CI values were 1.60 and 1.35 L/min/m^2 respectively (child three did not reach 60ml/kg). All had SVRI values greater than 2,800 at 40 ml/kg and for the two who reached it, over 3,600 at 60 ml/kg.

The following figure shows the clinical course of two further patients who initially appeared to be non-responders to fluid alone, and their response to dobutamine at 10 and 15 mcg/kg/min respectively. Both children survived.



In the first case (red line) there was no significant increase in stroke volume after 30ml/kg. The SMII was 0.94W/m^2 and dobutamine was commenced at 10mcg/kg/min. At 40 ml/kg and 60 ml/kg the SMII was 1.34 and 1.66 respectively.

In the second case (blue line) the SMII after 20 ml/kg was 0.88 W/m^2 and dobutamine was commenced at 15mcg/kg/min. At 40 and 60 ml/kg the SMII was 1.36 and 1.73 respectively.

The CI values were 2.35 and 2.15 L/min/m^2 respectively for each patient prior to the commencement of dobutamine. After 60 ml/kg, with dobutamine still infusing, the figures were 4.4 and 4.7 L/min/m^2 respectively.

From the above it would appear that septicaemic shock in these particular children presents with a low stroke volume (and consequently low CI) which may be due to inadequate preload and which responds to volume challenge, or from a combination of low preload and myocardial depression which may not respond to fluid alone. In effect, the non-responders appear to be on a lower, flatter Frank-Starling curve.

If we want to increase stroke volume when there is enough preload but in the face of cardiac failure, then we have to use inotropes. But which one?

As both of the non-responders who received dobutamine had very high SVRI values of 3,300 and 3,450 respectively, a vaso-dilating inotrope seemed a logical choice; hence dobutamine was selected in preference to noradrenaline or dopamine. Other cases may well show a different pattern of SVRI, more like the low vascular resistance/high cardiac output cases often seen in adult septicaemia, where a vasoconstricting inotrope would be a more appropriate choice, but a logical choice of inotrope can always be made on the basis of the CI, SMII and SVRI.

We should ask three questions:

1). Is the CI high, low or normal?
2). Is the SVRI high, low or normal.
3). Is the SMII normal or low?

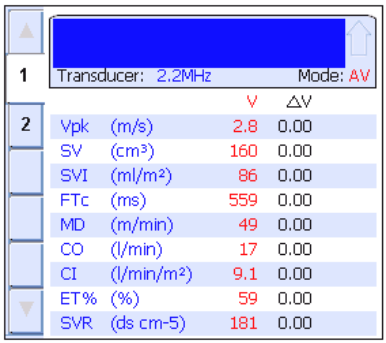
More simply, do we need to increase inotropy, and do we need vasodilation or vasoconstriction? There is no need for, or justification for, empiric treatment. We can measure the necessary variables. The only problem is can we achieve the result we want with the use of a single agent? Unfortunately, while the answer is often yes, frequently it is no. In these cases we have to use balanced inotropes.

Balancing Inotropes.

Let's look at an example of severe septicaemia. As you've probably gathered by now, septicaemia really is the "happy hunting ground" of haemodynamics!

This 57 year old, 87kg male presented to ED with a BP of 72/44 and a history of collapse at home.

His pulse rate was 136, his temperature 38.8, white cell count was 26,000 and his PaO_2 was 64mmHg and his SpO_2 94% on approximately 80% inspired oxygen. He was treated in ED by "aggressive fluid resuscitation" in the form of normal saline 4 litres over 80 minutes. His BP rose a little to 76/47, and his pulse rate fell to 106. Here is his initial USCOM result. What do you see?

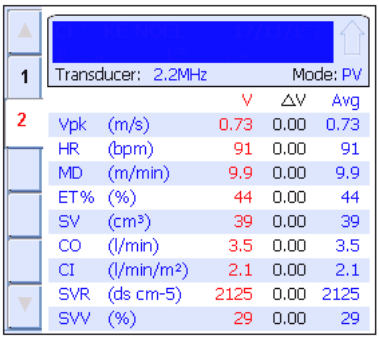


The most striking features are a CO of 17 L/min, CI of 9.1 L/min/m^2, SV of 160ml and an SVR of 181! In addition, the Vpk shows that blood is being ejected at high velocity from the ventricle. Looks pretty convincing for septicaemia doesn't it! It is hard to imagine that with a CI of 9.1 this man could have cardiac failure, but his SMII was just 0.63 W/m^2 (normal 1.6 – 2.2 W/m^2). His high CO and SV are possible only because his SVR is so low. His DO_2 was 3,104ml/min, or over twice the normal value for a man of his size (1,300ml/min or about 15ml/kg).

So how do we treat this? Clearly he needs vasoconstriction in the form of a vasopressor to raise the SVR and thereby the BP, but does his heart have sufficient power to handle an increase in afterload? Let's see. He was treated with an infusion of noradrenaline 100ng/kg/min which increased his BP to 97/58. The infusion rate was increased to 200ng/kg/min. His BP rose to 114/62.

Hey, looks like we've solved the problem, his BP is normal again. But by now you know that this is a dangerous conclusion because blood pressure does not mean blood flow. We don't believe anything until we see the haemodynamic figures. The registrar writing "haemodynamically stable" in the notes on the basis of BP and pulse alone is not going to convince us budding haemodynamicists!

Here is his second USCOM.

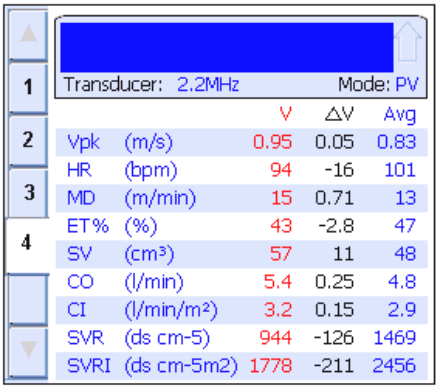


		V	ΔV	Avg
Vpk	(m/s)	0.73	0.00	0.73
HR	(bpm)	91	0.00	91
MD	(m/min)	9.9	0.00	9.9
ET%	(%)	44	0.00	44
SV	(cm³)	39	0.00	39
CO	(l/min)	3.5	0.00	3.5
CI	(l/min/m²)	2.1	0.00	2.1
SVR	(ds cm-5)	2125	0.00	2125
SVV	(%)	29	0.00	29

The noradrenaline certainly increased his SVR. It is now well above 1600, the upper limit of normal. His CI has fallen from 9.1 to 2.1, a figure which is clearly inadequate, given that cardiac failure is defined as a CI at rest of <2.4 L/min/m^2. His DO_2 has plummeted from over 3,000ml/min to just 652ml/min, almost exactly half the normal value. Reducing his noradrenaline caused his BP to fall back to 93/57. So what can we do?

Well let's look at his inotropy index. On this second reading his SMII had increased to 1.13W/m^2, better but still well below normal. We need more inotropy, but we cannot vasoconstrict him any further, in fact we need to allow him to vasodilate somewhat. The answer is to add in dobutamine. This will increase his inotropy to increase CO, and even though we allow his SVR to fall a little, as BP = CO x SVR a bigger gain in CO would outweigh a smaller fall in SVR, so we could still make a profit on the deal.

This is his fourth USCOM result. At this stage he is on noradrenaline at 200ng/kg/min and dobutamine 8mcg/kg/min. His BP is 122/66.



		V	ΔV	Avg
Vpk	(m/s)	0.95	0.05	0.83
HR	(bpm)	94	-16	101
MD	(m/min)	15	0.71	13
ET%	(%)	43	-2.8	47
SV	(cm³)	57	11	48
CO	(l/min)	5.4	0.25	4.8
CI	(l/min/m²)	3.2	0.15	2.9
SVR	(ds cm-5)	944	-126	1469
SVRI	(ds cm-5m2)	1778	-211	2456

Job done! The CO and CI are normal. The SVR is normal. The BP is normal. The DO_2 is 1018ml/min. This is still a little lower than we might like, but certainly adequate and 56% better than our second reading. His SMII was 1.48W/m^2, not quite normal, but much better than the first or second readings. In fact we increased his dobutamine to 10mcg/kg/min, which increased SMII to 1.56, which increased his CO to 6.1 and his CI to 3.6. His SVR fell a little to 736, and his BP remained stable at 124/62. But what happened to his DO_2? This increased to 1,162ml/min, not quite perfect, but near enough!

Balancing inotropes, or even using one single inotrope to its best effect, is a desperately difficult clinical skill to learn. By using the USCOM and SMII the job becomes a case of "painting by numbers". There is still some skill and wisdom involved, but it is so much easier than clinical acumen alone, and there is absolutely no guesswork involved!

Fluid Resuscitation Alone.

But, I hear you ask, would fluids alone have been able to get the job done? It certainly wasn't looking very promising after 4 litres of saline was it?! If we consider his initial SMII value, then he must be on the lower, flatter Frank-Starling curve. As such, increasing preload with extra fluid will give us a minimal increase in stroke volume at best, and risk overloading the ventricle with a fall in stroke volume at worst. Is there a point where inotropy is sufficient to give us a good chance of resuscitation with fluid alone? The answer is yes, and appears to be when the SMII is greater than 1.2W/m^2. Below this value the response to fluid alone is disappointing and inotropes are usually needed.

By the way, did you spot that his MD changed from hyperdynamic at 49m/min to hypodynamic at 9.9m/min and then up to normodynamic at 15m/min and finally up to 17m/min after the dobutamine was increased to 10mcg/kg/min? (Normal range in adults is 14-22m/min.) This leads us to another area where SMII can be very useful, the ratio between potential energy and kinetic energy, the P:K ratio or PKR.

Potential to Kinetic Energy Ratio, PE:KE Ratio - PKR.

Potential energy gives us blood ***pressure***. Kinetic energy gives us blood ***flow***. As with so much in haemodynamics, the balance is important. Pressure is meaningless unless there is flow, but flow alone can't maintain the vital organs. We need both in adequate amounts.

In the septicaemic example above, we started with high flow but low pressure. We then had good pressure but low flow after the noradrenaline. Finally we achieved adequate pressure ***and*** flow. The PE:KE ratio (PKR) is very informative here. The normal ratio is around 30:1, so far more of the ventricular power goes to generating blood pressure rather than flow. At the time of the first USCOM reading the PKR was just 3:1, plenty of flow but not much pressure. At the second USCOM reading the ratio had reversed to 64:1, enough pressure but inadequate flow. By the third reading the PKR was down to 42: 1. We're getting close to the right balance. Finally, after the increase in dobutamine to 10mcg/kg/min the PKR was 33:1, close enough!

In arterial hypertension due to excessive vasoconstriction (as opposed to excessive cardiac output) the PKR is in the range of 60:1 to 150:1. Appropriate therapy with vasodilating medication such as ACE inhibitors or calcium channel blockers can reduce the PKR to near normal. Conversely, in hypertension due to excessive cardiac output, the PKR is around 10-15:1, and increases with appropriate therapy, such as a β blocker, back towards normal. Not only do we have a simple tool to identify the underlying cause of the hypertension, we also have a method of optimizing therapy other than just looking at the BP with no regard to blood flow. How cool is that?

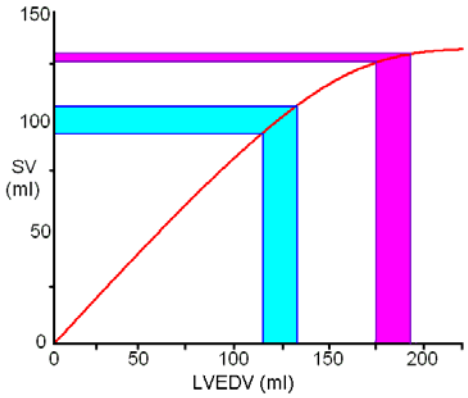
Measurement of Preload.

So by now you might be thinking that the USCOM, SMII and PKR have taken us into areas of haemodynamics that we never thought we should see at the bedside. You're dead right, but we haven't finished yet!
What is not yet clear from our simple view of haemodynamics in the examples given above, is do we need to increase the preload further? There are several clues to the answer. If we are still on the under-loaded side (left side) of the Frank-Starling curve then further volume challenge will lead to an increase in stroke volume. If it doesn't, then we are probably at or near the peak of the curve, or on a flat curve, but SMII will differentiate which it is. A flat curve will have a low SV and SMII, while an under-loaded patient will still have a low SV but more normal SMII (=>1.2W/m^2). Can we make this even easier?

Stroke Volume Variation SVV.

SVV has been used as an index of ventricular filling. The concept is relatively simple. As the intrathoracic pressure changes with respiration, so venous return to the heart will change in a cyclical way. Increased intrathoracic pressure will lead to reduced venous return and reduced ventricular filling. This will lead to a fall in SV.

An under-loaded patient will show a greater variation in SV than a well filled patient. If we look at a typical Frank-Starling curve we can see why.



In an under-loaded patient (blue band) a 20ml change in LVEDV will lead to about 15ml variation in SV as we are on the left side of the peak in the Starling curve. If we increase preload, then the same 20ml variation in LVEDV will result in only 5ml change in SV (purple band). If we increased preload even further till we reach the plateau of the Starling curve then SVV would be imperceptible.

There is a problem with this approach however. If the change in intrathoracic pressure is small, as in normal quiet breathing, then the change in LVEDV will also be very small and a very sensitive method is required to pick up the variation in stroke volume. Fortunately, Doppler ultrasound is extremely sensitive and even minor changes in SV can be tracked by the USCOM. With positive pressure ventilation, the SVV should be more marked due to the greater changes in intrathoracic pressure and therefore in LVEDV.

Passive Leg Raising.

In either case, a simple trick is to measure the SV and then raise the patient's legs to increase venous return. (N.B. the patient must not assist you in this, you or a helper have to do the work.) In an under-loaded patient the SV will increase whilst the SVV will decrease. If this is the case, then a further fluid challenge can be made. The leg-raising trick can be repeated as often as necessary. If the SV does not increase then we are near the top of the curve. If the worst case happens and the SV should fall, then we have an overloaded patient, but no harm has been done. We can just lower the legs again and go back to where we were. It's not so easy to do that with an i.v. fluid bolus challenge!

Corrected Flow Time FTc.

Corrected flow time (what the flow time would be if the heart rate were 60/minute, rather like QTc in the ECG) can also give us some guidance as to fluid loading, as a low figure is found in hypovolaemia. The reason for this is that the heart contracts at a force mainly determined by its inotropy and preload. For any given inotropy level, the time the heart takes to eject a stroke volume depends on how large that stroke volume is, which in turn depends on how full the ventricle was at the end of diastole. A low LVEDV leads to a low SV and a short flow time, whilst a high LVEDV leads to a high SV and a long flow time. Simple! Well, most of the time in healthy subjects, FTc and LVEDV are pretty close.

If inotropy is fairly normal then this relationship between preload and FTc holds, but it's actually not quite so simple once we start looking at sick patients with lower SMII values.

The main problem with this concept is that the flow time will, as mentioned above, depend on inotropy. As inotropy increases the flow time will become shorter, the ventricle will eject more rapidly. Inotropy changes with preload, increasing as preload increases, so the relationship between FTc and LVEDV is far from linear. It is often claimed in transoesophageal Doppler studies that FTc is a reliable indicator of preload, but clearly this cannot be true if inotropy is not constant. The other big problem is that flow time will also depend on afterload. The higher the afterload the higher the flow time will become as the heart has to work harder against the back pressure from the aorta. This can lead to a change in inotropy which can be either positive or negative depending on the exact pressures and ventricular size, and the resting inotropy. The relationship is clearly not simple. A high FTc may indicate a high preload but is also found in cardiac failure (well OK, we could say that that represents a high preload too) but either way, it suggests that more fluid is not wise.

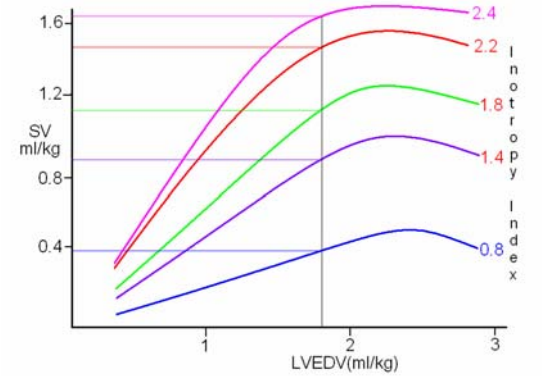
Left Bundle Branch Block.

With LBBB the ventricle depolarizes abnormally and invariably leads to a prolongation of systolic flow time. Under these circumstances can we still use FTc as an indicator of preload? The short answer is no, but it may still give us some guidance in therapy by looking at the response to preload *changes*. This is far from easy however, and it is probably better not to use FTc if the ECG shows LBBB.

FTc and SVV can therefore be regarded as useful guides to preload but must be interpreted in the light of the full clinical and haemodynamic picture. If only we could measure or calculate the true LVEDV then the problem would become a lot more straightforward. SMII can help us here.

SMII and LVEDV.

Let's go back to the Frank-Starling curve below and think backwards.



This figure shows five different Frank-Starling curves for five different levels of inotropy. For a given LVEDV of 1.8ml/kg, there are five possible stroke volume figures depending on the inotropy status. The gradient of the Starling curve is, in effect, the ejection fraction, being the stroke volume divided by the LVEDV. Indeed this is where the concept that ejection fraction equates to inotropy comes from. Therefore, if we know the gradient from the SMII, and we know the stroke volume, then the LVEDV can be calculated fairly simply.

For a 70kg patient with a stroke volume of 85ml and an SMII of 1.6W/m^2 (green line) the LVEDV would be 126ml. His ejection fraction would be 85/126 or 67.5%. For the same SMII and a stroke volume of 56ml his LVEDV would be 91ml.

In the same way as we use cardiac index to compare cardiac output in individuals of varying size, so we can use left ventricular end diastolic volume index, LVEDVI, to "standardize" the normal LVEDV. In practice this turns out to be around 65 - 85ml/m^2. A figure below 65ml/m^2 indicates inadequate preload, while values above 85ml/m^2 are seen in fluid overload and LVF. In clinical practice, SMII-derived LVEDVI correlates very well with echocardiographic measurements of LVEDV. One big difference however is that echocardiography takes 20 - 30 minutes to do properly, even in the hands of an expert echocardiographer; the SMII-derived LVEDVI takes about 2 minutes, and you can do it!

Limitations of this method.

The above makes the very important assumption that the heart is structurally normal. If there is significant valvular disease such as aortic stenosis or regurgitation then the true LVEDV will be higher than predicted. Similar problems relate to mitral valve regurgitation, VSD with left to right shunt and ventricular aneurysm or large akinetic segments of the ventricular wall. These should be excluded, or at least allowed for when estimating LVEDVI.

The calculation of LVEDV from SMII is still undergoing evaluation and validation and certainly appears to be valid in most cases, but results should be treated with some caution until validation has been completed and limitations of the method identified. Any feedback on this would be gratefully received! The calculation of LVEDV from SMII is included in the "Inotropy 2009" software.

Stroke Volume and Preload.

Perhaps the most obvious fact about preload however is the simplest. If SMII is anything like normal, and afterload is even in the right ballpark, then stroke volume is a direct function of preload. If SV is low (see normal values) then preload is low, and SV will rise and fall almost linearly with preload.

Similarly, if preload remains fairly stable then any increase in SV must be due to either an increase in inotropy, or a fall in afterload. The clinical situation should make it obvious which one has occurred in most cases.

Conclusion.

OK let's put it all together. What do we need to know to get the full picture in haemodynamics? The table below shows all the parameters we need, along with which can be obtained using the USCOM and the Inotropy 2009 software.

Parameter	Indicator	Shown by USCOM	Inotropy 2009
Preload	SV, SVV, FTc.	☑	
	LVEDVI		☑
Afterload	SVR	☑	
	BP mean	☑	
Inotropy	Vpk, SV, FT,	☑	☑
	Inotropy, SMII	*	☑
Cardiac Output	CO, CI, SV, HR	☑	
Oxygen Delivery	CO, SpO_2, DO_2	☑	☑
Oxygen Usage	VO_2		☑
Haemoglobin	Hb	*	
PE:KE Ratio	PKR	*	☑

(* = not yet, but watch this space!)

It's not hard to see why the USCOM has been called the "Swiss Army Knife" of haemodynamics!

With the simple tools outlined in the three companion booklets in this series you should be able to handle just about any haemodynamic disturbance that medicine can throw at you. What's more, you can do it quickly, non-invasively and without any guesswork.
The days of "let's try it and see" or "it could be anything, cardiac, pulmonary, who knows?" have gone. We can now take control of the situation and base our therapy on what we know will happen, not on what we hope will happen. Uncross your fingers and let's do some third millennium haemodynamics instead of third rate medicine!

Appendix 1 - Inotropy 2009 Software Installation.

If you run the "Setup" program it will install the software on to any windows-based computer from windows 97 through 2000, ME, XP and Vista.

The program needs your computer to have the Microsoft .NET Framework 3.5 installed. You can download this from the Microsoft Website, but the setup program will automatically check to see if your computer has this and if not, it will go to the website and download / install the .NET Framework 3.5 files for you. This can take up to 20 minutes or so, depending on the speed of your internet connection and how busy the Microsoft site is.

When .NET Framework 3.5 has been installed it will ask you to restart your computer to finish the installation / configuration. If you now run the Inotropy 2009 setup program again, it should install in just a few seconds. You may get warnings from the security software on your computer asking if you want to trust this unknown source - click “accept”.

If you now click “Start” then “All Programs” Inotropy will appear as the last in the list. From here you can either run the program or make a desktop icon if you wish.

I would recommend that you create a new directory / folder on your hard disk (C drive) called “Inotropy 2009” and save the downloaded Inotropy2009.zip file into this folder. The zip file can be unzipped (if it hasn't done so automatically) into the same folder and the setup program run from there.

The reason that I used the .NET Framework 3.5 is that ultimately the inotropy program can then run on PDA's, handheld devices like Blackberry and even on smartphones as well as computers, which is why the program installs as "Inotropy Mobile".

As ever, feedback from users would be greatly appreciated.

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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	**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**
30	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
days	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	**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**
12	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
mths	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
	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**
1	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
	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**
2	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
	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**
3	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
	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**
4	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
	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**
5	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
	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**
6	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	

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 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
	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**
7	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
	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**
8	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
	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**
9	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
	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**
10	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
	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**
11	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
	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**
12	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	**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**
to	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
16	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	

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 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	**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**
30	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
days	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	**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**
12	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
mths	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
	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**
1	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
	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**
2	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
	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**
3	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
	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**
4	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
	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**
5	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
	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**
6	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
	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**
7	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
	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**
8	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
	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**
9	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
	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**
10	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
	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**
11	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
	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**
12	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	**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**
to	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
16	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	

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