

The USCOM in Clinical Practice

A Guide for Junior Medical and Nursing Staff



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Introduction.

If you've read the first booklet in this series "The USCOM and Haemodynamics" then you may be getting the idea that the USCOM is an exciting new tool in clinical medicine. What this booklet aims to do is to show you just how you can use the USCOM in your clinical practice and just how the USCOM can help the treatment of your patients. No matter what area of medicine you practice in, there is probably a suitable application where the USCOM can improve the way you currently practice and improve the outcomes for your patients. Although this booklet is structured according to the hospital departments where the USCOM has been used successfully, it is probably worth reading those other areas outside of your own department too, as this will give you both help in interpreting USCOM findings and perhaps some new ideas for uses of your own.

When interpreting the data produced by the USCOM it is always worth recalling that very seldom in clinical medicine is a diagnosis based on one single finding or lab result. On the contrary, it is usually a pattern of clinical findings and other data which supplies the diagnosis. When looking at any USCOM examination, you should always look at the full data output and try to visualise just what is happening in the cardiovascular system as a whole. The heart does not work in isolation, nor does the peripheral vascular system exist without the heart and the connecting arteries. Visualising the circulation as one entire functional unit will tell you so much more than inspection of just one parameter. You should also remember that the prime function of the heart and circulation is to deliver oxygen to the tissues. Whenever you look at an USCOM evaluation it is always worth thinking about the level of oxygen delivery, DO_2 , to the tissues.

So let's get down to some real clinical cases and show you just how powerful the USCOM can be!

USCOM in the Emergency Department.

Rapid evaluation of haemodynamics is carried out in every emergency department in the world every single day. In the main however, this usually consists of looking at some general parameters such as blood pressure, pulse rate and perhaps oxygen saturation. Some clinical evaluation of perfusion may also be made, but how much better would it be if we knew exactly what the haemodynamics were doing. Because of the non-invasive

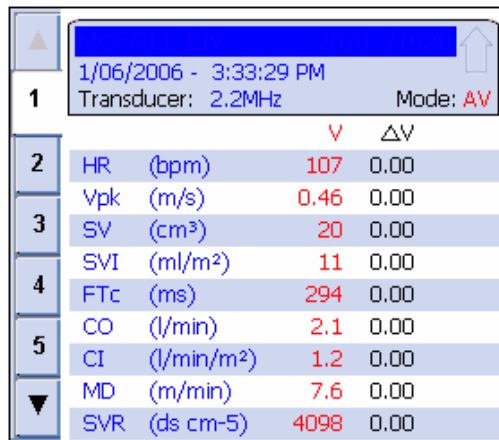
nature of the USCOM, and the speed with which such data can be acquired, the USCOM is beautifully suited to the emergency environment. Let's take a look of a couple of cases that presented in our own emergency department and see just how the USCOM improves clinical management of the patient.

Clinical Case 1.

Male, 68 years old, 76kg. Acute onset of severe central chest pain and dyspnoea 40 minutes prior to admission. He had a past history of hypertension and angina. ECG shows ST elevation antero-laterally.

Observations: BP 96/53, pulse 108, Resp Rate 32, JVP clinically elevated. SpO₂ 86% on 10 l/min O₂. He was confused and agitated. Arterial gas analysis showed PaO₂ 52, PaCO₂ 28, pH 7.18, Lactate 18. CXR showed florid changes of pulmonary oedema bilaterally.

This is the USCOM data screen. What can you see?



The screenshot shows a USCOM data screen with a blue header bar containing the date and time '1/06/2006 - 3:33:29 PM', the transducer frequency '2.2MHz', and the mode 'AV'. Below the header is a table of hemodynamic parameters. The table has three columns: parameter name and units, current value, and a change value (ΔV). The current values are in red text, and the change values are in black text. The parameters listed are HR (bpm), Vpk (m/s), SV (cm³), SVI (ml/m²), FTc (ms), CO (l/min), CI (l/min/m²), MD (m/min), and SVR (ds cm⁻⁵).

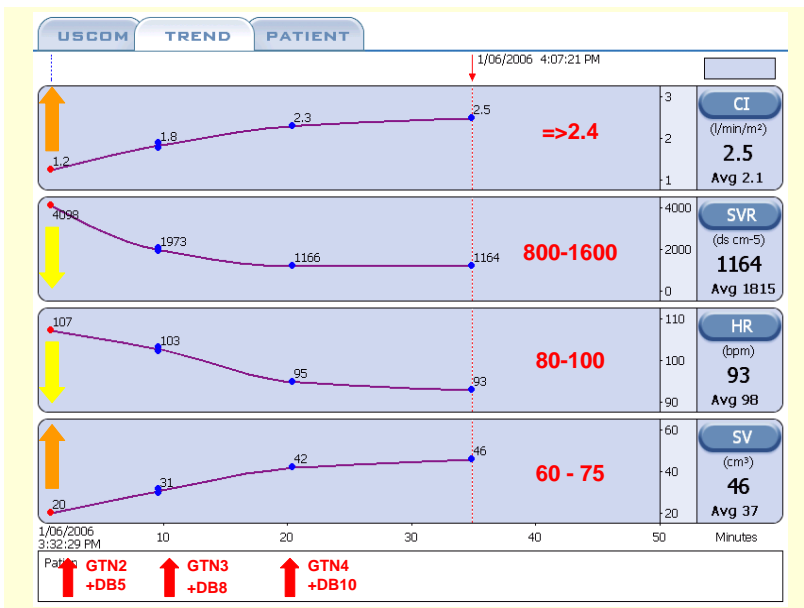
		V	ΔV
1	1/06/2006 - 3:33:29 PM Transducer: 2.2MHz		
2	HR (bpm)	107	0.00
	Vpk (m/s)	0.46	0.00
3	SV (cm ³)	20	0.00
	SVI (ml/m ²)	11	0.00
4	FTc (ms)	294	0.00
	CO (l/min)	2.1	0.00
5	CI (l/min/m ²)	1.2	0.00
	MD (m/min)	7.6	0.00
	SVR (ds cm ⁻⁵)	4098	0.00

The most obvious finding here is that his cardiac output and cardiac index are both low. The minimum cardiac index we should look for in a patient is 2.4 l/min/m². Clearly this patient comes nowhere near this. His stroke volume is low, his peak velocity is low, and his SVR is markedly raised. What's going on here?

His myocardium is incapable of producing an adequate stroke volume, and the low peak velocity suggests a very low myocardial contractility (inotropy) status. His peripheral circulation is responding to the low cardiac

output by vasoconstriction, giving him a high SVR of nearly four times normal. As a result of this, the blood flow in his aorta is much slower than normal as indicated by his MD (the normal is 14-22 metres/minute). It's clear that this is a hypodynamic circulation.

But what is actually killing the patient? On a superficial level we might answer "cardiogenic shock" but what do we actually mean by this? We know that shock is "any haemodynamic derangement leading to inadequate perfusion and oxygenation of the tissues". So what is this patient's oxygen delivery? We know from "The USCOM and Haemodynamics" how to calculate this. When we plug in the numbers in this case we find that the oxygen delivery is only 372ml/min. For a man of his size, a figure of even twice this value would be only just about enough.



This is the trend screen of the USCOM over the next 35 minutes. The figures in red on the right hand side have been added to show the normal values that we should aim for in a man of this age and size. In effect, these are our early goals in therapy. His cardiac index at 1.2 must be increased; his SVR at over 4,000 must be reduced; his heart rate should be reduced and his stroke volume needs to increase significantly. So how did we treat him?

GTN2 and DB5 refer to glyceryl trinitrate infusion at 2mcg/kg/min and dobutamine at 5mcg/kg/min. Why did we choose these agents? The USCOM shows that his cardiac output is inadequate and from clinical observation and from his chest X-ray it is clear that his preload is already very high. We need to off-load him urgently. Nitrates achieve this more rapidly than anything else. But why did we choose dobutamine? Well he needs one or other inotrope to increase his cardiac index, but in the presence of a low blood pressure many people would opt for dopamine or noradrenaline or even perhaps an adrenaline infusion, but the USCOM shows that this is not appropriate. His SVR is so high that we need to vasodilate his arterial tree (reduce his afterload) if we hope to increase his stroke volume, given that his myocardial contractility is low. The GTN will help a little but the most logical inotrope to use is dobutamine because of its vasodilator properties.

Repeat USCOM shows that his SVR, stroke volume, cardiac index and heart rate are all going in the right direction. The infusions are then increased to 3 and 8 mcg/kg/min. Again, repeat measurement shows that we are making good progress. Finally, the infusions are increased to 4 and 10 mcg/kg/min. Following this, we have achieved our early goal in terms of his overall haemodynamics.

As the time scale on the trend screen shows, the treatment of his cardiogenic shock was achieved in just 35 minutes and this was carried out in the Emergency Department. By the time the patient was transferred to the Coronary Care Unit his immediate problem had already been solved. The importance of diagnosing the haemodynamic problem and treating it appropriately and rapidly is obvious. His vital signs, laboratory results and radiology two hours post admission are interesting.

BP 108/64, pulse 74, SpO₂ 96% (on 4 l/min O₂), CI = 2.8 l/min/m², SVR = 1082, SV = 66ml.

PaO₂ = 93, PaCO₂ = 35, pH = 7.38, Lactate = 1.6

His oxygen delivery is now 926ml/min, an increase of 249%!

Over the next few hours his pulmonary oedema resolved completely.

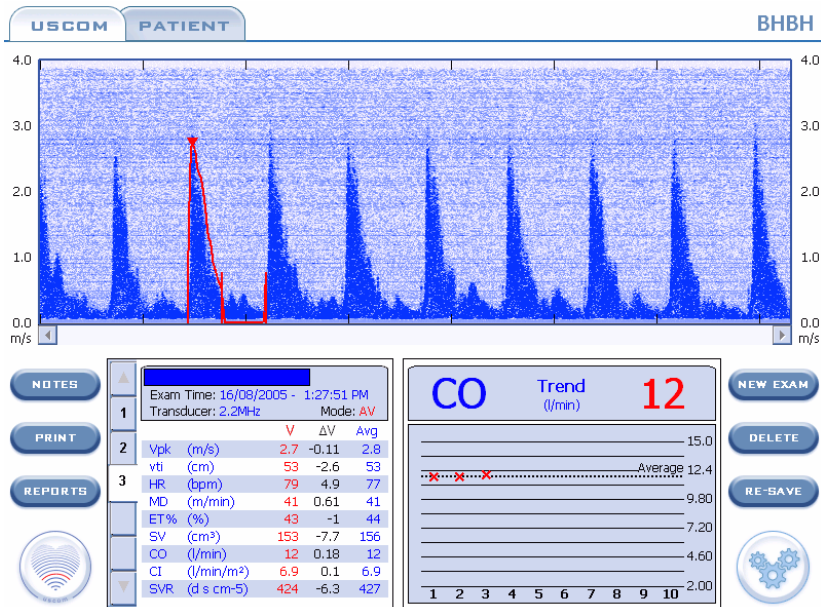
Coronary angiography showed triple vessel disease, not amenable to stenting. He subsequently underwent CABG x 3, on the 7th day after admission. He made an uneventful recovery and was discharged on the 21st day after presentation with no symptoms. At 6 month follow up he remained well with no angina.

Clinical Case 2.

24 year old female 58kg. Previously fit and well. Only medication is oral contraceptive pill. Brought in by ambulance as “collapse”. Patient very confused and little history available.

GCS 5-6, BP 73/42, pulse 80, Temp 38.3, SpO₂ 92% on 4 l/min O₂, Resps 26, Sweaty++, Right calf and foot visibly swollen. CXR - unremarkable. ECG – sinus rhythm. Blood glucose 4.3mM/L.

Initial diagnosis was right-sided DVT with pulmonary embolus. Here is her USCOM. What do you see?



This is indeed an odd pulmonary embolus! Her cardiac output is 12 l/min with a cardiac index of almost 7. It would be a very strange blood clot in the pulmonary artery that allowed such a large volume of blood to flow past it and yet rendered the patient prostrate! The Vpk shows that her myocardial contractility is excellent, her heart rate is not elevated, the MD shows that the circulation is hyperdynamic and she has a stroke volume of around 2.5 ml/kg. So what's going on here? The answer is immediately apparent when we look at SVR. At just 424, this is about one-third of

normal. We are looking at peripheral vascular collapse with a hyperdynamic circulation and high cardiac output.

What is your diagnosis now?

Closer clinical examination revealed an 8 x 5cm patch of cellulitis on the upper inner right thigh with small ischaemic areas within. Inguinal lymphadenopathy was present on the right. The diagnosis must be septicaemia.

She was treated with iv antibiotics, iv fluids, and phenylephrine, an alpha-agonist (vasoconstrictor). Dopamine or noradrenaline would also be reasonably logical choices.

BP increased within 30 minutes to 105/60. She regained full consciousness and was able to report that the patch on her thigh had appeared one day earlier “like an insect bite”. It had increased in size overnight. She had intended to see her GP “later today after work” but had not felt well enough to go to work so went to bed instead. She woke up in ED!

Her temperature settled over 36 hours, vasopressor infusion was required for 18 hours. She needed 4L of fluid in the first 2 hours, but only 3 litres over the next 24 hours. She required 28-35% oxygen for 18 hours until PaO₂ readings stabilised.

Subsequently (4th day) she required skin-grafting of a sloughing lesion of the right thigh for necrotising cellulitis. The infection was confirmed as streptococcus from wound swabs and the same organism was found in her blood cultures. She made a full recovery.

Clinical Case 3.

Female 5 years old, struck by car, multiple trauma.
Conscious, GCS 13. Clinically shocked. BP 65/? , Pulse 165, resps 46/min, SpO₂ 93% on 4L O₂.

Her examination and radiographic findings were:

Fracture - Pelvis (inferior and superior pubic rami, left side)

Fracture - Left femur

Fracture - Right humerus

Fracture - Left radius and ulna

Fracture - 5th, 6th, 7th, 8th ribs right side
 Scalp laceration, right parietal – 6cm, bleeding+
 Lacerations / contusions to both thighs
 Bruising over right upper abdomen
 Abdomen tender and guarding
 In severe pain

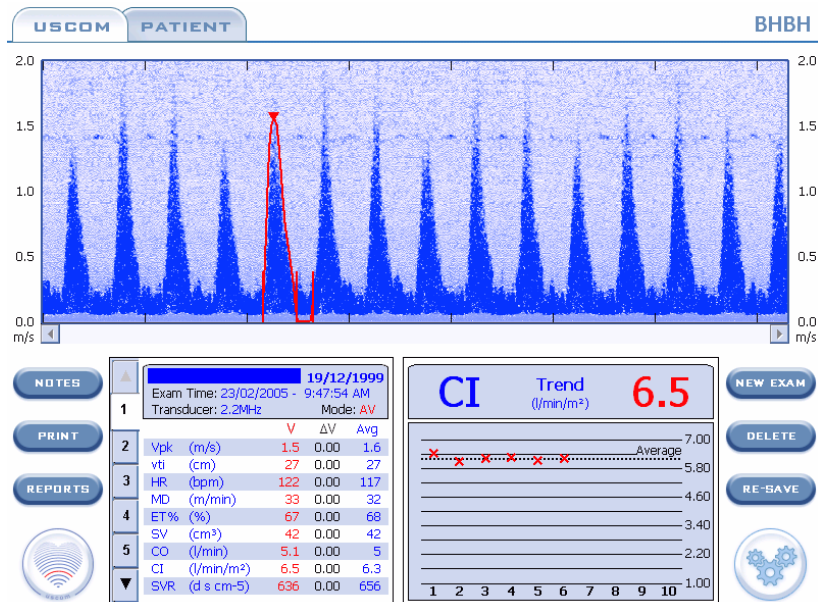
She was given 1L Hartmann’s at scene / during transfer. In ED, she was given N/Saline 1L, Gelofusine 500ml, and packed red cells (red cell concentrate) x 2, Morphine 4mg iv + further 1.5mg.

Her observations after 45 minutes were:

Pulse 124, BP (right thigh) 85/42, SpO₂ = 96% (4 l/min O₂)

The skin felt cool and she felt sweaty to the touch.

Is the patient adequately volume resuscitated?



Peak ejection velocity increases with fluid loading. The normal at 5 years is 1.3 – 1.4 m/s. 1.5-1.6 m/s indicates adequate to excessive fluid loading. It also suggests a vasodilated vascular tree, as vasoconstriction produces back pressure on the ventricle (a high afterload) which limits peak ejection velocity. Mean aortic flow velocity as indicated by the MD is increased

above normal, indicating a hyperdynamic circulation. Again, this suggests excessive fluid loading and vasodilation. The normal stroke volume is 1.5-2ml/kg at this age. A child of this age would typically weigh around 18kg and an SV of 42ml suggests she is “well filled”.

The cardiac index is high and given the stroke volume, is more due to excessive heart rate. The SVR of 636 shows that the patient is vasodilated. This could well be due to the morphine that she has received as morphine is a potent vasodilator. Although the potential exists with these injuries for major blood loss to occur, in fact, the patient is now slightly hypervolaemic rather than hypovolaemic.

When the patient was catheterised, urine output exceeded 3ml/kg for the next few hours. Over the first 24 hours, only 750ml of crystalloid and 1 unit of red cell concentrate were needed to maintain normal haemodynamics and urine output. The haematocrit at 24 hours was 0.41 and Hb 142g/L. The right lung showed patchy contusion, but no major opacification on CXR. Supplemental oxygen at 2 l/min was required for 24 hours, but no ventilatory support.

The child made a full recovery.

It would be extremely easy in a case such as this to over estimate the blood and fluid requirements for the patient. Injuries such as these could lead to virtual exsanguination of the patient if they all produced their maximum possible blood loss. In fact, in this case, the blood loss from the injuries was very much less than one might normally expect. Excessive fluid loading in this patient could well have led to significant pulmonary compromise with respiratory distress syndrome and the need for ventilatory support. Using the USCOM to guide fluid therapy may well have prevented this eventuality, we’ll never know, but it certainly made the clinical judgment of fluid requirements very much easier. Had the blood loss been on a more massive scale, then we could have used the USCOM to detect the hypodynamic circulation due to underfilling and responded accordingly. No more guesswork!

USCOM in the Operating Theatre.

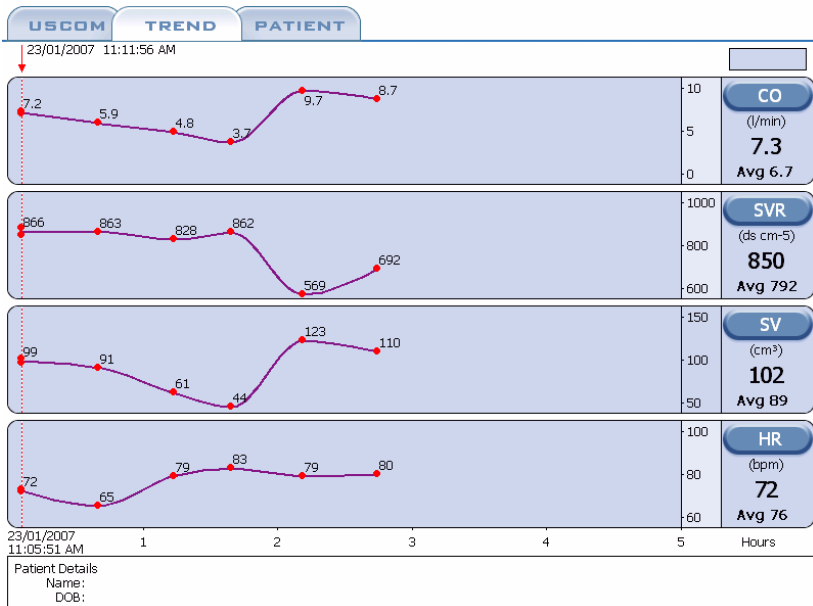
You will have seen the USCOM being used increasingly frequently in the operating theatre, particularly during major cases. Why is this so?

Let’s consider the average patient undergoing major surgery. Firstly, they may be on a host of medications for pre-existing conditions with heart

disease, hypertension and diabetes being particularly prevalent. Patients may well have been given a bowel prep, which can cause significant loss of fluid from the body, as can pre-operative vomiting. Even in an elective case, the patient will have been fasted for many hours or even overnight before their operation. The anaesthetist will then use a plethora of drugs which have significant cardiovascular activity, ranging from myocardial depression to vasodilation. There may be blood loss or evaporative loss of fluid from the open abdomen, there may be antibiotics given intravenously which can have vascular activity, and as if all this wasn't enough, the anaesthetist may well use an epidural or spinal anaesthetic as well. What if the patient has pain, what will that do to their haemodynamics? Given all of the above, it is hardly surprising that major changes in the patient's haemodynamic status occur during surgery.

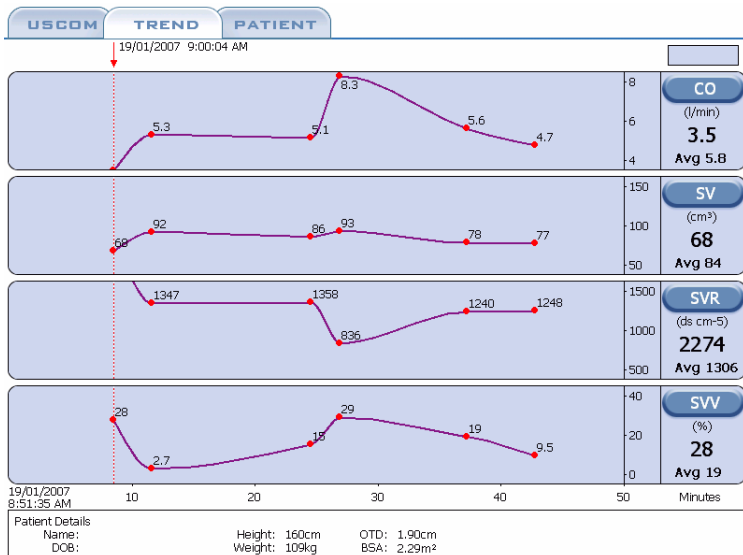
Whilst the measurement of blood pressure, heart rate and pulse oximetry are routine, do these really tell us much about the true haemodynamic picture?

Let's take a look at a typical example of the haemodynamic trends in a patient undergoing left hemicolectomy.



Throughout the procedure, which was carried out under a combination of general anaesthesia with IPPV and a thoracolumbar epidural, the blood pressure and pulse rate stayed within normal limits, whilst pulse oximetry and capnography were also normal. Just look at the changes in the haemodynamics shown in just these parameters! In particular, look at the readings for measurement points 3 and 5, at 1 hour 15 minutes and 2 hours 15 minutes respectively, where the pulse rate is exactly the same at 79. The pulse rate gave no indication whatever of the fact that the cardiac output and stroke volume had more than doubled, whilst the SVR had plummeted. The blood pressure also gave little indication of these profound changes.

The ease of using the USCOM in anaesthetized patients and the data that it provides, gives the anaesthetist a much clearer picture of just what his ministrations are actually doing to the patient, and also tells them exactly which way to go to correct the situation should a problem occur.



This second example is a 24 year-old female with pregnancy-induced hypertension undergoing caesarean section under a spinal anaesthetic. Due to the vasodilation which spinal anaesthesia produces, there can be marked falls in venous return with a consequent drop in stroke volume and cardiac output. As mentioned earlier, the drop in venous return can lead to a fall in myocardial contractility with reduction in the Vpk. In this situation, should we give fluid or should we use a vasopressor, and if so then how much?

Should we use a combination of the two? If we use an excessive dose of vasopressor will this reduce the uterine blood flow with harmful effects on the baby?

About half-way through the case we see an abrupt fall in SVR and a sharp rise in cardiac output. Why was this? In fact this followed the administration of 10 units of syntocinon i.v. Syntocinon causes uterine contraction which we want to occur just after delivery, but it can also produce marked vasodilation. This patient had sufficient cardiac reserve to maintain her blood pressure by increasing her cardiac output significantly, but what if she had pre-existing heart disease and was not able to do this? In this situation we could give the syntocinon in small increments while monitoring her cardiovascular responses. Simple! The same applies to many other cardiovascular drugs in a host of patients. It is so much easier to make progress in small safe steps rather than try to redeem a situation which has already become desperate.

SVV and FTc.

This case also illustrates one other very useful feature of the USCOM which you see when you look at SVV, the stroke volume variation. This is often regarded as a very useful sign of cardiac preload. Although it has its limitations, in general, SVV increases as preload decreases. In a patient with a normal preload, SVV would be below 10% during spontaneous breathing and below 15% during mechanical ventilation at normal inflation pressures. Initially, following the vasodilation produced by the spinal block, the patient has a relative hypovolaemia, which shows as a high SVV. In response to rapid fluid administration, her SVV decreases to a more normal level. However, following the vasodilation produced by the syntocinon, we see an abrupt rise in SVV again indicating a relative hypovolaemia due to the vasodilation.

The USCOM also measures FTc, the corrected flow time. This is an indication of how long systole actually lasts, the correction being to enable different patients to be compared by adjusting the flow time to a nominal heart rate of 60 beats per minute, rather like the QTc of the ECG. If you think about it, the stroke volume that is ejected by the heart, taken together with the FTc, the time it takes to produce a stroke volume, must give a good indication of myocardial contractility. The more powerful the heart, the bigger the stroke volume (higher SV) the faster it will be ejected (higher Vpk) and the shorter the time needed for systole (shorter FTc). Many researchers have found that the FTc gives a better indication of left ventricular preload than does pulmonary capillary wedge pressure (PCWP

or PAWP) or pulmonary artery diastolic pressure (PADP). The pairing of SVV and FTc are now regarded as a better index of total cardiac preload than the CVP and PCWP. The normal FTc ranges from around 325 in children to about 475 in older adults for the aorta and slightly longer at 325 – 500 for the pulmonary artery.

USCOM on the Ward.

One major advantage of the USCOM is that it is readily portable and therefore the USCOM can be taken to the patient rather than the patient coming to the USCOM. There are a large number of haemodynamic problems that may arise in inpatients, from the point of view of diagnosis, from guiding their therapy, and also for explaining unexpected eventualities. Let's take a look at a couple of clinical examples.

USCOM on the Medical Ward.

A 63 year-old man was admitted to the ward with a history of increasing breathlessness over two days. In the past he had suffered a myocardial infarction some four years previously and had been on digoxin and diuretics since that time for treatment of mild to moderate cardiac failure. He also had a history of hypertension for which he was on an ACE inhibitor. In the past he had been a miner and had a long-standing history of chronic obstructive pulmonary disease. His admission observations are shown below.

BP 116/68, Pulse 104, Resps 28. SpO₂ of 88% on 4 l/min O₂ via Hudson mask. Temp 37.1. He was using his accessory muscles of respiration. JVP was clinically elevated. The liver appeared enlarged and slightly tender. Auscultation revealed widespread crackles throughout both lower and mid zones. There were also widespread rhonchi in all areas. CXR showed increased lung markings and a general fluffiness bilaterally. Blood gas analysis showed a pH of 7.28, PaO₂ of 68, PaCO₂ of 32, BE –8.

This is a typical example of the clinical problem of "is it pulmonary or is it cardiac?" If we look at his USCOM results then things begin to make more sense.

1		V	ΔV	Avg
11/05/2007 - 4:37:48 PM				
Transducer: 2.2MHz Mode: AV				
2	Vpk (m/s)	1.1	0.00	1.1
	SV (cm ³)	67	0.00	59
	FTc (ms)	423	0.00	364
	MD (m/min)	23	0.00	18
	CO (l/min)	6.4	0.00	5.6
	CI (l/min/m ²)	3.5	0.00	3
	SVR (ds cm-5)	1310	0.00	1546
	SpO2 (%)	89	0.00	89
	DO2 (ml/min)	1024	0.00	892

His cardiac index is 3.5 which very strongly suggests that this is not cardiac failure. His stroke volume is a reasonable 67ml and his Vpk is 1.1 which again suggests that this is not cardiac failure. His FTc is 423, which is entirely normal, while his MD at 23 suggests a borderline hyperdynamic circulation. There is no evidence of vasoconstriction as his SVR is 1310. It is far more likely, therefore, that what we're dealing with here is a primary pulmonary condition. In fact, over the next 24 hours, he developed typical lobar pneumonia.

Let's look at a second example with a very similar clinical presentation. If we look at the results they are very different from those of the case above.

1		V	ΔV	Avg
19/08/2006 - 10:06:29 PM				
Transducer: 2.2MHz Mode: AV				
2	Vpk (m/s)	0.72	0.00	0.84
	HR (bpm)	86	0.00	83
	MD (m/min)	10	0.00	12
	SV (cm ³)	42	0.00	50
	SVI (ml/m ²)	20	0.00	24
	CO (l/min)	3.6	0.00	4.2
	CI (l/min/m ²)	1.7	0.00	2
	SVR (ds cm-5)	2193	0.00	1960
	SVV (%)	49	0.00	38

Perhaps the most obvious finding is that the cardiac index is 1.7 and the cardiac output just 3.6. The Vpk is only 0.72 which suggests a low

myocardial contractility, whilst the MD of only 10 indicates a hypodynamic circulation. The stroke volume at 42 is clearly low, and when we look at the SVR it is quite apparent that the patient is markedly vasoconstricted. This is very typical of low cardiac output states.

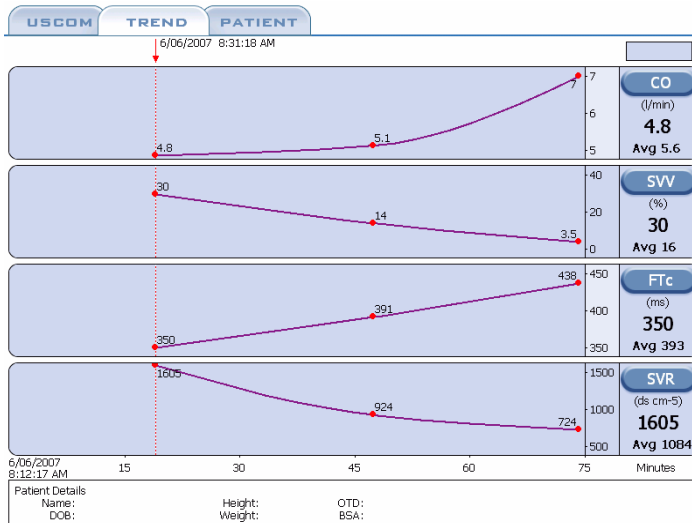
But what about the stroke volume variation? What does an SVV of 49% mean in this case? Surely it cannot indicate that the patient is severely volume depleted? His FTc was raised at 526 (normal at this age is about 450) indicating a higher than normal preloading. In fact the patient had pulsus alternans, a classical finding in cardiac failure. Again, this is a case of taking the whole clinical picture into account rather than laying too much emphasis on just one single parameter.

USCOM on the surgical ward.

But can the USCOM help in surgical patients too? You bet it can!

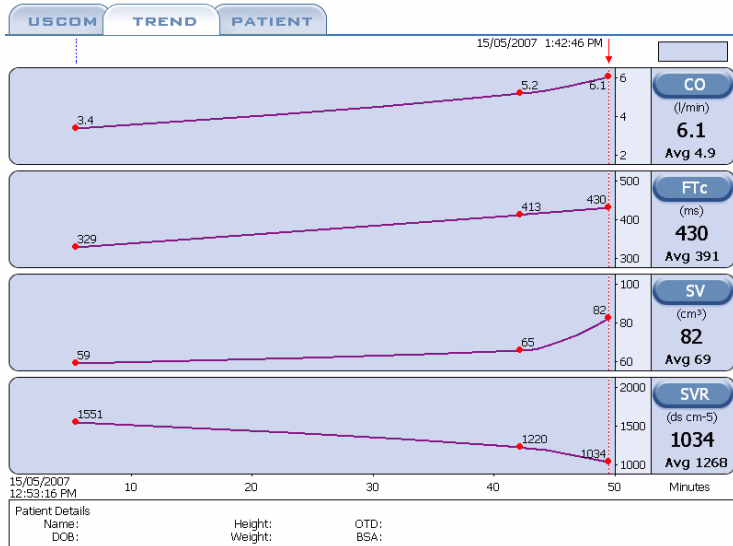
Perhaps one of the commonest problems that we see is the hypotensive postoperative patient. Here the differential diagnosis lies between bleeding and other causes of hypovolaemia, and a coronary event or even pulmonary embolus, that has led to the low blood pressure. USCOM will very quickly tell us which of these it is. Hypovolaemia from whatever cause will show a low CI, a low MD, a high SVR indicating vasoconstriction, and a low FTc with raised SVV. A septic patient will show a raised CI, reduced SVR and evidence of a hyperdynamic circulation with a raised MD. We have already seen in this booklet how easily a USCOM can spot myocardial failure. A pulmonary embolus of any significance produces very similar results. Again, what we are looking for is a typical *pattern* of haemodynamics rather than just individual values.

Perhaps the USCOM is of even greater value however in the patient who is simply becoming dehydrated. The BP may be normal although the pulse tends to be increased. We will see a raised SVR and a low FTc with an elevated SVV. BP and cardiac output may be maintained simply by the degree of vasoconstriction present as shown in the trend display here.

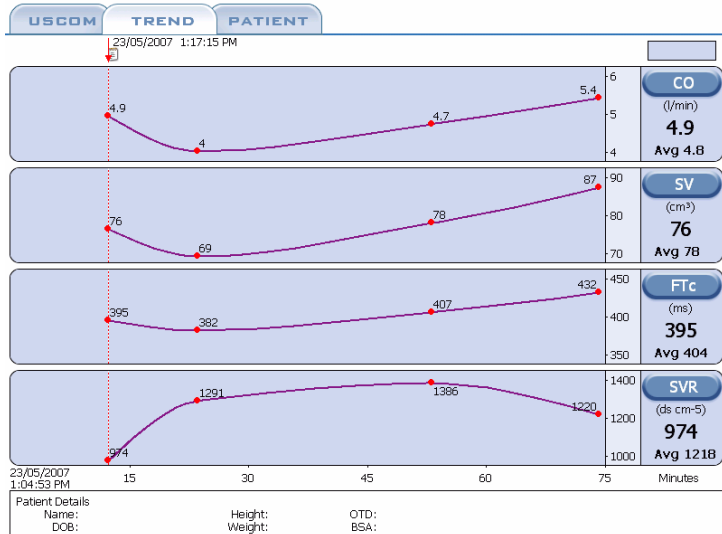


This shows the effect of giving 1L of normal saline over about 45 minutes. The CO increases from 4.8 to 7 litres, the SVV falls from 30% to just 3.5%, the FTc increases steadily from the moderately low value of 350ms to an entirely normal 438ms. But what happened to the peripheral circulation? Well the saline must have done the trick as the SVR fell by more than 50%! We now have a nicely vasodilated patient with a higher CO and better peripheral perfusion, exactly what we wanted, and achieved in under an hour with no guesswork.

So if we think the patient is dehydrated then why not give the patient a fluid challenge to see if the situation improves? Even if we were seriously concerned that the patient might already be overloaded then there is still no problem - we can just elevate the patient's legs and see if the stroke volume increases with the increased venous return. If so, then this clearly indicates the need to increase the preload, i.e. they need fluid. Should the stroke volume go down, then they are already overloaded. All we have to do is put the legs back down and no harm has been done, and now we know for certain which way to go to solve the problem. Primum non nocere – first do no harm!

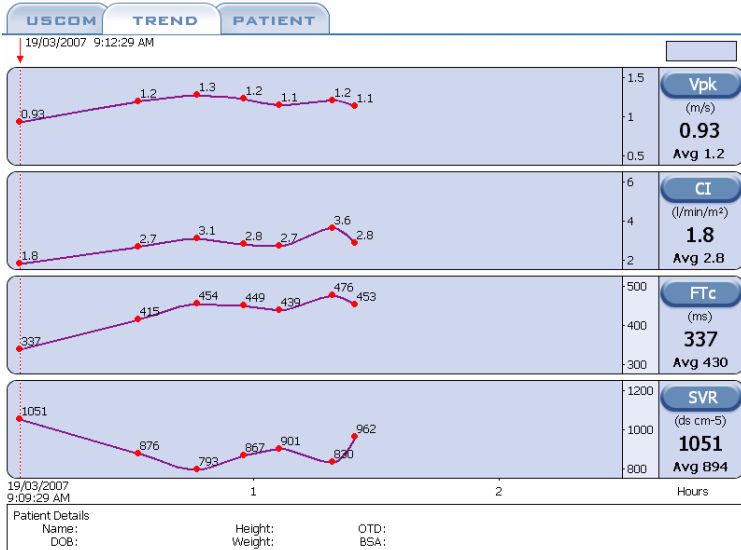


In this trend display we see a patient who was moderately hypotensive following abdominal surgery. The cause is easy to spot. We know from “The USCOM and Haemodynamics” that $BP = CO \times SVR$. In this case the BP is low because the CO is low. The SVR is close to the upper end of normal. In this trend we see the response of the patient to simple fluid replacement. We see the CO and SV rising in line with the increasing FTc, whilst the SVR progressively falls. The sharp increase in SV in relation to FTc at the right hand end of the trace is quite typical as we approach optimum fluid loading, typically when the FTc is around 425ms.



In this trend screen we see a postoperative hip-replacement patient who has been bleeding steadily in the hour following surgery and has gradually dropped her blood pressure to around 90/45. The SVR is increasing whilst the cardiac output and stroke volume are falling. At around the 20 minute mark, transfusion of whole blood commences and as the FTc rises from 382 to 432, we see the stroke volume increasing from 69 to 87ml whilst the cardiac output rises from 4 to 5.4 litres per minute. Her blood pressure gradually increased throughout this time reaching 125/65. From the trend we can see that the ideal FTc for this patient lies in the region of 425 to 450, again a fairly typical FTc in normovolaemia.

Have you noticed in all these trend displays the clearly interactive behaviour between FTc, SVV, CO, CI, SV and SVR? This is how we obtain an overall view of the circulation, look at how it behaves as a whole. Everything interacts with everything else. The trick is to “get the feel” of what the heart and circulation are saying to you. Now that you’re getting the hang of this, let’s try a couple of interesting pictures!



How did we optimise the cardiac output in this case? Can you see the Frank-Starling Laws in action here? What were the haemodynamic values at optimum loading?

USCOM in the Out Patient Clinic.

No that's not a misprint, the USCOM really does have major applications in the Out Patient Department and in the doctor's office. Try this example.

	V	ΔV	Avg
Vpk (m/s)	0.94	0.00	0.94
SV (cm ³)	102	0.00	102
FTc (ms)	412	0.00	412
MD (m/min)	14	0.00	14
CO (l/min)	5.7	0.00	5.7
CI (l/min/m ²)	2.4	0.00	2.4
SVR (ds cm ⁻⁵)	1653	0.00	1653
SpO2 (%)	99	0.00	94
DO2 (ml/min)	1144	0.00	1144

These are the haemodynamics of a 58 year old man with a history of hypertension. He is currently on atenolol 50mg daily, and has a BP of 155/95 and pulse of 56/minute. Would you increase his atenolol? Whilst you're thinking about that, what if I told you that he is complaining bitterly that "I just don't have any energy to do anything. I'm falling asleep all the time. I never used to have that problem"? The USCOM tells us exactly why this man is not happy. His cardiac output is depressed to the point that he would almost qualify as chronic heart failure, the lower limit of CI being 2.4 l/min/m². His SVR at over 1600 tells us where the problem lies. If we start from BP = CO x SVR then there can only be two causes of hypertension, either the CO is too high or the SVR is too high. (Very rarely, both are too high.)

In this case the way to treat the raised BP is surely with an agent that reduces the SVR. This could be an ACE inhibitor, ARB, calcium antagonist or whatever. By lowering the SVR the BP will fall, even though there will also be an increase in cardiac output once the afterload has been reduced. The increased CO will help his symptoms of lack of energy. Ultimately, he might require a combination of agents. Time and the USCOM will tell, but there is no need for guesswork anymore.

So now you might be thinking that vasodilators such as ACE inhibitors and angiotensin receptor blockers are the way to go in hypertension. Well try this patient.

7/05/2007 - 4:49:58 PM				
Transducer: 2.2MHz Mode: AV				
		V	ΔV	Avg
1	HR (bpm)	82	0.00	84
2	Vpk (m/s)	1.4	0.00	1.3
3	SV (cm ³)	105	0.00	105
	SVI (ml/m ²)	56	0.00	56
	MD (m/min)	29	0.00	30
	FTc (ms)	457	0.00	487
	CO (l/min)	8.7	0.00	8.9
	CI (l/min/m ²)	4.6	0.00	4.7
	SVR (ds cm-5)	712	0.00	762

This 88 year old lady is normally very fit and well, but has been to see her doctor several times in the last two months complaining of tight chest pains, breathlessness on exertion and dizziness on standing or coughing.

She claimed that she had been “just fine until they changed my blood pressure tablets”. What does the USCOM show? Can you guess what type of medication she is on for her hypertension? What does the USCOM data suggest that we should do to treat this patient?

O.K. so ACE inhibitors and ARB’s aren’t the be-all and end-all of hypertension. There are horses for courses. β -blockers have their place, as in this case. Some patients are better on a thiazide, some on β -blockers, some on ACE inhibitors, some on calcium antagonists or whatever. Some do better with combinations of agents depending on their haemodynamics. What is abundantly clear is that claims by pharmaceutical companies that their drug is *the* answer to hypertension are patent nonsense, if you pardon the pun. With the USCOM, we can not only see which way to go for the best, but we can easily spot adverse effects of our treatment. Perhaps it’s not too strong to say that the days when we treated hypertension by just measuring the blood pressure are gone. As for prescribing on the basis of the drug written on the side of the “free” ball-point pen or other advertising gimmicks, don’t you think we owe our patients rather more?

Chronic Heart Failure.

This is one of the commonest conditions seen in Medical Out-Patients or in doctor’s offices. There are literally millions of patients treated every day with combinations of drugs for chronic heart failure. Digoxin, diuretics, ACE inhibitors, nitrates and β -blockers are all prescribed to patients daily, yet how many patients have their cardiac output measured or even reliably estimated? How do we gauge the effectiveness of our therapy? In the main, this consists of asking the patient if they feel better or can do more. It is at best highly subjective. It would be inconceivable to treat hypertension without measuring the blood pressure, so why do we treat heart failure without measuring the cardiac output? Until recently the answer would be “because we *can’t* measure cardiac output”. Well not any more. We now have a simple and accurate tool for doing what we have all wanted to do for years, so let’s get to it!

Exam Time: 2/11/2004 - 5:53:07 PM				
1		Transducer: 2.2MHz	Mode: AV	
		V	ΔV	Avg
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

This 63 year old woman has a history of breathlessness, orthopnoea, palpitations on exertion and tiredness. She has pitting oedema of the feet, ankles and lower calves. What do you see in her haemodynamics? Her medication at present consists of digoxin 0.125mg daily and frusemide 40mg daily. What medication changes would you consider here? What would you like her readings to be after you have modified her therapy?

The USCOM not only makes the diagnosis easy (not that this one is difficult!) but *quantifies* the problem. It shows us that the afterload is the main issue that we need to address, it almost tells us which drugs to use, and then gives us our therapeutic targets. Goal directed therapy as it should be. But best of all, after the change of therapy, we can re-measure the haemodynamics and make sure that we are helping the problem primarily, but then we can go on to optimise therapy. Why settle for “better” when you can have “best”?

The USCOM in Intensive and Coronary Care.

Just imagine that you had been admitted to Coronary Care having just had a heart attack. Is there any one of you who *wouldn't* want to know your haemodynamics? You might think that would be enough of an answer, but just to labour the point, try this case.

A 47 year old male office manager is admitted to CCU from the ED with a confirmed ST-elevation myocardial infarction. He was hypotensive on admission to CCU with a BP of 96/52 and was commenced on noradrenaline at 100ng/kg/min. Here are his haemodynamics 20 minutes later. His BP had risen to 108/68.

12/03/2007 - 10:06:44 AM				
1		Transducer: 2.2MHz	Mode: PV	
		V	ΔV	Avg
2	Vpk (m/s)	0.45	0.00	0.45
	HR (bpm)	96	0.00	96
	MD (m/min)	7.3	0.00	7.3
	ET% (%)	33	0.00	33
	SV (cm ³)	34	0.00	34
	CO (l/min)	3	0.00	3
	CI (l/min/m ²)	1.8	0.00	1.8
	SVR (ds cm-5)	3937	0.00	3937
	SVRI (ds cm-5m ²)	7237	0.00	7237

What do you see? Are you happy with his haemodynamics? How would you alter this patient's management, and what drugs would you use to achieve improved haemodynamics? What would you like his parameters to be?

The Intensive Care Unit.

From all that has been said in this booklet and in the first booklet "The USCOM and Haemodynamics" you probably think that just about all the features of the USCOM have been described already. Well yes and no!

I have repeatedly stressed the importance of looking at the circulatory system as a whole rather than individual parameters. In intensive care, we have to look at the patient as a whole and interpret the USCOM readings accordingly. Let's pick a typical example of the interaction between ventilation and circulation.

The patient is a 67 year old female with pneumococcal pneumonia leading to bibasal consolidation.

1		Transducer: 2.2MHz Mode: PV		
		V	ΔV	Avg
2	Vpk (m/s)	1.9	0.00	1.5
	SV (cm ³)	82	0.00	65
3	FTc (ms)	388	0.00	389
	MD (m/min)	34	0.00	26
	CO (l/min)	8.8	0.00	7.1
	CI (l/min/m ²)	6.1	0.00	4.9
	SVR (ds cm-5)	623	0.00	791
	SpO2 (%)	85	0.00	85
	DO2 (ml/min)	839	0.00	826

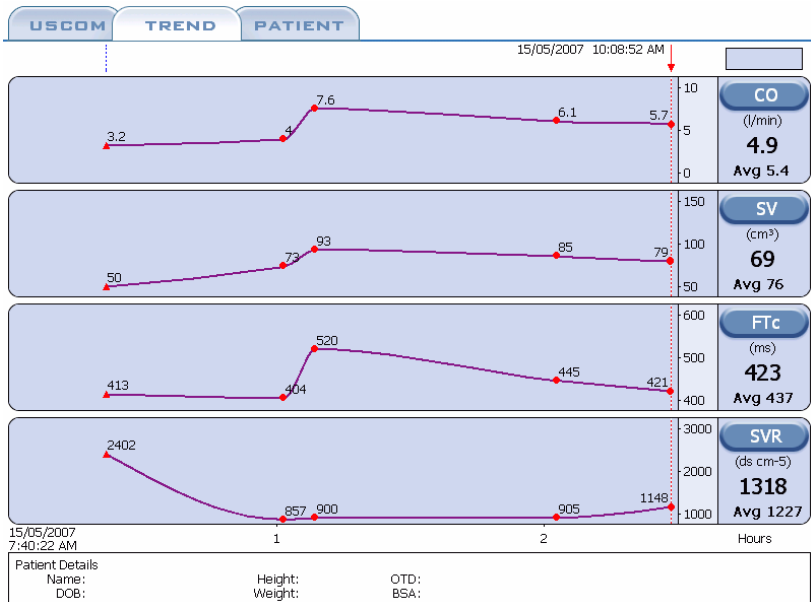
1		Transducer: 2.2MHz Mode: PV		
		V	ΔV	Avg
2	Vpk (m/s)	1	-0.08	1.1
	SV (cm ³)	55	18	56
	FTc (ms)	351	5	349
	MD (m/min)	18	-2.7	19
	CO (l/min)	5.6	-0.84	6
	CI (l/min/m ²)	3.9	-0.59	4.2
	SVR (ds cm-5)	1198	117	1139
	SpO2 (%)	94	3.3	95
	DO2 (ml/min)	668	0.00	668

On the left we see her haemodynamics when she was on SIMV with an inspired oxygen concentration of 70%. She was on PEEP at 5cm H₂O. Her SpO₂ was just 85% with a PaO₂ of 58mmHg. Her PaCO₂ was normal.

Would you increase her FiO₂, increase her PEEP, or leave well enough alone? I think that just about everybody would opt for increasing PEEP from 5 to 10 or even 15cm H₂O. The clever doctors will tell you that you titrate PEEP to achieve the optimum result. Unfortunately, they seldom spell out what exactly they mean by “optimum” or so-called “best PEEP”. Do you take an increased SpO₂ as a good result? Do you measure the arterial gases and assume an increased PaO₂ is a good outcome? Many intensivists do just that.

The right hand panel shows what happened when her PEEP was increased to 12cm H₂O. Her saturation certainly improved. Unfortunately, this is not the only thing that matters. Whilst the SpO₂ has risen by a little over 10%, (with a commensurate rise in her PaO₂), the cardiac output has gone down by over 35%. The net result of this is that her oxygen delivery, DO₂, has fallen by 20%. The pulse oximeter may say she’s better on the higher PEEP, but the USCOM tells the real story. The search for “best PEEP” is a great deal easier when you have appropriate tools to measure the response to your manipulations, especially now that the USCOM can give you DpO₂ directly.

The next case is a 62 year old man admitted to ICU following an emergency laparotomy for a strangulated inguinal hernia. At surgery approximately 1.5 metres of small bowel was removed as being of doubtful viability. His BP during the surgery had been consistently low averaging 85/50. Towards the end of surgery he was commenced on a noradrenaline infusion which increased his BP to 105/60. The presumptive diagnosis was that he had developed septicemia. Here is his trend display.



Looking at his admission figures (first reading), do you agree with the diagnosis? What would you do instead?

In fact, the haemodynamics suggest that he is volume depleted, with a low SV and CO and a high SVR indicating excessive vasoconstriction. He has a moderately low FTc in spite of the vasoconstrictive effects of the noradrenaline. He was treated by gradual withdrawal of the noradrenaline with simultaneous volume expansion. At the 1 hour marker his noradrenaline was discontinued and 500mls of Hartmann's solution infused rapidly. Although the USCOM showed that this bolus of fluid was largely equilibrated after one hour, his haemodynamics remained adequate and maintenance fluid only was sufficient to keep his cardiovascular parameters in the normal range.

In this final case, a 46 years old male, 118kg, type 1 diabetic was admitted to ICU with hypotension (85/40) following incision and drainage of a large axillary abscess under general anaesthesia. His ECG showed ST depression in the anterior and lateral chest leads, with a normal heart rate and normal conduction. Here are his admission haemodynamics. What is your diagnosis? How would you treat him?

28/03/2007 - 11:16:37 AM		Mode: AV	
1	Transducer: 2.2MHz		
		V	ΔV
2	HR (bpm)	67	0.00
	Vpk (m/s)	2.8	0.00
	SV (cm ³)	260	0.00
	SVI (ml/m ²)	136	0.00
	FTc (ms)	559	0.00
	CO (l/min)	17	0.00
	CI (l/min/m ²)	9.1	0.00
	MD (m/min)	49	0.00
	SVR (ds cm ⁻⁵)	181	0.00

With a CO of 18 l/min it is not surprising that he has evidence of myocardial ischaemia, especially with a diastolic BP of only 40. This is the pressure that perfuses his coronary arteries. His CI is more than 3 times normal yet he is still hypotensive. The FTc of 559 shows that he is well pre-loaded, indeed it suggests overloaded, so what is the problem? The MD shows that this is a very hyperdynamic circulation. The SVR makes this a no brainer! He is septic with peripheral vascular collapse. Although his heart is ejecting more than 3 times the normal CO, his SVR is only one-sixth of normal. From $BP = CO \times SVR$, even this high CO cannot maintain the BP in the face of such vasodilation.

As regards treatment, whilst your first thought might be to just use a vasoconstrictor (pressor) agent, you must always beware of underlying myocardial depression. Whilst the patient is this vasodilated, there may not seem to be any evidence of this, but as the SVR rises the true state of myocardial function may become apparent. Consider dopamine or noradrenaline as the situation unfolds, and repeat the USCOM regularly!

As a final thought, why do you think his heart rate is only 67? He is a type 1 diabetic so it is unlikely to be due to a β -blocker. What else could be going on?

USCOM in Paediatrics.

Can the USCOM be used in children? The answer is an emphatic “yes!” and very much more easily than in adults in the main. Kids are very amenable to “would you like to listen to your heart with my special

computer”. Provided they’re not too ill, they will usually help you press the buttons, and they love hearing the whoosh-whoosh of their cardiac output. Because of their smaller size, the aortic and pulmonary valves are much nearer to the insonation site and are therefore much easier to pick up. The shorter distance also results in less signal attenuation and better quality traces in less time than in adults.

A word of warning however; children are not small adults. At times I think they are actually a different species! Whilst the basic rules of haemodynamics apply in paediatrics, some of the haemodynamic values might surprise you. Take a look at these data screens. The screen on the left is from a child of just over 6 years who weighed 20kg. The screen on the right is from a 5 year old who weighed 17.5kg. What do you see?

		6/03/1999		
		Exam Time: 25/03/2005 - 3:58:49 PM		
		Transducer: 2.2MHz Mode: AV		
		V	ΔV	Avg
2	Vpk (m/s)	1.4	0.03	1.3
	CO (l/min)	4.5	0.28	3.8
3	CI (l/min/m ²)	4.2	0.22	3.5
	HR (bpm)	79	2	80
4	MD (m/min)	22	0.01	20
	ET% (%)	45	-1.4	42
	SV (cm ³)	57	2.1	47
	Pmn (mmHg)	3.2	1.2	3
	SVR (d s cm ⁻⁵)	1214	6.3	1221

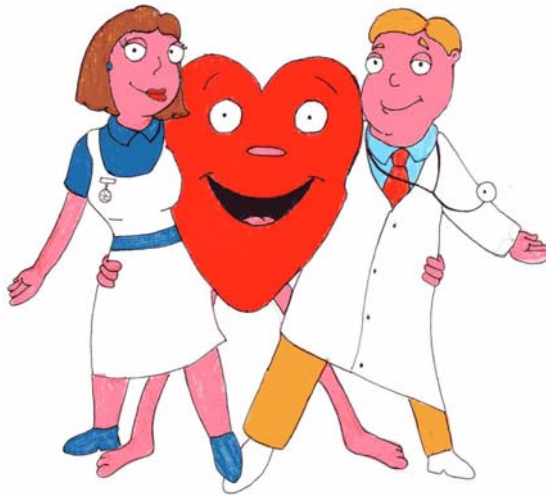
		19/12/1999		
		Exam Time: 23/02/2005 - 9:47:54 AM		
		Transducer: 2.2MHz Mode: AV		
		V	ΔV	Avg
2	Vpk (m/s)	1.5	0.00	1.6
	vti (cm)	27	0.00	27
3	HR (bpm)	122	0.00	117
	MD (m/min)	33	0.00	32
4	ET% (%)	67	0.00	68
	SV (cm ³)	42	0.00	42
5	CO (l/min)	5.1	0.00	5
	CI (l/min/m ²)	6.5	0.00	6.3
▼	SVR (d s cm ⁻⁵)	636	0.00	656

The numbers are certainly interesting, but I expect that there a few things you need to know before you can draw any definite conclusions. The clinical history is obviously important, as are other observations like BP, temperature, skin colour and so on. But more than all this, I bet you said to yourself “what’s the normal value for that in a child of 5/6?”. Now there’s a whole new ball-game to explore! Haemodynamics is just as applicable in children, but because children are so different in so many crucial ways, they will get a booklet all to themselves. Watch out for part three of the USCOM trilogy “USCOM – Haemodynamics in Children”.

USCOM in the Future.

In this brief guide I have tried to give you some insights in to the incredibly powerful tool that is the USCOM. This guide is not exhaustive, but I’m sure that you can already think of many uses for the USCOM in your own practice and of many past patients in whom you wished you had the

USCOM available. As a little final food for thought, what about the USCOM in pre-hospital care at the scene of accidents, in patient transport and retrieval, in sports medicine, in pacemaker implantation, in verifying other monitoring systems, in the early diagnosis of sepsis and even in public health projects. As a tool to teach physiology and therapeutics it is unrivalled. How about clinical trials of new medications or treatment protocols? Even if the trial is not primarily aimed at the cardiovascular system, wouldn't you like to know that it has no harmful effects on haemodynamics? I would if I were the patient! USCOM is probably limited only by your imagination....



Acknowledgements.

My thanks are due to my friends and colleagues Dr Antony Parakkal MD, Staff Specialist in Anaesthesia, Ms. Veronica Madigan, Senior Lecturer, School of Public Health, Charles Sturt University and Dr. Julia De Boos. I must also thank the Nursing staff of Broken Hill Base Hospital and Bathurst Base Hospital, N.S.W., Australia and Mildura Base Hospital, Victoria, Australia, for their input and feedback, and especially the staff of Uscom Ltd, Sydney, Australia, for their advice and criticism.

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	

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
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	

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