**Quality Indicators in Perfusion - Part 2**

**Ed Overdevest** [00:00:12] Do you think that go direct to perfusion therapy can affect also other CPB mortalities such as on cerebral perfusion and its neurological aspects?

**Prof. Marco Ranucci** [00:00:25] Well, that's a very important and difficult question. The reason why we have been always concentrating on kidney is that for its peculiar physiology, kidney is particularly prone to the insult of a low oxygen delivery. It is not the most sensitive organ that is gut mucosa, but the second organ suffering from a dysoxia is kidney. So it's a sort of marker of the quality of the perfusion, but even of the natural cardiac output before and after CPV. Then the second reason is that kidney is very, very active in providing us with signals, apart from the urine output. But the most important signal is changing creatinine. This is something that The gold standard is very well validated. We know we have this classification, acute kidney injury, stage one, if you have 50% increased creatinine, stage two, if you double the baseline creatinines and so on. So this is very defined. And we have these numbers. I'm quite sure that as the kidney is suffering, even other organs probably are. The difficult thing is that it's difficult to measure. Again, in this case, on the y-axis, if you put mortality, I'm quite convinced that GDP could change, but you need, to demonstrate this, you need thousands and thousands of patients because, luckily, mortality occur at a very low rate. Acute kidney injury is about 15 to 30%. Mortality is about three to five percent. So you will never demonstrate differences in mortality. There are other outcomes that probably are of interest. But again, sure, brain is an interesting organ from the point of view of the oxygen delivery. But with our clinical setting, usually we define a brain damage based on microscopic insult like a stroke. To see more subtools. Complication, then we should either do a magnetic resonance to everyone that is unfeasible, but we would see something. Or to look at cognitive function, so there are these tests of memory, attention, and we probably could see something. But again, this on a large scale is not that easy because you You need a dedicated psychologist or you need a magnetic resonance. At the end of the day, I think that there's no reason why only kidney would be affected by low oxygen delivery or other factors. But that is very difficult to demonstrate that this applies to other organs.

**Ed Overdevest** [00:03:41] You mentioned cognitive function as one of the options to measure post-operative cerebral dysfunction. What is your opinion about post-op delirium or maybe biological markers that may indicate some cerebral dysfunction?

**Prof. Marco Ranucci** [00:04:01] That's a possibility. Well, delirium, there's a great bias that is the anaesthetic management. Because the more we are investigating the reason for deliriums, the more it appears that one major determinant is a too deep level of anaesthesia. We have been, for decades, scared by awareness. But it seems that now the problem is absolutely the opposite. Then, of course, there are markers, but I've been never thinking that markers are outcomes. You can have an increase in tropoline, but you don't have myocardial infarction. You can even increase in the S100 protein, but this does not necessarily mean that you have a clinically relevant damage. So outcome is something different from markers.

**Ed Overdevest** [00:04:58] I'd like to come back to the guidelines, and I would like to ask you why is it that respiratory co-chand is not often referred to as one of the goal-directed perfusion parameters. It's like a biological indication of the metabolic status. It's less influenced, such as VCO2 and DO2, VCO 2 by patient size and shape and gender. What is it? In your opinion.

**Prof. Marco Ranucci** [00:05:30] Well, that's another very important question. Let me go a step back. There are some of the indicators that are related to what we call goal-directed perfusion, but basically to the metabolic state of the patient. Some of them are what we give to the patient, oxygen delivery is something that we can give to the patients according to the hematocrit or to the pump flow. And then there is something that the patient is providing back to us as a signal of the adequacy of our perfusion. And VCO2 and lactase are typical examples of what the patient is providing us as an information. And the respiratory quotient, of course, is very much important. We know that whenever the respiratory quotient overcomes a level of about 1.2, then this means that there is an anaerobic CO2 production. And there is a nice relationship between lactate formation and VCO2. This is absolutely true. And the big advantage of VCO2 and respiratory quotient that is calculated based on VCO two and VO two, the big advantages that you can have this continuously online where is lactate formation, it is a serial measurement. Having said this, there are basically two points. One is that it is not that easy to standardise the The data collection, actually, the CO2 at the exhaled port of the oxygenator, it depends on which kind of capnograph you are using. It could be side screen, could be mainstream. So there are differences. The second thing is that, as you know better than me, whenever there is a CO2 floating in the surgical field, then this will actually inevitably... Introduce a big bias because this CO2 will be suctioned by the aspirators in the surgical field and will fill the venous reservoir. So at least during the CO2 floating this number is not is not actually available but from a theoretical and even practical point of view whenever all the setting is correctly on site. I think that. Seeing that it's coming from the patient as a CO2 production is the best marker of perfusion. Because when we say oxygen delivery should stay at around 280, this is the mean value from the mean population. Then I can guarantee you that there are people who can safely stay at 240 and others that maybe are in need for 320. But we must apply the mean level. But when it comes to CO2, that is the CO2 of all that specific patient. So this is the concept of a Taylor perfusion. We give the mean level and then we see what the patient is giving back to us as a marker of the adequacy of perfusion

**Ed Overdevest** [00:09:00] Thank you very much for all your input and is there anything that you would like to address again or comment on?

**Prof. Marco Ranucci** [00:09:09] No, I think we have touched many, many interesting points, and I thank you again for the opportunity of discussing them with you.