Cardiac surgery utilising cardiopulmonary bypass has long been associated with alterations in vascular tone and function. These in turn may have important effects on vital organ blood flow, and in due course affect patient outcomes. This lecture examines some of the factors that affect vascular biology in cardiac surgery patients. The subject will primarily assess the following:

- Physiologic effects of non-pulsatile hypotension
- Effects of relative hypothermia
- Effects of haemodilution
- Inflammatory mechanisms and pro-inflammatory mediators
- Pharmacologic effects of preoperative medications and anaesthetics

**Physiologic effects of non-pulsatile hypotension**

Blood pressure control is managed acutely by the baroreceptor system in the carotid sinus and aortic arch. These receptors are simple stretch receptors, and high pulsatile pressure will activate them and send signals along the efferent nerves to the vasomotor centre in the medulla. Increased signalling, associated with hypertension, has the effect of causing an inhibition of medullary discharge, leading to a reduction in VMC activity, and reduced sympathetic tone. In contrast, reduced baroreceptor activity (hypotension) will fail to inhibit medullary discharge, leading to an increase in VMC output and sympathetic nervous system activity. This has a primary vasoconstrictive effect, and a secondary effect on rennin and angiotensin release causing further vasoconstriction. Thus non-pulsatile hypotension causes a marked increase in reflex vasomotor tone and increased serum vasopressor amines. In the post-operative period, this frequently manifests itself as hypertension.

**Effects of relative hypothermia**
If the skin temperature drops below 37°C a variety of responses are initiated to conserve the heat in the body and to increase heat production. These include vasoconstriction to decrease the flow of heat to the skin, cessation of sweating, shivering to increase heat production in the muscles, and secretion of norepinephrine, epinephrine, and thyroxine to increase heat production.

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Importantly, these effects may be further modified by cardiopulmonary bypass, which may lead to significant rises in plasma adrenaline and noradrenaline levels. [1-3] These effects may be modified by modified by pulsatile flow. [4-9]. Thus the effects of the combination of non-pulsatile hypotension and hypothermia are additive, and only partially offset by modification of bypass flow and normal temperature.

**Effects of haemodilution**

The normal management of CPB involves a reduction of haematocrit from approximately 40% to 20%. This can be achieved by using a priming volume of 1000-1500mls crystalloid/colloid. Although of questionable benefit in modern practice, this may be aided by normovolaemic haemodilution pre-bypass. The effect of haemodilution is to reduce blood viscosity which will, in turn, improve microvascular flow and reduce pressure. Both these processes may be aided by a dilution of endogenous pressor amines as a result of the pump prime.

**Inflammatory mechanisms and pro-inflammatory mediators**

In recent years an enormous amount has been written about the effect of inflammatory mediators in the surgical patient. The use of CPB focused interest on cardiac surgery patients, and the potential for blood activation in this setting is clear. Factors that are relevant inflammatory mediators in the cardiac surgery patient include cold [9-11], contact activation [12-16], the issue of surgery on or off pump [17-22], the presence of ischaemia, hypoperfusion, and infarction, and the duration of surgery and nature and severity of surgical trauma.[23-27]
However, a number of studies have suggested that mechanisms of inflammation in cardiac surgery patients are not restricted to CPB-related effects, and that blood contact with wound surfaces is a potent pro-inflammatory stimulus.[28-30] Many of the inflammatory mediators have a common final pathway involving a receptor-mediated increase in the activity of inducible NO-synthetase, leading to enhance NO levels. This in turn causes pathological vasodilatation and myocardial depression by a number of mechanisms.

**Pharmacologic effects of preoperative medications and anaesthetics**

These are almost always vasodilator in their effects, and may produce intraoperative hypotension. Although this is usually simply manageable, there has been controversy about continuing or discontinuing some medications, especially ACE inhibitors and K+ channel openers, before surgery.

**Conclusion:**

There is no doubt that hypothermia and non-pulsatile perfusion have been the subject of much research. However, the simple vascular effects that these entities provoke are usually simple to control. Of more difficulty, is the vascular response to cytokine release that appears to be related not just to bypass, but also to the extent of surgery, and is much less predictable both in its nature and severity. The drive towards avoiding CPB has not eradicted this problem. It will be interesting to see whether minimal access surgery is able to have a significant impact in the future.

**References**

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