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LETTER TO THE EDITOR

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# Bradycardia in children less than two years of age during liver transplantation

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Sir: High caval cross-clamping in an adult, with the associated reduction in venous return, causes a tachycardia via the baroreceptor reflex. This is a well-recognised physiological response and can be demonstrated clinically by the Valsalva manoeuvre. However, we have noticed in children less than 2 years of age that there is a progressive fall in heart rate following caval clamping.

In a retrospective search of the Liver Transplant Anaesthetic Database there were, in the last 7 years, 90 cases involving children aged less than 24 months. Of these, the total number for which complete data were available was 46. The computerised record collection documents vital signs and other parameters every 2 min and was able to demonstrate a fall in heart rate in the majority of cases. None of the records showed a rise, as would have been expected in adults, though there were nine cases where there was no change.

Below is a typical example: Knowledge of paediatric physiology in this age group is limited, and much of our practise and understanding is based on neonatal and adult data. We have found no satisfactory explanation for this trend and can only postulate the following.

Firstly, we appreciate that there are many volume shifts occurring at or around this time and that preload may vary greatly. From our records there is no definite trend in the documented central venous pressure, although we appreciate that in paediatric cases this is not necessarily a good indicator of preload. It is not our common practise to prophylactically increase preload at this time, but only to respond to arterial pressure changes. Similarly, cross-clamping of the aorta would artificially elevate systemic vascular resistance and, hence, lead to a bradycardia. However, this again is not a universal practise, and in our data analysis we found no obvious correlation between the observed bradycardia and aortic cross-clamping. Also, the fall in heart rate is gradual, which is not what would be anticipated with such a sudden rise in afterload.

Alternatively, changes in temperature and arterial $CO_2$ would offer a potential explanation, but these parameters remained unchanged throughout. We have no method of measuring cardiac output and systemic vascular resistance in small children, but the end-tidal $CO_2$ (a recognised reflection of cardiac output) remained unchanged during this period. If the cardiac output were to fall as a direct result of caval clamping, we might anticipate a bradycardia in patients with a fixed stroke volume. Infants have a rate-dependent cardiac output, and this has been demonstrated when patients are volume-loaded. However, if the converse were true, we would expect to observe a bradycardia in infants that are volume-depleted. This is not the case.

In addition, this time period corresponds to the anhepatic phase and, consequently, changes in drug metabolism may offer an explanation with the accumulation of the opioid used (fentanyl). Muret et al. [2] demonstrated that baroreflex

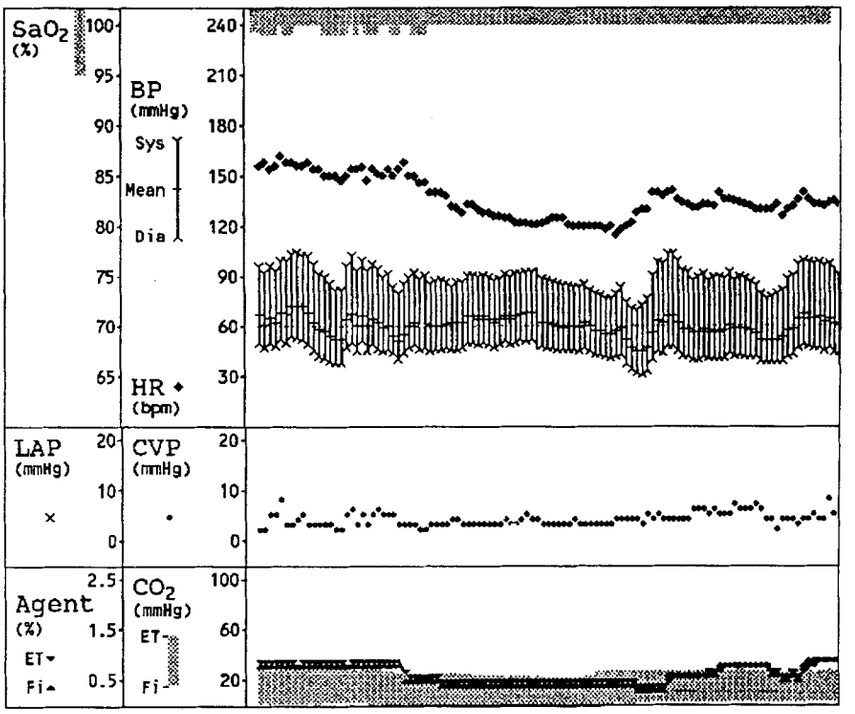


**Fig. 1** A computerized record showing a typical example of bradycardia following caval clamping in an infant undergoing liver transplantation. The fall in heart rate is gradual until hepatic reperfusion occurs

control in term neonates was markedly depressed during fentanyl anaesthesia. Whether or not fentanyl does significantly accumulate would require further investigation. The liver is also responsible for the breakdown of endogenous substances. The raised levels of noradrenaline measured by Estrin et al. in liver transplantation [1] may be responsible for a reflex bradycardia as a consequence of a raised systemic vascular resistance.

Speculatively, there may be a physiological reflex that might involve atrial, ventricular or pulmonary stretch receptors. The time period over which the heart rate falls, approximately 20–30 min, may be indicative of a reflex resulting in an elevated systemic vascular resistance. This, in turn, would result in the observed bradycardia. However, we can find neither a reference nor a description of such a reflex.

It is known that the parasympathic system in infants is more mature than the sympathetic system, but we find it difficult to explain why caval cross-clamping should be vagotonic.

Then again, there may be a simple answer to this trend, namely, that in the face of an unchanged anaesthetic there is less surgical stimulation during the anhepatic phase.

In conclusion, as paediatric physiology is an underdescribed subject, we have found little in the literature to guide us to the cause of bradycardia following caval cross-clamping in this age group. We appreciate that the theories we have outlined to explain the trend are not exhaustive, and we present this letter to invite further discussion.

## References

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