

Vasomotor Induced Blood Pressure Activity During Hypothermic Cardiopulmonary Bypass

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Abstract

The observation of an aspect of the blood pressure waveform during hypothermic cardiopulmonary bypass is reported. Appearance of this activity may be pertinent to the management of clinical perfusion.

Introduction

Adequate cerebral perfusion during cardiopulmonary bypass is fundamental to metabolic homeostasis throughout the course of bypass. This is true in regard to the achievement of basic tissue oxygenation, and also insofar as the state of cerebral perfusion affects the autonomic aspect of vasomotor center function.¹ In general, adequate cerebral perfusion is inferred from analyzed systemic blood gas status, but this assumed correlation may not in fact be reliably valid.^{2,3}

There may be evidence of this fact in an observed phenomenon which may, when recognized, prove of clinical value to the effective conduct of perfusion. A series of patients exhibiting this phenomenon is presented.

Description

During hypothermic, non-pulsatile perfusion, the blood pressure waveform display normally ap-

pears essentially flat. Upon closer inspection, this arterial waveform occasionally exhibits a very slow sinusoidal pattern which is typically quite regular in both frequency and amplitude. The frequency may be as long as forty seconds in duration, and requires a recording paper speed of one millimeter per second in order to be readily perceptible on record (Figure 1). The phenomenon does not begin until well into core hypothermia, and disappears with rewarming.

Blood pressure activity of this type in the clinical or experimental setting is known variously as Mayer waves, Traube-Hering waves, or simply, vasomotor waves.^{4,5,6} Excluding consideration of the regimen and depth of anesthesia, these pressure waves may represent vasomotion in response to baroreceptor reflexes, chemoreceptor reflexes, or the Central Nervous System ischemic response.⁶ With respect to cardiopulmonary bypass, it is this latter mechanism which is theorized to most likely be of significance.

The medulla of the brain is conveniently referred to as the vasomotor center.^{1,7} Afferent impulses reach the medulla from various points of the body, including the cardiac and respiratory centers of the medulla itself. Efferent impulses are initiated from the medulla to stimulate vasomotor nerve terminals. These nerves are considered to effect noradrenaline release, as a result of which smooth muscle in the blood vessel walls may contract to cause vasoconstriction. The resultant increase in blood pressure improves perfusion to the point where this nervous control is then inacti-

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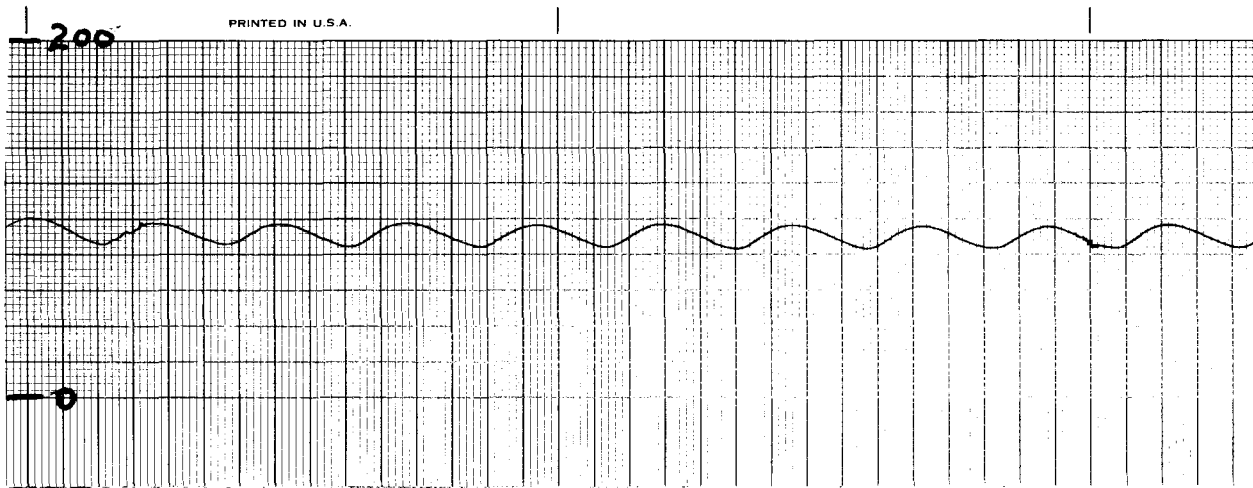


FIGURE 1. Recording paper speed 1 mm/sec. Full-scale deflection 0–200 mm/HG.

vated.⁶ This process defines a continual feedback mechanism which will when necessary produce effect upon the systemic blood pressure and its waveform display.

The medulla is subject to ischemia by mechanisms which can include those contributed by cardiopulmonary bypass. Such mechanisms might include blood flow rate, distribution, oxygen and carbon dioxide content, and cerebral edema.^{8,9} It is suggested here that cerebral hypoxia of varied possible causes may produce a cyclical vasomotor response during hypothermic bypass which leads to the observed rhythmical arterial pressure wave activity. States of hypothermic perfusion can be sufficiently dynamic so as to permit compromised circulation and oxygen availability to the brain. Blood flow, pressure, and dissolved gas contents may prove independent of each other in their delivered values during this period.^{2,3}

Method

Records of eleven adult cardiopulmonary bypass patients were selected over the period of a year as representative of this vasomotor wave phenomenon. The average bypass time was 110 minutes. The patients were cooled to 28 degrees Celsius core, at which temperature the average blood flow was 2.10 ml/minute/meter². The average mean arterial pressure was 81 mmHg. The averaged blood gas analysis values (temperature corrected) were $p_aH = 7.43$; $p_aO_2 = 176$ mmHg, $p_aCO_2 = 30$ mmHg, VO_2 saturation = 88.5%. Average CVP was 4.6 cmH₂O. Arterial blood flow

was predicated upon mixed venous oxygen saturation values, or upon arterial-venous oxygen transfer derivations. SciMed^a oxygenators were used in all instances, with an independently controlled mixture of 100% oxygen and room air administered.

The hypothermic arterial pressure strip chart record shown in Figure 1 is one typically representing this series.

Discussion

It is suggested that, in this series profile, perfusion of the medulla was either inadequate or interfered with, producing transient and cyclical CNS ischemic responses despite the appearance of reasonable systemic lab analysis parameters.^{2,3} Conjectural causes of the ischemic response which may be posited include:

Elevated intracranial pressure. This condition has been shown to exist during cardiopulmonary bypass, but obviously cannot be a routine measurement.¹⁰ Its cause has been attributed to excessively high blood flow, or to elevated systemic and central venous pressures, or both.¹⁰ In this condition the cerebral vasculature can be compressed, either directly, or indirectly through concomitant cephalic edema. The CNS ischemic response may be elicited as a result, causing vasoconstriction and an attendant rise in blood pressure until the condition is alleviated. Subsequently the cycle may repeat itself again in ongoing fashion.

^a SciMed Life Systems Inc., Minneapolis, Minnesota 55441.

Hypocarbica. Hyperventilation during hypothermia is easy to produce and can be difficult to control depending upon oxygenating device and ventilating gas flow/mixture regimen. Sufficiently high $p\text{CO}_2$'s are long known to be important during hypothermia in order to ensure adequate cerebral perfusion.^{2,11} Diminished cerebral perfusion secondary to insufficient carbon dioxide tension may have elicited the CNS ischemic response in this series of patients and provoked the vasomotor condition, however an average corrected $p_a\text{CO}_2$ of 30 mmHg is not particularly low out of range. Keeping the $p_a\text{CO}_2$ sufficiently low in the rewarming process is no longer a problem for the perfusionist due to the improved efficiency of blood oxygenators. This is especially true with the use of membrane oxygenators, which permit the independent management of total ventilating gas flow and its oxygen content; or which provide a measure of control over the blood film thickness.

"Non-adjusted" pH protocols. Recent research suggests that, during hypothermic bypass, blood pH should be adjusted following poikilotherms to a respiratory alkalotic value (irrespective of the hypocarbica) rather than maintaining the more common values near pH 7.40.¹² With this technique cerebral blood flow was increased significantly, systemic tissue oxygen consumption was enhanced and systemic lactic acid metabolism appeared more favorable. Limited clinical experience with a similar approach by one of the authors of this paper (JK) tends to corroborate the "adjusted pH" technique. Vasomotor wave activity has not been observed in patients managed in this manner during hypothermia.

Low cerebrospinal fluid oxygen tension has been shown to exist during cardiopulmonary bypass, and may also exist independently of clinically acceptable systemic oxygen tension and transfer values.² This would seem to also attest that standard perfusion protocols do not necessarily ensure sufficient cerebral perfusion. Low CSF $p\text{O}_2$'s seen during cardiopulmonary bypass are an effect of cerebral hypoxia secondary to the diminution of perfusion caused by reasons which likely include those posited above.² The presence of low CSF $p\text{O}_2$'s may contribute to the CNS ischemic response and the appearance of vasomotor waves. Unfortunately, CSF oxygen tension is not a readily available measurement.

Conclusion

The phenomenon that vasomotor waves exist during hypothermic cardiopulmonary bypass is evident. Their appearance may, when understood, be useful in the management of perfusion. Research has shown that during hypothermic cardiopulmonary bypass, cerebral perfusion may be diminished in spite of "normal" systemic values. The reasons for this may relate to bypass management. Blood flow, filling pressure(s), $p\text{CO}_2$, and pH values should be monitored and considered in this regard. Pulsatile perfusion may be indicated.¹³ The arterial pressure waveform display is an easy routine observation for the appearance of vasomotor waves.

Addendum

The mechanisms postulated here in explanation of this phenomenon during bypass are essentially theoretical and by no means exclusive. Other experience with, or determination on this topic is solicited in reply through the JECT editorial column.

References

1. Bell, G. H., Davidson, J. H., Emslie-Smith, D.: *Textbook of Physiology and Biochemistry*. Baltimore: Williams and Wilkins Co., 1972, pp. 559–560.
2. Hasbrouck, J. D., Rigor, B. M.: The oxygen tension of cerebrospinal fluid during cardiopulmonary bypass, *J. Thor. Card. Surg.* 58:754–757, 1969.
3. Stockard, J. J., Bickford, R. G., Schauble, J. F.: Pressure-dependent cerebral ischemia during cardiopulmonary bypass, *Neurology*. 23:521, 1973.
4. Best, C. H., Taylor, N. B.: *The Physiological Basis of Medical Practice*. Baltimore: Williams and Wilkins Co., 1950, p. 69.
5. Ganong, W. F.: *Review of Medical Physiology*. Los Altos: Lange Medical Publications, 1979; p. 461.
6. Guyton, A. C.: *Textbook of Medical Physiology*. Philadelphia: W. B. Saunders Co., 1976, pp. 274–275.
7. Wiedeman, M. P.: *Handbook of Physiology*. Section 2: Circulation, editors W. F. Hamilton and Philip Dow. Washington, D.C.: American Physiological Society, 1963, pp. 925–929.
8. Berger, E. C.: *The Physiology of Adequate Perfusion*. St. Louis: C. V. Mosby Co., 1979, p. 155.
9. Reed, C. C., Clark, D. K.: *Cardiopulmonary Perfusion*. Houston: Texas Medical Press Inc., 1975, p. 307.
10. Timmis, H. H., Hardy, J. D., Davis, D. J., Raju, S.: Intracranial pressure during C-P bypass, *Ann. Surg.* 174:887–891, 1971.
11. Reivich, M.: Arterial $p\text{CO}_2$ and cerebral hemodynamics, *Am. Jour. Physiol.* 206:25, 1964.
12. Becker, H., Vinten-Johansen, J., Buckberg, G. D., Robertson, J. M., Leaf, J. D., Lazar, H. L., Manganaro, A. J.: Myocardial damage caused by keeping pH 7.40 during systemic deep hypothermia, *J. Thor. Card. Surg.* 82:810–820, 1981.
13. Taylor, K. M.: "Why Pulsatile Flow during Cardiopulmonary Bypass?", in *Towards Safer Cardiac Surgery*, editor D. B. Longmore, Boston: G. K. Hall Medical Publishers, 1981, pp. 483–500.