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Editorial Views

Arterial Manometry under Pressure?

Each time a change can be observed in the height of a manometer applied to an artery, the experimenter must ask himself which of the two factors of the arterial pressure has varied, or whether the two factors, flow (power) and resistance have been modified at the same time. In the absence of a criterion which would allow one to decide with complete certainty this litigious question, often enough physiologists have chosen the hypothesis which best accorded with their own preconceived ideas. (Author's translation.) ETIENNE JULES MAREY, 1881[1]

TO THE CLINICAL ANESTHETIST, to whom the measurement of arterial pressure is commonplace, Marey's *caveat* is as pertinent today as it was to the physiologist of yesteryear. Whether the arterial pressure is measured directly or indirectly, two aspects of its measurement deserve consideration: first, those criteria which must be satisfied in order to measure the arterial pressure accurately, and second the interpretation of the measurements obtained.

It is often tacitly assumed that the combination of sphygmomanometry and auscultation of the Korotkoff sounds represents a standard and accurate measurement of systolic and diastolic arterial pressures in man. However, even if all the recommendations[2] concerning the width of the occluding cuff, its position, and the procedure for its inflation and deflation are followed, the absolute accuracy of both systolic and diastolic pressures is seriously in doubt. Geddes, in his exhaustive review of the literature up to 1968,[3] concluded that auscultatory indirect measurements consistently underestimated both systolic and diastolic values. In normal and mildly hypertensive patients, Van Bergen and colleagues[4] found that oscillometry was more accurate than either the auscultatory or palpatory methods, but that with all three methods there was a progressive tendency to underestimate the true arterial pressure, especially as the level of pressure rose. In our own studies, where indirect auscultatory measurements by intensive therapy nurses were compared with direct arterial pressures in patients with severe tetanus or polyneuritis, Kerr[5] found that there was reasonable agreement of systolic pressures in the range of 100 to 150 mm Hg, but that indirect pressures overestimated the true value at pressures between 80 and 100 mm Hg, and seriously underestimated the true value above 150 mm Hg. Nevertheless, the indirect methods can give a repeatable estimate of *changes* in arterial pressure even though absolute values cannot always be guaranteed.

The accuracy of directly measured intra-arterial pressure is also frequently taken for granted, and while the criteria for accurate reproduction of arterial and other intravascular pressure waveforms have been clearly stated,[6,7,8] too often these are either forgotten or ignored by those who use direct arterial manometry during clinical anesthesia. While most transducers which are commercially available have an acceptable amplitude/frequency response, the characteristics of the hydraulic transmission line connecting the patient's artery to the transducer may completely modify the performance of the whole

system. Too often one has seen transducers connected by one or two meters of relatively compliant tubing to the patient's arterial cannula. In this situation, it is only the fortuitous existence of an invisible or unnoticed air bubble in the system which causes sufficient damping to cancel or modify the excessive resonance associated with long transmission lines. Resonance in such a system can lead to serious overestimation of systolic arterial pressures and the recording of unduly low diastolic pressures. The appearance in an oscillographic trace of a signal which looks like an arterial pressure wave does not guarantee that it is a faithful reproduction of the true pressure signal! In practice, a stiff-walled manometer connecting tube of *not more than* 60 cm should connect a pressure transducer to the patient's artery,[9] and bubbles should be rigorously excluded.

Given that the measurement of arterial pressure is accurate, the interpretation of that measurement is still one of the main controversies in clinical monitoring. In this issue, Dr. Cullen has gathered data from a number of studies in man and animals, and his findings fail to support a number of cherished notions. In particular, he draws attention to the potential hazards of inferring about the state of the circulation on the basis of measurements of diastolic arterial pressure and the pulse pressure.

Pulsatile pressure changes reflect those in the energy content of the flowing blood, this energy being derived from the mechanical process of left ventricular contraction, and being largely dissipated in the form of heat in overcoming the viscous resistance to flow in the terminal blood vessels of the arterial system. Left ventricular stroke volume, and thus cardiac output, is largely determined by the interaction of the power generated by the left ventricle and the dissipation of that power in the systemic vascular system, thus alterations in the caliber of the arteriolar vessels may be a major factor in the causation of beat-to-beat changes in stroke volume.[10] When both variables, stroke volume and systemic vascular resistance, are changing simultaneously, it is unlikely that observations of arterial pressure will provide an adequate reflection of the change in either variable. While many[11,12,13] have claimed to be able to derive estimates of changes in stroke volume from calculations based on the pressure–pulse contour, McDonald[6] originally regarded this as a physiologists' El Dorado. Nevertheless, he and his colleagues[14,15] obtained excellent correlations between estimates of stroke volume derived from the pressure–pulse contour and those derived from electromagnetic flowmetry in the dog, and from dye-dilution curves in man. However, they stressed that where the changes in blood pressure resulted from drug-induced changes in systemic vascular resistance, the values of stroke volume derived from the pressure–pulse contour were completely unreliable.

Cullen's findings are based on correlations with amplitude values of the pressure pulse, and it is only to be expected that these cruder indices which we can measure every day would correlate less strongly with estimates of stroke volume, heart rate, or systemic vascular resistance. It is hardly surprising that the best correlations in Cullen's study were between stroke volume and systolic arterial pressure in the individual groups during halothane anesthesia. In this situation we have found a very strong correlation between stroke volume and mean arterial pressure during studies of the human dose-response for halothane,[16] a situation in which we have found negligible changes in systemic vascular resistance.[10] Conversely, the lack of correlation in Dr. Cullen's data, even between systolic and diastolic pressures, during hypercapnia, and during two levels of isovolemic anemia, lends further weight to the concept that in the presence of widespread arteriolar dilatation (lowered SVR), changes in arterial pressure amplitude will provide a much less reliable index of changes in circulatory state.

Where, therefore, lies the salvation of the clinical anesthetist who relies heavily on indirect, intermittent measurements of blood pressure? First, the anesthetist is looking for an index of change from the normal pattern of events, and recognizes that sudden hypertension or hypotension represents an acute change in cardiovascular behavior. He has learnt, by a process of calibration of his own

impressions, that these changes in blood pressure are well tolerated by normal patients, but poorly tolerated by those with cardiovascular disease. It is not necessary, in order to appreciate the significance of a change in arterial pressure, to infer whether changes in pressure result from those of either cardiac output or of systemic vascular resistance. However, in many clinical situations, the hemodynamic behavior of patients has been well established, and the value of correlations obtained in such research studies lies in the extrapolation of their prediction to the clinical situation. Thus, an anesthetist observing a fall in blood pressure during halothane anesthesia with mechanical ventilation may justifiably predict that the cause is related to a reduction of cardiac output rather than a change in resistance. It would appear from Dr. Cullen's data that systolic arterial pressure is a sufficiently good index upon which to calibrate our impressions of circulatory changes. At least we have advanced a great deal since the time when I started anesthesia and was chided thus by one of my early mentors: "... if you must measure the blood pressure, you've only yourself to blame if you get worried by hypotension."!

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