Arterial Waveforms and Systemic Vascular Resistance: Is There a Correlation?

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Clinicians use the contour of the arterial waveform to derive information regarding cardiovascular performance.[1,2] The location of the dicrotic notch on the arterial waveform tracing may be an indicator of systemic vascular resistance.[1-4] To test this hypothesis, we designed a study in which we evaluated the components of the radial artery waveform and their relation to systemic vascular resistance and cardiac output.

METHODS

Following a protocol approved by the Human Investigation Committee, 21 patients (mean age 63 ± 2.8 yr) undergoing elective coronary artery bypass surgery were evaluated. Patients excluded from the study were those who were not in sinus rhythm, who had valvular heart disease, or who required an intra-aortic balloon pump. All patients were anesthetized with fentanyl 50 mcg·kg^{-1} iv. Pancuronium iv was given to provide muscle relaxation, and F_{IO_2} was 1.0.

The radial arterial pressure was obtained from a percutaneously inserted catheter (20-gauge) which was connected to a Bentley Trantec (Model 800) transducer by 48 inches of high pressure tubing with a continuous flush device. The resonant frequency and damping coefficient of each system were determined, as described by Gardner.[5] Mean values were: for resonant frequency, 13.4 Hz ± 2.6 SD; and for damping coefficient, 0.33 ± 0.011 SD. Simultaneous electrocardiographic and pressure measurements (right atrial, pulmonary artery, pulmonary capillary wedge) were recorded on a strip chart recorder.

Thermodilution cardiac outputs were obtained in duplicate (Model 9520A Edwards Laboratory Cardiac Output Computer®). For each patient, a minimum of three sets of hemodynamic data were obtained. All measurements were recorded at end exhalation and prior to cannulation for cardiopulmonary bypass. Esophageal temperatures were between 36–37° C.

The height of the dicrotic notch (H) was measured from the baseline to point A (fig. 1). The slope of the diastolic run-off was determined by dividing the peak of the diastolic run-off (point B), by the length of segment B'C. Systemic vascular resistance (SVR) was calculated as mean blood pressure (BP) minus central venous pressure (CVP) divided by cardiac output (CO) × 80.

Data are presented as mean ± SD. Statistical analysis was performed using the coefficient of correlation; $P < 0.05$ was considered significant.

RESULTS

Eighty-nine sets of data were collected from these 21 patients (table 1). No statistically significant correlation was found between systemic vascular resistance and the height of the dicrotic notch (r = 0.28). In addition, the slope of the diastolic runoff correlated poorly with systemic vascular resistance (r = 0.18). There was no statistically significant correlation between the height of the dicrotic notch and cardiac output (r = 0.03).

Figures 2 and 3 obtained from the arterial tracings of patients in the present study highlight our data. In figure 2a and b, the arterial waveforms have different heights of the dicrotic notch: 24 mm and 18.5 mm, respectively. However, the calculated systemic vascular resistance associated with each waveform is similar, 1158 and 1192 dynes·sec·cm^{-5}, respectively. In contrast, another patient's arterial waveforms have the same value for the height of the dicrotic notch, 19 mm, but each is associated with significantly different systemic vascular resistance, 1136 and 1486 dynes·sec·cm^{-5}, respectively (fig. 3a,b).
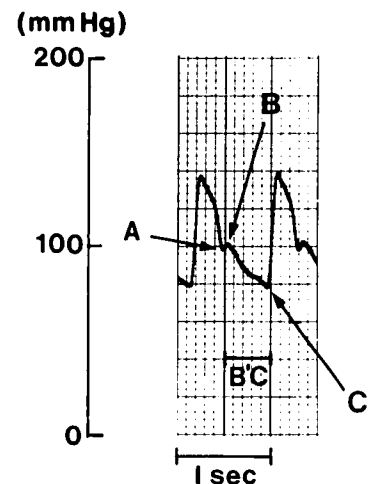
FIG. 1. Analysis of the arterial waveform. Components are defined as the dicrotic notch (A), peak diastolic run-off (B) and end of the diastolic run-off (C). The height of the dicrotic notch (H) is measured from the baseline to point A and the slope of the diastolic run-off is represented by B/B'C.

TABLE 1. Hemodynamic Variables

	Mean ± SD	Range
Cardiac output ($l \cdot min^{-1}$)	5.5 ± 0.8	2.9–8.5
Mean blood pressure	83.6 ± 11.1	57–120
Right atrial pressure (mmHg)	9.6 ± 136.3	1–22
Systemic vascular resistance ($dyne \cdot sec \cdot cm^{-5}$)	1,280 ± 136.3	404–1973
Heart rate (bpm)	62 ± 11.4	41–88

DISCUSSION

The components of the arterial waveform have been used to imply adequacy of hemodynamic performance; specifically, systemic vascular resistance.[1] In our patient population, however, we could not demonstrate a statistically significant relationship between the location of the dicrotic notch or the diastolic runoff (peripheral or arterial waveform) with systemic vascular resistance.

The dicrotic notch on a proximal aortic waveform is created by isovolumetric relaxation, closure of the aortic valve, sudden deceleration of blood flow, and diastolic runoff to more distal segments of the aorta. As the pressure wave moves towards the periphery, its contour undergoes modifications as a result of several factors. The most important of these factors is reflection of the pressure waves along certain segments of the aorta and at the periphery.[3,4,6] Due to the varying resistance and capacitance of vessels and the multitude of branch points within the vascular tree, reflection is an inherent characteristic to the arterial system. This produces resonant waves which are directed back toward the proximal aorta. The resulting arterial waveform, therefore, is a summation of initial and reflected waves.
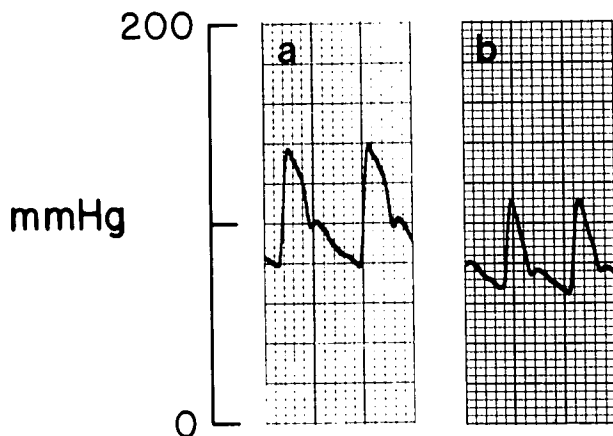

FIG. 2. Although waveforms *a* and *b* (same patient) have similar values for SVR (*a*. 1158 dynes·sec·cm⁻⁵; *b*. 1192 dynes·sec·cm⁻⁵), the height of the dicrotic notch (*a* = 24, *b* = 18.5) is different in the two tracings.
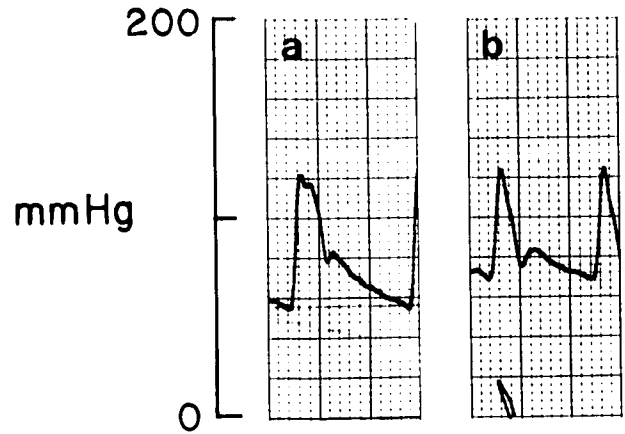

FIG. 3. Two waveforms (same patient) with similar heights of the dicrotic notch (*a* = 19, *b* = 19), but significantly different SVR (*a* = 1136 dynes.sec.cm⁻⁵, *b* = 1486 dynes.sec.cm⁻⁵).

Furthermore, at each site of reflection, the degree of reflection can vary. Latham *et al.* demonstrated that, at the renal arteries, a major site of reflection in the distal aorta, proximal reflection is reduced with the valsalva maneuver.[7] A similar physiologic situation may occur in the anesthetized patient receiving positive pressure ventilation. In addition, Murgo *et al.*[8] examined the shape of aortic waveforms in normal humans in response to alterations in impedance. They reported that no relationship could be found between differences in the configuration of the arterial waveform and cardiac function. Their data suggest that the differences in the arterial pressure waveform were due to reflections in the arterial system, rather than differences in cardiac function.[8]

In conclusion, the contour of the arterial waveform is a result of interaction of several variables, including distensibility and pulse wave velocity, as well as cardiac function. These variables modify the waveform from its inception at the aortic root, and continue to influence waveform contour to the level of peripheral arterial trace. Consequently, when the arterial contour changes, it is difficult to isolate the factor which is responsible. It is not surprising, therefore, that, under conditions of this study, we were unable to demonstrate any statistically significant correlation between the height of the dicrotic notch and: (1) systemic vascular resistance, (2) the diastolic runoff, or (3) cardiac output.

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Epidural Hydromorphone: A Double-blind Comparison with Intramuscular Hydromorphone for Postcesarean Section Analgesia

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Epidural narcotics, including morphine,[1-5] hydromorphone,[6,7] meperidine,[8] methadone,[9] and fentanyl[10] are effective analgesics after cesarean delivery. Epidural hydromorphone may be preferable to other narcotics, since it has a longer duration of action than epidural methadone,[9,11] meperidine,[8] and fentanyl,[10] and may have a lower incidence of side effects than morphine.[6,11,12]

Since Bromage et al.[11] first demonstrated the efficacy of epidural hydromorphone for postoperative analgesia following thoracic and upper and lower abdominal surgery, two other groups have described the use of epidural hydromorphone for postcesarean section analgesia. Albright,[6] in a study to assess the use of a respiratory apnea monitor following epidural narcotics, described effective postcesarean analgesia, with a mean duration of 6.2 h, using 1-1.25 mg epidural hydromorphone. Chestnut et al.[7] found 1 mg of epidural hydromorphone provided excellent analgesia with a mean duration of 13 h, at the cost of an increased incidence of pruritus, nausea, and vomiting when compared to epidural bupivacaine.

Comparison of im or iv administration with epidural administration of morphine or meperidine for postcesarean delivery analgesia has demonstrated improved duration and quality of analgesia following epidural administration.[3,5,8] The incidence of side effects with epidural and im administration has been comparable, with the exception of pruritus and urinary retention, which have a higher incidence following epidural narcotics.

While it would seem reasonable to assume that epidural administration is superior to im or iv administration for hydromorphone and other narcotics, this may not be true. For example, it appears that epidural methadone may not be superior to iv methadone for postoperative analgesia. Gourley et al.,[13] using iv methadone doses titrated to be just above the minimal effective concentration, achieved a mean duration of action of approximately 22 h following upper abdominal surgery. This long duration of action has been attributed to the long terminal elimination half-life of methadone. With epidural methadone, adequate pain relief has been obtained with lower doses than used by Gourley et al.,[13] but the duration of action was only 4.9-8.7 h.[9,11]

A prospective, randomized, double-blind, single-dose study was designed to test the hypothesis that epidural hydromorphone is superior to im hydromorphone for postcesarean delivery analgesia. The duration and quality of analgesia and the incidence of side effects are reported.

METHODS

The protocol was approved by our Institutional Review Board, and written informed consent was obtained from each patient upon entry into the study. The study design was a first dose only, non-crossover, placebo-controlled study, with randomized, double-blind assignment to the patient groups.

Postoperative analgesia was studied in 30 ASA physical status I or II patients who had undergone elective cesarean section under lumbar epidural anesthesia. Patients with major complications of pregnancy, major organ system disease, or a history of drug or alcohol abuse were ex-