Interpretation of Blood-pressure Measurements in Anesthesia

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Changes in systolic (SP), diastolic (DP), and pulse pressures (PP) caused by various stress-induced changes (hypoxia, anemia, hypercarbia, halothane, cyclopropane) in heart rate (HR), stroke volume (SV), and total peripheral resistance (TPR) were studied in dogs and man. Pooled data from 13 separate studies \( (n = 85) \) revealed highly significant correlations of \( \Delta SP \) with \( \Delta DP \) \( (r = 0.88) \), and \( \Delta SP \) with \( \Delta SV \) \( (r = 0.82) \) only. There was no correlation of \( \Delta SP \) or \( \Delta DP \) with either \( \Delta TPR \) or \( \Delta HR \). \( \Delta DP \) correlated poorly with \( \Delta SV \), and \( \Delta PP \) correlated poorly with all variables. Changes in TPR and SV could not be inferred from \( \Delta DP \) or \( \Delta PP \), respectively. Within each individual group, few correlations were found except for \( \Delta SP \) with \( \Delta DP \). Inferences made from diastolic or pulse-pressure measurements on the state of the circulation, particularly TPR and SV, are misleading and do not add to knowledge obtained from measurement of systolic pressure alone. (Key words: Blood pressure; pulse pressure; Blood pressure: systolic; Blood pressure: diastolic; Heart: stroke volume.)

SYSTEMIC arterial blood pressure is determined by interaction of cardiac output and total peripheral resistance. Indirect measurement of blood pressure generally provides three bits of information: first, systolic pressure, which depends on left ventricular stroke volume, peak rate of ejection, and aortic wall distensibility; second, diastolic pressure, which depends on the systolic pressure at which the system starts, total peripheral resistance, and diastolic time interval as determined by heart rate; third, pulse pressure, which represents a volume change per beat coupled with arterial capacitance.12

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The usefulness of systolic (SP), diastolic (DP) and pulse pressure (PP) measurements during clinical anesthesia is determined by their ability to reflect cardiovascular function. For example, in the isolated heart-lung preparation, changes in DP reflect changes in total peripheral resistance (TPR), and widening of PP signifies an increase in stroke volume (SV). However, in intact animals and man the interactions between various determinants of blood pressure render their interpretation difficult. If systolic, diastolic and pulse pressures are indeed determined by the factors mentioned above, then known changes in these factors should alter these pressures in a predictable manner.

This paper reports that changes in DP and PP caused by stress-induced changes in heart rate (HR), SV, and TPR in the intact animal, human volunteer, or patient are not consistent, may be misleading, and cause one to question the value of routinely measuring DP and PP. The stresses examined included acute isovolemic anemia, moderate and severe hypoxia, and five alveolar concentrations of halothane with or without hypoxia in dogs; hypercarbia in awake subjects, and two concentrations of cyclopropane in human volunteers; intravenous administration of morphine sulfate, and carotid thromboendarterectomy in patients.

**Methods**

In every volunteer and dog experiment, arterial blood pressure was transduced with a Statham P23 strain gauge and recorded on a Grass Model 7 polygraph. The strain gauge was calibrated statically with a mercury manometer. Cardiac output was measured by dye dilution with a Beckman Cardiodensitometer. Indocyanine green dye was injected into the right atrium and sampled from the brachial or radial artery in man, and the femoral artery in the dog, by continuous
withdrawal using a Harvard pump. HR was directly recorded with an electrocardiogram, or, in some of the dog experiments, from the direct recording of blood pressure. The values of SV and TPR were calculated. Preparations were similar in patient studies except the data were recorded on a Sanborn polygraph, and the concentration of indocyanine green dye was sampled through a Gilson densitometer.

**HYPERCARBIA** (GROUP I)

In each of seven healthy, conscious, unmedicated, male volunteers, ventilation was controlled to maintain $P_{aCO_2}$ at 35 torr. Following control measurements of cardiac output, heart rate, arterial pressure and arterial blood gases, $P_{aCO_2}$ was increased by adding CO$_2$ to inspired gas. Values for HR, SV, TPR, and systolic, diastolic, and pulse pressures were obtained at the highest $P_{aCO_2}$ achieved ($52 \pm 1.1$ torr) (1 SE).

**CYCLOPROPANE** (GROUPS II AND III)

In 11 healthy, unmedicated, young adult male volunteers, control measurements were obtained before cyclopropane anesthesia and after 15 or 20 per cent, and 35 or 40 per cent, end-tidal cyclopropane.

**ANEMIA** (GROUPS IV AND V)

Six healthy mongrel dogs were anesthetized with halothane and air. After control measurements had been obtained at MAC 1 halothane (1.1 per cent end-tidal halothane), exchange transfusion with Dextran induced iso-volemic anemia. Measurements were obtained at hematocrits of 24 and 10 per cent.

**HYPOXIA** (GROUPS VI AND VII)

Six healthy mongrel dogs were anesthetized with halothane-oxygen. After control values had been obtained at MAC 1 halothane (0.96 per cent end-tidal halothane), $P_{aO_2}$ was reduced to 30 torr, while $P_{aCO_2}$ was held constant. Measurements were recorded after 15 and 60 minutes of severe hypoxia.

**DOSE RESPONSES TO HALOTHANE WITH OXYGEN OR HYPOXIA** (GROUPS VIII-XIII)

Six healthy mongrel dogs were anesthetized with 1 per cent end-tidal halothane and oxygen. Control measurements were obtained at $P_{aCO_2}$ 34 torr during mechanical ventilation. Measurements were then recorded at 1.5 and 2 per cent halothane in oxygen. Then, $P_{aO_2}$ was reduced to 45 torr (moderate hypoxia).

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**Table 1.** Correlation Coefficients and Regression Equations for Blood Pressure and its Determinants  
(Pooled Data, Groups I –XIII, n = 85)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Variable</th>
<th>Correlation Coefficient ($r$)</th>
<th>$r^2$</th>
<th>Regression Equation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic pressure</td>
<td>Diastolic pressure</td>
<td>0.88*</td>
<td>0.77</td>
<td>$y = 1.13x + 2.35$</td>
</tr>
<tr>
<td>Systolic pressure</td>
<td>Pulse pressure</td>
<td>0.69*</td>
<td>0.49</td>
<td>$y = 0.7x + 0.07$</td>
</tr>
<tr>
<td>Diastolic pressure</td>
<td></td>
<td>0.3</td>
<td>0.09</td>
<td></td>
</tr>
<tr>
<td>Heart rate</td>
<td>Systolic pressure</td>
<td>0.04</td>
<td>0.002</td>
<td></td>
</tr>
<tr>
<td>Heart rate</td>
<td>Diastolic pressure</td>
<td>0.05</td>
<td>0.003</td>
<td></td>
</tr>
<tr>
<td>Heart rate</td>
<td>Pulse pressure</td>
<td>0.04</td>
<td>0.002</td>
<td></td>
</tr>
<tr>
<td>Stroke volume</td>
<td>Systolic pressure</td>
<td>0.82*</td>
<td>0.66</td>
<td>$y = 0.94x + 14.7$</td>
</tr>
<tr>
<td>Stroke volume</td>
<td>Diastolic pressure</td>
<td>0.68*</td>
<td>0.46</td>
<td>$y = 0.78x + 15.3$</td>
</tr>
<tr>
<td>Stroke volume</td>
<td>Pulse pressure</td>
<td>0.45*</td>
<td>0.2</td>
<td></td>
</tr>
<tr>
<td>Total peripheral resistance</td>
<td>Systolic pressure</td>
<td>0.05</td>
<td>0.003</td>
<td></td>
</tr>
<tr>
<td>Total peripheral resistance</td>
<td>Diastolic pressure</td>
<td>0.09</td>
<td>0.008</td>
<td></td>
</tr>
<tr>
<td>Total peripheral resistance</td>
<td>Pulse pressure</td>
<td>0.19</td>
<td>0.04</td>
<td></td>
</tr>
</tbody>
</table>

* $P < 0.05$.  
† $P < 0.001$ between r (stroke volume-systolic pressure) and r (stroke volume-diastolic pressure).
and data were recorded at 1, 1.5, and 2 per cent end-tidal halothane. Finally, \( P_{O_2} \) was reduced to 30 torr (severe hypoxia) and data were obtained at 0.75 per cent end-tidal halothane.

**Morphine Sulfate** (Groups XIV-XV)

In eight normal patients scheduled for major abdominal operations, control data were obtained during oxygen breathing. Thirty minutes after morphine sulfate, 1 mg/kg, repeat measurements were obtained.

In patients with aortic stenosis and/or aortic regurgitation, control measurements were obtained while the patients breathed \( O_2 \) by face mask; measurements were then obtained 20-30 minutes after morphine sulfate, 0.5 mg/kg, iv.

**Unilateral Carotid Thromboendarterectomy (Group XVI)**

Eleven patients undergoing unilateral carotid thromboendarterectomy were studied by Drs. R. Brons and J. Gabel in our laboratories. Measurements were obtained prior to anesthesia and operation and repeated approximately eight hours after operation. In some patients, a phenylephrine drip was necessary in addition to vigorous hydration to maintain adequate blood pressure intraoperatively as well as postoperatively.

**Statistical Analysis**

Pooled data from Groups I-XIII (human volunteer and dog studies), as well as data for each individual group, were expressed as per cent change from control and were analyzed by linear regression. The patient studies were not included in the analysis of the pooled data, but each group was analyzed individually. Correlation coefficients were computed on an Olivetti Programma 101 and tested for significance. When appropriate, differences between correlation coefficients were tested with a method described by Williams.

**Results**

**Pooled Data, Table 1**

The correlation coefficient \( r \) for change in systolic and diastolic pressure was 0.88 (fig. 1). It thus follows that correlations of diastolic pressure with HR, SV, and TPR will be somewhat similar to correlations with systolic pressure because of the dependency of diastolic pressure on systolic pressure \( (r^2 = 0.77).^\dagger \) \( \Delta \)Systolic pressure correlated well with \( \Delta SV \) \( (r = 0.82) \), while \( \Delta \)diastolic pressure correlated less well with \( \Delta SV \) \( (r = 0.68) \). The difference between these two correlation coefficients was highly significant \( (P < 0.001) \). There was no correlation between \( \Delta \)systolic pressure or \( \Delta \)diastolic pressure and

\[ y = 1.13X + 2.35 \]
\[ r = 0.875 \]
\[ r^2 = 0.765 \]
ΔTPR (fig. 2) or ΔHR. Combining ΔTPR and ΔHR together to correlate with Δdiastolic pressure yielded an r of 0.15, implying that 2 per cent of the change in diastolic pressure depended on changes in both HR and TPR ($r^2 = 0.02$). ΔPulse pressure correlated poorly with ΔSV ($r = 0.45$), significantly less than either Δsystolic pressure or Δdiastolic pressure with ΔSV.

**INDIVIDUAL GROUPS, TABLES 2 AND 3**

Within individual groups, where n was only 5 to 11, ΔSP and ΔDP correlated well (table 2). Otherwise, only ten of 96 other possible correlations were significant (table 3). Only during halothane anesthesia with hypoxia did ΔSV correlate with ΔSP or ΔDP. In the eight normal patients who received morphine sulfate, 1 mg/kg, the only significant correlation was that of systolic pressure with diastolic pressure. No correlation existed in the six patients who had aortic-valve disease. However, in this group, mean change in systolic pressure was $-2 \pm 0.9$ per cent (1 SE) and mean change in diastolic pressure was $-0.5 \pm 3$ per cent. Thus, one could not expect significant correlations when so little change occurred in BP. In patients undergoing carotid TEA, the correlation coefficient between ΔSP and ΔDP was 0.86. The correlations for ΔSV–ΔSP (0.67) and ΔSV–ΔPP (0.70) were far poorer, although statistically significant.

During administration of 15–20 per cent cyclopropane to 11 human volunteers, TPR and DP both increased, while HR and SV were unchanged. Yet r was only 0.22 for ΔTPR vs. ΔDP, compared with 0.83 for ΔSP vs. ΔDP.
TABLE 2. Correlation Coefficients Within Individual Groups for ΔSystolic Pressure–ΔDiastolic Pressure

<table>
<thead>
<tr>
<th>Experimental Group</th>
<th>Correlation Coefficient</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Hypercarbia</td>
<td>0.56</td>
<td>7</td>
</tr>
<tr>
<td>II. 15–20 per cent cyclopropane</td>
<td>0.83*</td>
<td>11</td>
</tr>
<tr>
<td>III. 35–40 per cent cyclopropane</td>
<td>0.9*</td>
<td>9</td>
</tr>
<tr>
<td>IV. Anemia (Het 24 per cent)</td>
<td>0.2</td>
<td>6</td>
</tr>
<tr>
<td>V. Anemia (Het 10 per cent)</td>
<td>0.45</td>
<td>6</td>
</tr>
<tr>
<td>VI. Hypoxia 15 min</td>
<td>0.91*</td>
<td>5</td>
</tr>
<tr>
<td>VII. Hypoxia 1 hour</td>
<td>0.88*</td>
<td>5</td>
</tr>
<tr>
<td>VIII. 1.5 per cent halothane–O2</td>
<td>0.92*</td>
<td>6</td>
</tr>
<tr>
<td>IX. 2 per cent halothane–O2</td>
<td>0.83*</td>
<td>6</td>
</tr>
<tr>
<td>X. 1 per cent halothane, Pao2 45 torr</td>
<td>0.98*</td>
<td>6</td>
</tr>
<tr>
<td>XI. 1.5 per cent halothane, Pao2 45 torr</td>
<td>0.96*</td>
<td>6</td>
</tr>
<tr>
<td>XII. 2 per cent halothane, Pao2 45 torr</td>
<td>0.89*</td>
<td>6</td>
</tr>
<tr>
<td>XIII. 0.75 per cent halothane, Pao2 30 torr</td>
<td>0.81</td>
<td>6</td>
</tr>
<tr>
<td>XIV. Morphine sulfate, normal patients</td>
<td>0.87*</td>
<td>8</td>
</tr>
<tr>
<td>XV. Morphine sulfate, patients with aortic-valve disease</td>
<td>0.47</td>
<td>6</td>
</tr>
<tr>
<td>XVI. Carotid TEA patients</td>
<td>0.86*</td>
<td>11</td>
</tr>
</tbody>
</table>

* P < 0.05.

Discussion

During several different experimental stresses, in intact dogs and man, the change in diastolic blood pressure did not correlate with the changes in HR and TPR, the two prime determinants of diastolic pressure. Systolic pressure correlated well with SV, one of its prime determinants, but correlated not at all with HR and TPR. In fact, the best correlation observed was between systolic and diastolic pressures themselves, suggesting that any independent variable which affects one component of systemic arterial pressure affects the other almost equally. Hence, can diastolic pressure and calculated pulse pressure yield better understanding of cardiovascular function during anesthesia than knowledge of systolic pressure alone?

TPR is considered a determinant of diastolic pressure, yet the lack of correlation between change in TPR and change in diastolic pressure suggests that changes in diastolic pressure cannot be used to infer changes in peripheral arterial tone. Since the same holds true for pulse pressure, the major usefulness of diastolic pressure is to calculate mean pressure, something rarely done in clinical anesthesia. Since diastolic and systolic pressure correlate so well with each other, calculation of mean pressure is unlikely to yield additional information except in patients with aortic-valve disease.

Recently, analysis of data from the Framingham study on hypertensive heart disease demonstrated that systolic pressure correlated slightly better than diastolic pressure with incidence of congestive heart failure. Assuming accurate, direct intra-arterial measurement of the blood pressure, these poor correlations obtained in well-controlled experiments demonstrate that many factors affect the autonomic nervous system to render useful interpretation of blood pressure difficult, if not impossible. In addition, clinical anesthesia introduces other variables that confuse matters further. These include premedication, induction agents, narcotics, muscle relaxants, changes in Pao2 and body temperature, patient’s age, nature and extent of illness, body position, blood volume, mental status if awake, surgical stimulation if anesthetized, type of inhalation anesthetic, depth of anesthesia, and duration of anesthesia. However, one cannot assume that direct measurement of blood pressure is entirely accurate. Preliminary experiments in our department by Dr. J. Bruner, with dynamic calibration of strain gauges (which simulate an arterial pulse wave striking the transducer), demonstrate overshoot of the peak pressure wave, yielding a falsely high systolic pressure. This probably relates to the frequency response and harmonics of the tubing, rather than the transducer itself. The form and transmission of the pulse wave itself are subject to several variables, rendering interpretation less accurate. These difficulties are partially obviated by having each dog, subject, or patient serve as his own control, but to some extent, direct blood-pressure measurement remains inaccurate.

In clinical practice, indirect measurement of blood pressure is also fraught with error. Perhaps even more important is the fact that diastolic pressure is less accurate than systolic pressure.
pressure when both are measured indirectly.\textsuperscript{16-18} This discrepancy is exaggerated further as hypotension develops.

The correlation of \(\Delta Systolic\) pressure with \(\Delta SV\) was quite good, significantly better than the correlation of \(\Delta Diastolic\) pressure with \(\Delta SV\). It should be emphasized that this is not necessarily a cause-and-effect relationship, but may result from a third factor that acts on systolic pressure and on SV independently.

Pulse pressure correlated poorly with all measured variables. Pulse pressure is said to increase when factors which raise systolic pressure are present. Yet the one factor (\(\Delta SV\)) which correlated with \(\Delta Systolic\) pressure related poorly, if at all, to pulse pressure. Thus, a widening or narrowing of pulse pressure is as difficult to interpret as a change in diastolic pressure.

Data obtained in the patient studies further demonstrate that systolic and diastolic pressures correlated well only with each other (except in patients with aortic stenosis who received morphine sulfate, in whom blood-pressure changes were minimal). Other variables correlated poorly with systolic, diastolic, and pulse pressures.

Obviously other factors besides SV, TPR, and HR contribute to a given level of blood pressure. These include ejection rate and blood-vessel compliance.\textsuperscript{12}

In clinical practice of anesthesia, routine measurement of blood pressure allows one to monitor and maintain, if necessary, an adequate perfusion pressure. Inferences made from diastolic or pulse pressure may be misleading and do not add to knowledge obtained from systolic pressure alone. Perhaps only systolic pressure need be monitored. If so, this can easily, quickly, and most accurately be accomplished by oscillometry rather than by auscultation or palpation.\textsuperscript{16}

Dr. Gene Smith (Associate Professor of Psychology in Anesthesia) and Dr. William G. Cochran (Professor of Statistics) reviewed the statistical treatment of the data. Drs. Richard J. Kitz, Myron B. Laver, M. Jack Fruhin, and Edmond I. Eger, II, aided in final preparation of the manuscript.

\textbf{References}

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Metabolism

ALKALOSIS IN CRITICALLY ILL PATIENTS Of 1415 critically ill patients, 177 developed arterial pH's in excess of 7.55. Of these, 142 were assisted by respirators, 57 had severe trauma, and 52 were severely septic. Forty had cardiopulmonary-bypass surgery. The alkalosis was designated metabolic when PCO2 was above 35 mm Hg and standard bicarbonate 27 mEq/l or higher; respiratory when PCO2 was 34 mm Hg or less and bicarbonate 26 mEq/l or less; and combined when the PCO2 was 34 mm Hg or less and bicarbonate more than 27 mEq/l. The number of patients with each type was: combined, 90; respiratory, 60; metabolic, 27. The mortality was highest in the combined type, with the highest pH levels (80 per cent of those over 7.64).

The authors note that the incidence of metabolic acidosis has decreased because of vigorous early therapy with blood and electrolyte solutions. Respiratory alkalosis is attributed to spontaneous hyperventilation caused by excessive anxiety, pain or early hypoxia, and then to vigorous use of mechanical ventilators. Metabolic alkalosis may also be caused by the administration of large quantities of citrated blood, bicarbonate- and lactate-containing solutions, and diuresis with potassium loss, and/or nasogastric suction.

When alkalosis develops it should be treated vigorously. The administration of alkalinizing agents should be avoided and gastric suction stopped if and when possible. The chloride deficit should be corrected using ¼ KCl, ¼ arginine chloride, and ½ sodium chloride. When alkalosis is respiratory in nature it should be treated by the addition of mechanical deadspace or a decrease in minute volume, or both. The patient may try to breathe too much; use morphine, diazepam, or paralyzing drugs to reduce respiratory efforts. (Wilson, R. F., and others: Severe Alkalosis in Critically Ill Surgical Patients. Arch. Surg. 105: 197–203, 1972.) EDITOR'S NOTE: Do not treat arterial pH but the source of its change! Moreover, a patient's attempt to "breathe too much" must be treated by direct attention to the cause, not respiratory depression with narcotics. The argument is similar to that used for hyperpyrexia: treat the cause of the elevated temperature, do not simply attempt to lower body temperature.