Circulatory Response to Intra-abdominal Manipulation During Ether Anesthesia in Man

By Leroy D. Vandam, M.D., Hans J. Schweizer, M.D., and Yukio Kubota, M.D.

In the course of anesthesia for abdominal operations, a pronounced decrease in arterial blood pressure is occasionally detected as manipulation within the abdomen is performed. Most observers have thought that this vascular response was reflex in origin; it has been variously called the celiac, splanchnic, or abdominal traction reflex. Other than changes in blood pressure, pulse, and the electrocardiogram, little is known of the circulatory alterations underlying this reflex nor of the receptors, neural pathways, or the influence of anesthesia on its development. In a previous report from this laboratory, the reflex origin of hypotension during various types of anesthetic management was reaffirmed and the frequency of appearance and probable receptor sites described. The stimuli consisted, for the most part, of rubbing the parietal peritoneum in the upper abdomen or occasionally traction on the gall bladder and stomach. The present report details the hemodynamic changes found in a group of patients studied during ether anesthesia before the major part of the operation was performed. An understanding of these circulatory changes is of importance in the prevention and treatment of hypotension during anesthesia and may cast light upon the role of abdominal receptors in general circulatory regulation.

Methods

Thirty-eight patients, 26 women and 12 men, from 23 to 73 years of age, were studied during ether anesthesia and abdominal operation. These subjects were in good physical condition without cardiovascular or pulmonary disease, except for one individual (Y.H., Table 2) with hypertension. Prenesthetic medication consisted of 50 to 150 mg. of pentobarbital by mouth one to two hours preoperatively, and 0.3 to 0.4 mg. of atropine hypodermically one hour before induction of anesthesia. Anesthesia was induced with 50 to 100 mg. of thiopental intravenously followed by nitrous oxide-oxygen and ether for maintenance using a closed-circle carbon dioxide absorption inhalation system. In most cases, endotracheal intubation was performed within a minute after an intravenous injection of 40 to 60 mg. of succinylcholine, and respirations were subsequently spontaneous in most instances. Breathing was assisted or controlled only when indicated.

In most cases, baseline circulatory measurements were made with the patient lying supine in a quiet induction room before induction of anesthesia and repeated in all cases after a presumed steady state of anesthesia had been reached prior to opening of the peritoneum. There was a range of from 30 minutes to one hour after induction of anesthesia, and the attainment of a steady state was confirmed by circulatory parameters, clinical and electroencephalographic signs of depth of anesthesia, and measurement of arterial blood ether levels. The intra-abdominal stimuli consisted of rubbing the parietal peritoneum with gauze sponges, traction on the stomach, gall bladder or mesenteries, or pressure applied to the celiac plexus. These stimuli were applied successively in all cases until one of the manipulations produced significant hypotension. When maximal hypotension was produced, circulatory measurements were again performed.

CIRCULATORY MEASUREMENTS

Peripheral arterial pressure was measured by either a PE 160/S36 polyethylene catheter† or an 18-gauge Courmand needle inserted into the brachial artery percutaneously or after direct dissection.

†Table 2 has been deposited as document number 7196 with the ADI Auxiliary Publications Project, Photoduplication Service, Library of Congress, Washington 25, D.C. A copy may be obtained by citing the document number and by remitting $1.25 for photoprints, or $1.25 for 35 mm. microfilm. Advance payment is required. Make checks or money orders payable to: Chief, Photoduplication Service, Library of Congress.

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tion of the artery. A similar catheter was inserted through a 14-gauge thin-walled needle into an antecubital vein and threaded centrally for measurement of venous pressure at the superior vena cava level. This was determined by pressure measurement and observation of the length of catheter inserted. The level for central venous pressure was established at 5 cm below the angle of the sternum. Pressures were transduced by Statham P23D strain gauges and recorded on a Grass Model 5 direct ink-writing oscillograph. Mean arterial blood pressures were obtained by electrical integration. Simultaneously, tracings of the electroencephalogram were made by fronto-occipital leads, and pulse rate was determined from a continuous recording of lead II of the electrocardiogram. Electroencephalographic determination of the level of anesthesia was determined according to the criteria of Faulconer.

Cardiac output, mean circulation time, and intrathoracic blood volume were determined by the dye-dilution principle of Stewart and Hamilton, employing indocyanine green as the indicator. A Waters cuvette oximeter inserted into the arterial line and a Grass modification of the Waters oximeter control circuit were used to obtain dye-dilution curves on the direct writer. To sample arterial blood, a Harvard Apparatus Company constant rate infusion-withdrawal apparatus was used and the blood reinfused after each determination. In order to estimate the precision of the method with these modifications, duplicate determinations of cardiac output were performed on nine subjects in a basal state. The standard deviation of the differences within individual determinations was ±2.4 per cent (table 1), a finding that agrees with the findings of Etsten et al. We are unable to account for the finding that the second cardiac output is at least equal to, or higher than, the first measurement. This suggests a systematic difference in the two determinations. Cardiac output and stroke volumes were expressed as both absolute values and their respective indices by dividing the actual values by the surface area of the subject in square meters.

Total peripheral resistance (T.P.R.) was calculated according to the formula of Aperia:

\[
\text{T.P.R. (dyne-sec/cm}^5) = \frac{\text{mean arterial blood pressure \times 1,332}}{\text{cardiac output cc/sec}}.
\]

In calculating peripheral resistance, the brachial artery mean pressure was assumed to be the same as that in the aorta.

**ARTERIAL BLOOD ANALYSES**

Blood gas studies were performed on arterial blood samples drawn aseptically into heparinized syringes and the analyses immediately made. Duplicate readings of pH were made with a Beckman Model G pH Meter and corrected to 37 C. Duplicate determinations of \(O_2\) and \(CO_2\) content and \(O_2\) saturation were made by Goldstein's modification of the manometric method of Van Slyke and Neill. Carbon dioxide tension was read from the nomogram of Singer and Hastings. Arterial ether concentrations were measured by a spectrophotometric modification of the method of Price and Price.

The probability, or \(P\) value, for the various data was obtained by means of the \(t\)-test.

**Results**

Hypotensive responses to intra-abdominal stimulation occurred in only 8 of the 38 patients studied. The unpredictability of the appearance of hypotension indicates the difficulty in making circulatory measurements under these circumstances. Since we were interested only in those cases showing circulatory changes, the pertinent data from the eight patients, before and after stimulation, are summarized in table 2; mean values, the standard deviation and standard error of the mean, the mean percentage changes, and probability of \(P\) values are shown in table 3. Electroencephalographic levels and arterial blood ether concentrations at the time of stimulation are shown in table 4.

It should be pointed out that certain changes of statistical significance were to be expected since only those patients showing obvious changes are reported. In some cases, the circulatory changes disappeared rather promptly after withdrawal of the stimulus, while in others injection of ephedrine in various dosages was required to correct the hypotension.

**BLOOD PRESSURE**

**Systolic Pressure**

The characteristic fall of pressure took place after intraperitoneal stimulation within a matter of seconds. The mean systolic blood pressure before the induction of anesthesia

*See footnote, page 287, second column.

*Cordis-green, Hynson, Westcott and Dunning, Inc., Baltimore, Maryland.
TABLE 1

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Sex</th>
<th>Body surface area M²</th>
<th>(I) Cardiac output L/min.</th>
<th>(II) Cardiac output L/min.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. R.M.</td>
<td>59</td>
<td>M</td>
<td>1.60</td>
<td>4.73</td>
<td>4.83</td>
</tr>
<tr>
<td>2. R.M.</td>
<td>59</td>
<td>M</td>
<td>1.60</td>
<td>4.60</td>
<td>4.77</td>
</tr>
<tr>
<td>3. M.G.</td>
<td>59</td>
<td>M</td>
<td>1.69</td>
<td>7.87</td>
<td>8.08</td>
</tr>
<tr>
<td>4. W.E.</td>
<td>55</td>
<td>F</td>
<td>1.48</td>
<td>4.31</td>
<td>4.31</td>
</tr>
<tr>
<td>5. G.C.</td>
<td>56</td>
<td>M</td>
<td>1.95</td>
<td>4.84</td>
<td>4.92</td>
</tr>
<tr>
<td>6. G.B.</td>
<td>68</td>
<td>F</td>
<td>1.60</td>
<td>3.81</td>
<td>3.98</td>
</tr>
<tr>
<td>7. Y.H.</td>
<td>62</td>
<td>F</td>
<td>1.88</td>
<td>4.19</td>
<td>4.38</td>
</tr>
<tr>
<td>8. P.E.</td>
<td>39</td>
<td>M</td>
<td>1.74</td>
<td>3.77</td>
<td>3.97</td>
</tr>
<tr>
<td>9. J.H.</td>
<td>31</td>
<td>F</td>
<td>1.60</td>
<td>4.65</td>
<td>4.87</td>
</tr>
</tbody>
</table>

*Standard deviation for the difference within individuals was 2.4 per cent.

was 139 mm. Hg, with a standard deviation of ± 31 and a range between 190 and 105 mm. Hg. After attainment of a steady anesthetic state there was little change in the pressure, the mean being 137 (S.D. ± 32) and the range between 140 and 80 mm. Hg. After stimulation, however, the mean systolic pressure fell to 75 (S.D. ± 12), a change of 45 per cent which was statistically significant (P < 0.001). The fall in systolic pressure tended to be greater in those patients with an initial higher level of pressure. In the hypotensive state, the pressure ranged from 76 to 48 mm. Hg in most instances, with a mean pressure hardly sufficient for myocardial or cerebral perfusion. The change in systolic pressure suggested an underlying diminution in cardiac output.

**Diastolic Pressure**

The mean diastolic pressure in the resting state was 77 (S.D. ± 12), again hardly altered after a period of ether anesthesia when the mean pressure was 78 (S.D. ± 13) mm. Hg. The pressures ranged from 100 to 65 mm. Hg. Upon stimulation, however, the mean diastolic pressure fell to 48 (S.D. ± 10) mm. Hg, a statistically significant alteration of 35 per cent (P < 0.001). The range lay between 65 and 35 mm. Hg. This fall in diastolic pressure was less than that of the systolic pressure, the percentage figure being misleading because of the initially lower level of diastolic pressure. Although the fall in diastolic pressure suggested vasodilation and a decrease in peripheral resistance, this was not confirmed by the calculated total peripheral resistance.

**Mean Arterial Pressure**

The decreases in systolic and diastolic pressures were reflected in a change in the mean pressures following stimulation, the mean arterial pressure falling to dangerously low levels. Thus the mean pressures of 102 (S.D. ± 21) in the resting state, and 99 (S.D. ± 25) mm. Hg after maintenance in ether anesthesia, fell to 57 (S.D. ± 10), a fall of 41 per cent (P < 0.001).

**Pulse Pressure**

Largely as a reflection of the fall in systolic pressure, greater than the diastolic pressure, pulse pressure decreased, being 62 (S.D. ± 27) mm. Hg before anesthesia, 59 (S.D. ± 22) after maintenance in ether anesthesia, and 27 (S.D. ± 8) after hypotension was produced. This was a statistically significant change of 54 per cent (P < 0.005).

**Pulse Rate**

It was not possible to make detailed electrocardiographic analyses since the paper speed for simultaneous recording of dye-dilution curves allowed only a counting of the pulse rate. The mean pulse rate before induction of anesthesia was 91 (S.D. ± 18), essentially unchanged after anesthesia, 89 (S.D. ± 10), with a range between 102 and 76 before stimulation. After the appearance of hypotension, the mean pulse rate was 76 (S.D. ± 9), a fall
## TABLE 3
Average Circulatory Values Before Anesthesia, After Induction of Anesthesia, and After Stimulation

<table>
<thead>
<tr>
<th></th>
<th>A. Baseline</th>
<th>B. Prestimulation</th>
<th>Per cent change between A and B</th>
<th>C. Response</th>
<th>Per cent change between B and C</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic pressure (mm. Hg)</td>
<td>139 ± 31 (S.D.)†</td>
<td>137 ± 32 (S.D.)†</td>
<td>- 1</td>
<td>75 ± 12 (S.D.)</td>
<td>-45</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diastolic pressure (mm. Hg)</td>
<td>77 ± 12 (S.D.)†</td>
<td>78 ± 13 (S.E.)†</td>
<td>+ 1</td>
<td>48 ± 10 (S.E.)</td>
<td>-35</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Pulse pressure (mm. Hg)</td>
<td>62 ± 27 (S.D.)†</td>
<td>59 ± 22 (S.E.)†</td>
<td>- 5</td>
<td>27 ± 8 (S.E.)</td>
<td>-54</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Mean arterial pressure (mm. Hg)</td>
<td>102 ± 21 (S.D.)†</td>
<td>99 ± 25 (S.E.)†</td>
<td>- 3</td>
<td>57 ± 10 (S.E.)</td>
<td>-42</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Pulse rate (beats/min.)</td>
<td>91 ± 18 (S.D.)†</td>
<td>89 ± 10 (S.E.)†</td>
<td>- 2</td>
<td>76 ± 9 (S.E.)</td>
<td>-15</td>
<td>&lt;0.025</td>
</tr>
<tr>
<td>Mean circulation time (seconds)</td>
<td>15.3 ± 3.3 (S.D.)†</td>
<td>18.7 ± 4.2 (S.E.)†</td>
<td>+22</td>
<td>28.2 ± 1.8 (S.E.)</td>
<td>+51</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Cardiac output (L/min.)</td>
<td>4.16 ± 0.38 (S.D.)†</td>
<td>3.92 ± 0.99 (S.E.)†</td>
<td>- 6</td>
<td>2.56 ± 0.70 (S.E.)</td>
<td>-35</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Cardiac index (L/min./M.²)</td>
<td>2.48 ± 0.24 (S.D.)†</td>
<td>2.34 ± 0.59 (S.E.)†</td>
<td>- 6</td>
<td>1.50 ± 0.59 (S.E.)</td>
<td>-36</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Stroke volume (cc.)</td>
<td>47 ± 9 (S.D.)†</td>
<td>45 ± 12 (S.E.)†</td>
<td>- 4</td>
<td>34 ± 9 (S.E.)</td>
<td>-24</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Stroke index (cc./M.²)</td>
<td>28 ± 5 (S.D.)†</td>
<td>27 ± 7 (S.E.)†</td>
<td>- 4</td>
<td>20 ± 4 (S.E.)</td>
<td>-26</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Total peripheral resistance (dynes-sec./cm.²)</td>
<td>1997 ± 404 (S.D.)</td>
<td>2145 ± 625 (S.E.)</td>
<td>+ 7</td>
<td>1965 ± 1074 (S.D.)</td>
<td>-8</td>
<td>&gt;0.6</td>
</tr>
<tr>
<td>Central blood volume (L.)</td>
<td>1.06 ± 0.25 (S.D.)†</td>
<td>1.22 ± 0.40 (S.E.)†</td>
<td>+15</td>
<td>1.20 ± 0.36 (S.E.)</td>
<td>-2</td>
<td>&gt;0.9</td>
</tr>
<tr>
<td>Central blood volume index (cc./M.²)</td>
<td>627 ± 116 (S.D.)</td>
<td>719 ± 201 (S.E.)</td>
<td>+15</td>
<td>701 ± 166 (S.D.)</td>
<td>-3</td>
<td>&gt;0.8</td>
</tr>
</tbody>
</table>

*S.D.=Standard deviation.
†S.E.=Standard error of the mean.
of 15 per cent ($P < 0.025$). It was interesting that there was not an overall increase in the pulse rate, as is often seen during hypotension. The pulse rose in two cases, and in the others the lowest value was only 60 per minute. Thus, it can hardly be said that bradycardia was produced, and from the pulse rate change alone it could not be assumed that a vagal influence was involved. The rate changes were in the range normally observed during anesthesia.

**CARDIAC OUTPUT**

The absolute cardiac output in the resting state ranged between 4.83 and 3.74 L/min. with a mean value of 4.16, a figure slightly lower than the average reported in the literature but still within the range reported by various observers and that usually found in our laboratory. It was interesting that, after attainment of surgical planes of ether anesthesia, the cardiac output ranged between 5.34 and 2.20 with no overall change in the mean values. The circulatory changes following ether anesthesia will form the subject of a separate communication. The mean cardiac index before anesthesia was 2.48 (S.D. ± 0.24) and after anesthesia, 2.34 (S.D. ± 0.59), a change of 6 per cent, not one of significance. However, after hypotension appeared, the mean cardiac index fell to 1.50 (S.D. ± 0.29), a remarkably low value representing a significant change of 36 per cent ($P < 0.005$) (fig. 1). Rather than a change in peripheral resistance, this may be considered the basis for the decrease in blood pressure.

**STROKE VOLUME AND STROKE INDEX**

The stroke volume and stroke index were unaltered between the resting and anesthetic states. However, after hypotension appeared, the stroke volume decreased by 24 per cent, from 45 (S.D. ± 12) ml./min. to 34 (S.D. ± 9), and the stroke index by 26 per cent, from 27 (S.D. ± 7) to 20 (S.D. ± 4), both remarkably low and statistically significant at $P$ values less than 0.05 and 0.01, respectively. Thus, the decline in cardiac output seemed to be more a reflection of a change in the stroke volume than in pulse rate. However, in case 3 (table 2)* with little change in the stroke volume the decrease in output was primarily due to the decline in pulse rate, and in subjects 4 and 7 the change in pulse rate contributed significantly to the decrease in output.

**MEAN CIRCULATION TIME**

As expected from the circulatory changes already enumerated, the mean circulation time increased by 53 per cent, after the hypotensive state was reached, from a mean value of 18.7 (S.D. ± 4.2) minutes to 28.2 seconds (S.D. ± 1.8) ($P < 0.001$).

**TOTAL PERIPHERAL RESISTANCE**

The mean calculated T.P.R. during the resting and prestimulation states was 1,997 (S.D. ± 404) and 2,145 (S.D. ± 625) dynes-sec./cm.$^5$, respectively, indicating a slight, but not statistically important, increase of 7 per cent following ether anesthesia. It is of importance that no significant difference was found in T.P.R. with the appearance of hypotension; T.P.R. was 1,965 (S.D. ± 1,074). This represented a change of only 8 per cent, but the standard deviations are large, suggesting that many circulatory variables may have been involved. That the calculated T.P.R. did not change strengthens the concept that hypotension produced by abdominal manipulation

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*See footnote, page 287, second column.*
Average changes in cardiac output and stroke volume in response to abdominal stimulation.

is largely due to a decrease in cardiac output, but does not rule out changes in resistance to blood flow in specific circulatory beds.

CENTRAL BLOOD VOLUME

There was no significant alteration in the central blood volume after maintenance in ether anesthesia or when the hypotensive response appeared, the values being 1.22 L ± 0.4 S.D., and 1.20 ± 0.36, respectively. In both instances, there were individual changes in either direction. Changes in central venous pressure were not consistent.

Discussion

The hemodynamic changes described in this report relate to a specific situation, the circulatory response to intra-abdominal manipulation during ether anesthesia in man. This point is emphasized because there has been confusion in the description of the traction reflex. The reason is that the circulatory change has usually been noticed by chance during various types of anesthetic management. Under these circumstances, there has not been an opportunity to make detailed circulatory studies, the hypotensive response being recorded simply by auscultation. Studies in animals have been few and poorly controlled from the standpoint of interpreting the circulatory alterations. Despite these deficiencies, most observers have maintained that the arterial hypotension resulting from intra-abdominal manipulation is reflexly produced, the fall in pressure largely the result of systolic hypotension, and that bradycardia is relatively infrequent. The remaining question is the nature of the circulatory alteration.

In this study, hypotension produced by abdominal manipulation was seen infrequently and unpredictably. Moreover, in each patient in whom hypotension was induced, the same type of stimulus did not necessarily lead to the hypotensive response. Various manipulations were tried until hypotension was in evidence. It is conceivable, therefore, that the response may have been mediated in different ways and that the stimulus could lead to hypotension through varying mechanisms. This also suggests that the reflex is not an ordinary type of circulatory adjustment and that its appearance may relate more to the condition of the patient studied or to the anesthetic utilized. Indeed, the circulatory response may have been enhanced by ether. However, we believe the circulatory response was due basically to abdominal stimulation since the conditions of anesthesia were moderately well controlled. A presumed steady state of anesthesia had been reached, pulmonary ventilation was close to normal levels, and the operation had not advanced beyond opening of the peritoneum. Thus, a hypotensive response probably was not due to a change in the depth of anesthesia, to the placement of abdominal retractors or gauze packs, or to blood loss, the common causes of hypotension during operation. It can also be argued that the reflex might be more active in the unanesthetized normal individual, hence its unpredictability and infrequency in the group of individuals studied.

It is possible that the circulatory changes found may be related to pain subconsciously.
perceived and to activating parasympathetic mechanisms. Likewise, this could have been the same type of stimulus response phenomenon leading to vomiting and hypotension in lighter planes of general anesthesia. It is frequently seen during manipulation in the upper abdomen during spinal anesthesia.

Hypotension was largely due to a fall in systolic blood pressure with a lesser fall in the diastolic pressure and a consequent decrease in pulse pressure. This change has, in the past, suggested a decrease in cardiac output, as indicated by a change in pulse pressure contours in continuous recordings of arterial blood pressure and, in one instance, by alteration in the ballistocardiogram. A statistically significant fall in cardiac output was demonstrated during all of the hypotensive episodes observed in the present study. Although this seemed largely due to a decrease in stroke volume, slowing of the pulse in several cases contributed significantly to the change in output. A decrease in pulse rate, however, was an inconsistent finding. Furthermore, the protocol of these experiments was not designed either by method employed or time interval chosen to detect rapid or early changes responsible for the final steady state measurements that were made.

It remains to speculate on how the stroke volume and the cardiac output might, in the absence of change in pulse rate, have been altered by this vascular abdominal reflex initiated from within the abdomen. By analogy, the action of the carotid and aortic pressor receptors may be recalled. Stimulation of these stretch receptors leads to reflex inhibition of sympathetic discharge or to increased parasympathetic action, in either case the result being a negative inotropic effect on the myocardium with a fall in cardiac output. In relation to vagal activity, a negative inotropic effect can be exerted only on the atrial myocardium, according to present knowledge.

One can postulate the presence of similar receptors within or about abdominal blood vessels. Sarnoff and Yamada have suggested that abdominal baroreceptor activity in cats is probably more important in circulatory regulation than the carotid and aortic mechanisms. The receptors, assumed to be the pacinian corpuscles, were stimulated by distention of mesenteric arteries leading to systemic hypotension, while occlusion proximally led to a pressor effect. Others have disagreed with this concept, Heymans’s studies in the dog having shown less of an effect from abdominal than carotid stimulation. Circulatory changes secondary to occlusion of abdominal vessels were attributed to changes in blood volume since complete denervation did not abolish the pressor effect.

The rapidity of the circulatory change resulting in a beat to beat decrease in stroke volume suggested to Peterson that the vagus, despite lack of anatomical evidence, might innervate the ventricular myocardium in man thereby affecting contractility. Many have disagreed with this concept, including Sarnoff et al., who demonstrated the dependence among atrial systole and the initial fiber length, end-diastolic pressure, and output of the left ventricle. Sympathectomy, or vagal stimulation in turn, by effecting a decrease in atrial contractility led to a decrease in the initial fiber length of ventricular muscle, lessened the initial end-diastolic pressure, and decreased ventricular stroke volume and work. The dissociation between the hypotensive effect and lack of a major change in pulse rate in several cases in the present report enhances the concept of sympathetic inhibition, although this assumption is highly speculative. A modifying factor in the degree of hypotension observed might have been a pharmacological effect of ether which depresses myocardial contractility and is usually counteracted by endogenous release of catecholamines. Thus, under conditions of increased sympathetic discharge, sudden withdrawal of sympathetic activity by a reflex mechanism might unmask the depressant effect of ether and produce more marked effects on myocardial contractility. There is some evidence to show that ether increases the sensitivity of baroreceptor responses.

Failure to demonstrate a significant alteration in the overall peripheral vascular resist-
ance enhances the concept of the central origin of the hypotension. Yet local vasodilatory changes might still have been present. It has been suggested that abdominal vascular receptors are important in local circulatory regulation within the abdomen. Such changes could give rise to venous dilatation and decrease the return of blood to the heart. The lesser, but still significant, decrease in diastolic blood pressure observed suggests that some degree of peripheral vasodilation may have been present. In view of the lack of uniformity of the circulatory changes, it is indeed possible that changes in peripheral resistance may have been more important than the data suggest.

A finding difficult to explain was the lack of change in central venous pressure or central blood volume. There was no correlation between these and the change in cardiac output and stroke volume. Were the changes in cardiac output due to venous dilatation and decrease in return of blood to the heart rather than to alteration in myocardial contractility as suggested above, one would expect to find a decreased central venous pressure and central blood volume. On the other hand, in the presence of decreased ventricular contractility, a rise in both values might be expected. Measurements of pulmonary wedge pressures or atrial and ventricular end-diastolic pressures could settle this issue. Insofar as central blood volume is concerned, the large standard deviations found suggest that there is considerable latitude for change in volume without detection by the methods employed. However, it is important to understand that a lack of alteration in superior vena caval pressure is little proof that venous return has not changed, especially when caval pressure is normal to begin with.

Summary

Of 38 patients studied during a steady state of ether anesthesia before performance of the definitive phase of operation, hypotensive episodes were produced in 8 by deliberate intra-abdominal stimulation of various types. The production of hypotension was therefore not only unpredictable in the group as a whole, but it was also possible that a positive response in each case could have been mediated by a different circulatory mechanism. The protocol of these experiments was not designed either by method employed or time interval chosen to detect rapid or early changes responsible for the final steady-state measurements that were made. Arterial systolic, diastolic, mean, and pulse pressures decreased with a decline in pulse rate in several cases. As a result of a decreased stroke volume or slowing of the pulse, the cardiac output declined and was responsible for the pressure alterations observed. There was no directional change in the calculated overall peripheral resistance. The remainder of the circulatory measurements, including central venous pressure and central blood volumes, suggest that the circulatory changes are by no means simply explained and that vasodilation in peripheral vascular beds cannot be ruled out.

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time and on the influences which affect it: IV. Output of the heart. J. Physiol. 22: 159, 1897.


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