Pressure Pulse in Small Arteries

By Takero Sugiura, M.D., and Edward D. Freis, M.D.

The present study was designed to answer the following questions: (1) What is the pressure gradient that occurs between the large and small arteries; (2) what changes occur in the dynamic characteristics of the pressure pulse during its transmission from large to small arteries; and (3) do small arteries play an important part in the control of the peripheral vascular resistance, or do they function primarily as transport vessels?

Methods

Hypodermic needles were constructed using 0.007- or 0.008-inch diameter stainless steel tubing. These were cemented into conventional needle hubs with epoxy resin. The needle shaft was made quite short (0.9 to 1.2 mm.), and the hub was attached directly to a model P23Db Statham strain gauge in order to reduce as much as possible the volume of fluid through which the pulse wave was transmitted (fig. 1). Freshly filtered, deaerated heparin saline solution, heated to 37 C, was used to fill the needle-gauge system. Great care was taken to exclude air bubbles, not only in the dome of the gauge, but also in the three-way stopcock and in the needle hub. Frequently, air bubbles trapped in stopcock or needle hub were not dislodged by repeated flushing, making it necessary to take all connections apart and to refill them separately before reattaching to the strain gauge. If proper filling, the frequency response of the needle-gauge system was found to be essentially flat and free of phase shift to at least 30 cycles per second (fig. 1).

Mongrel dogs, 13 to 15 Kg., in weight, were anesthetized either with 27 mg. per Kg. pentobarbital sodium or 100 mg. per Kg. chloralose intravenously, the latter being preceded by morphine, 2 mg. per Kg. The ability of the small needle-gauge system to record relatively undamped pressure pulses was checked prior to, and at frequent intervals during each experiment, as follows: One femoral artery of the anesthetized dog was cannulated with a 30-cm. length of PE 260 polyethylene tubing, which was then attached to a matching strain gauge and carefully flushed free of air bubbles. The opposite femoral artery was exposed and the small needle inserted into it. Pressure pulses were recorded simultaneously from both femoral arteries, using Sanborn model 350-110 carrier wave preamplifiers and oscillograph recorders. Systolic and diastolic pressures were measured in both tracings and were also inspected for similarity of contour. Damping was characterized by a lesser value for both systolic and diastolic (especially systolic) blood pressure. If a difference greater than 2 per cent systolic and 1 per cent diastolic pressure was found, the needle-gauge transducer was taken apart and refilled. This procedure was repeated until the two pressure recordings agreed within the limits specified.

The femoral catheter was then advanced to the midabdominal aorta, and the abdomen was opened via a midline incision. A loop of small intestine was withdrawn, and with the aid of magnifying glasses, the needle was threaded in a retrograde direction into arteries of various diameters in the mesentry and serosa of the intestine (fig. 1). After recording aortic and small artery pressure pulses simultaneously, the needle was withdrawn, the intestine was replaced, the pressure transducer and needle system carefully flushed, and another loop of intestine withdrawn for further measurements of small artery pressures. At the end of four or five such recordings, the small needle was reinserted into the femoral artery and the resulting pressure pulse inspected to be certain that repeated flushing had not altered the response characteristics of the needle-gauge system. The intestinal small arteries lay close to the surface and were readily punctured. Penetration of the mesenteric vessels was facilitated by gently removing the overlying fat by spreading with the points of a small scissors.

Norepinephrine diluted in 5 per cent dextrose in water was infused intravenously at a rate of approximately 0.001 mg. per minute, sufficient to maintain a significant elevation of arterial pressure. Angiotensin was similarly diluted to provide a concentration of 1 µg. per ml. Hexamethonium was injected intravenously in a dose of 2.2 mg.
of the ion per Kg. In the hemorrhage experiments, blood was withdrawn usually from a femoral vein or occasionally from a femoral artery. A total of approximately 250 ml. of blood was removed, usually in two approximately equal portions.

Results

Pressure pulses were obtained in small arteries down to and including those of 200 µ internal diameter. As can be seen in figure 2, the changes in the pressure pulse in the small arteries, as compared to the aortic pressure, were (1) a greater reduction in systolic than in diastolic pressure, (2) a more gradual systolic upstroke, and (3) a smoothing out of the dicrotic notch. In addition, high-frequency components, such as the small presystolic wave observed in some dogs, were either entirely or almost entirely obliterated. In figure 2, the curves taken from dog 179 show somewhat less damping during transmission of the pressure pulse to the small arteries than in dog 182, and serve to illustrate the range of response that was observed in normal, anesthetized dogs.

The systolic and diastolic pressure drops from the aorta to arteries of 1,000, 500, and 200 µ diameter are listed in table 1. The mean pressure fall from aorta to intestinal arteries of 200 µ diameter, representing the eighth to ninth bifurcation from the aorta, averaged approximately 17 per cent systolic and 12 per cent diastolic. The decreasing gradient in arterial pressure was continuous, a measurable decrease being present in arteries 1 mm. in diameter.

During the hypertension induced either by norepinephrine or angiotensin, the pressure gradient from aorta to arteries of 200 µ diameter decreased (table 2). The decreased pressure drop was more pronounced in regard to systolic than to diastolic pressure, with a consequent relatively greater increase in pulse pressure in the small arteries as compared with the aorta. In addition, the pulse-wave contour in the small arteries assumed more of the pattern of the aortic pressure with a steeper systolic upstroke and a sharper configuration to the dicrotic notch than before the drug (fig. 3).
Simultaneous recordings of pressure pulses from the aorta and an artery of 200 μ diameter before and after elevation of the arterial pressure with norepinephrine infused intravenously at a rate of 0.001 mg. per minute.

Reduction of arterial pressure following hexamethonium or hemorrhage increased the pressure gradient from the aorta to the small arteries, the effect on systolic pressure being somewhat more prominent after hemorrhage than after hexamethonium (table 2). When extreme hypotension (systolic pressures below 70 mm. Hg) was produced, either by hemorrhage or by the ganglion-blocking agent, the pressure pulse was almost entirely obliterated in the small arteries (fig. 4).

**Discussion**

Damping seemed to be the principal factor affecting the pressure pulse during its transmission from large to small arteries. Damp-

**TABLE 1**

<table>
<thead>
<tr>
<th>Arterial diameter (μ)</th>
<th>Number of bifurcations from aorta</th>
<th>Pressure change from abdominal aorta per cent decrease</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Systolic Mean S.D.</td>
</tr>
<tr>
<td>1,000</td>
<td>3</td>
<td>7.5 2.2</td>
</tr>
<tr>
<td>500</td>
<td>4 to 5</td>
<td>9.1 1.9</td>
</tr>
<tr>
<td>200</td>
<td>8 to 9</td>
<td>16.9 1.8</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>Diastolic Mean S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1,000</td>
<td>3</td>
<td>5.5 2.1</td>
</tr>
<tr>
<td>500</td>
<td>4 to 5</td>
<td>7.4 2.2</td>
</tr>
<tr>
<td>200</td>
<td>8 to 9</td>
<td>11.8 1.7</td>
</tr>
</tbody>
</table>

**TABLE 2**

Mean Values of the Per cent Fall in Blood Pressure from Aorta to Arteries of 200 μ Diameter Before and After Pressor or Depressor Agents

<table>
<thead>
<tr>
<th>Drug</th>
<th>Number of dogs</th>
<th>Systolic</th>
<th>Diastolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control Drug</td>
<td>Control</td>
<td>Drug</td>
<td>Control</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>8</td>
<td>16.0 10.4</td>
<td>11.3 8.9</td>
</tr>
<tr>
<td>Angiotensin</td>
<td>8</td>
<td>17.4 12.3</td>
<td>11.9 10.4</td>
</tr>
<tr>
<td>Hexamethonium</td>
<td>8</td>
<td>15.5 18.5</td>
<td>9.8 12.5</td>
</tr>
<tr>
<td>Hemorrhage</td>
<td>8</td>
<td>15.5 20.5</td>
<td>12.0 14.0</td>
</tr>
</tbody>
</table>
ing could explain all of the major changes seen, including the greater percentage drop in systolic than in diastolic pressure, the progressive loss of steepness of the systolic upstroke, the widening and smoothing of the dicrotic notch, and the disappearance of higher frequency components of the pulse wave.

Elevation of arterial pressure decreased the degree of damping, as evidenced by the decrease in the percentage pressure drop from aorta to small arteries, the steeper systolic upstroke, and the more abrupt deflections at the dicrotic notch in the small arterial pressure pulse. The decrease in damping observed during acute hypertension could result from increased stiffness of the arterial walls due either to direct action of the drug on arterial smooth muscle, increased tension on the arterial walls produced by the elevated lateral blood pressure, or both. Reduction of arterial pressure, either by hexamethonium or by hemorrhage, produced opposite effects, with evidence of increased damping of the pressure pulse in the small arteries. These hypotensive procedures could reduce the stiffness of the walls of the arteries by lowering the tension against the arterial wall as a result of the reduced lateral pressure.

The fact that the pressure drop from the aorta to arteries of 200 μ diameter decreased during the hypertension induced by either norepinephrine or angiotensin indicated that the increase in peripheral vascular resistance must have occurred in blood vessels distal to the point of measurement. Thus, there was no evidence to indicate that arteries of 200 μ diameter or larger play an important role in controlling the peripheral vascular resistance. The evidence indicates rather that the small arteries of 200 μ diameter or larger, aside from providing a moderate frictional resistance as indicated by the gradual pressure drop, function primarily as transport vessels.

Probably because of the technical difficulties involved, there have been few studies of small artery blood pressure. The classic investigations of Landis used methods that were unsuitable for recording the dynamic pressure fluctuations in such vessels. Haddy and his associates have threaded pipettes or, more frequently, plastic catheters into wedged positions in small arteries. The recordings obtained were valuable for the study of changes in mean small artery pressures in response to various physiological and pharmacological stimuli, but did not permit accurate study of the shape and dimensions of the pressure pulse contours in small arteries. Rappaport and Bloch and their associates developed a method for recording dynamic pressure changes in small arteries, using a small capacitance manometer. They recorded pressure pulses in small arteries of the systemic circulation of frogs and of the pulmonary circulation in kittens. To our knowledge, high fidelity recordings of pressure pulse contours have not been obtained previously in the small arteries of the mammalian systemic circulation.

**Summary**

Pressure pulse contours have been obtained in the small mesenteric and intestinal arteries of the dog. The principal change in the pressure pulse during transmission from large to small arteries appeared to be the gradual and progressive development of damping. The pressure drop from the aorta to arteries of 200 μ internal diameter averaged 17 per cent systolic and 12 per cent diastolic. Elevation of blood pressure with norepinephrine or angiotensin decreased the large-to-small artery pressure gradient, as well as the apparent degree of damping. Reduction of arterial pressure following hexamethonium or hemorrhage had the opposite effect. The evidence suggested that arteries of 200 μ diameter and larger function primarily as transport vessels and play no important role in altering the peripheral vascular resistance.

**Acknowledgment**

The fabrication of the small needles, the construction of the pulse-wave generator, and the testing of the dynamic response of the needle-gauge system were carried out by Merlin Davis and Winter D. Hampton of the National Bureau of Standards. We are also indebted to Mrs. Susan Babes for
valuable technical assistance during recordings of the pressure pulse contours.

References

This handbook is intended for students of physiotherapy. It includes a brief discussion of the causes and appearance of an ulcer. The major portion of the book is devoted to techniques of treatment, with special reference to medicaments for cleansing and dressing ulcers and preparation for skin grafting.

This book represents the proceedings of a symposium held in Queen's College, Dundee, in the fall of 1960. The conference began by a definition of the pathology of thrombosis. Several papers are devoted to the laboratory control of anticoagulant therapy. Several chapters cover the use of anticoagulants in relation to myocardial infarction, peripheral vascular disorders, and cerebrovascular disease. The relatively recent use of anticoagulants in extracorporeal circulations is also included.

This is the most complete monograph written on prothrombin and related factors of blood coagulation. It represents a summary of a quarter century of work by the author and his associates. The historical account of the discovery of prothrombin is interesting reading. The purification, activation, and inactivation of prothrombin are excellently discussed. The anticoagulants are completely covered, and a special chapter is devoted to the oral anticoagulants.

This monograph outlines, in a concise fashion, the modern diagnosis and treatment of acute arterial embolism, thrombosis, and pseudoembolism. It is directed toward the practicing clinician dealing with such entities which often require rapid diagnosis and therapy.
A short chapter is dedicated to the general pathophysiology of acute arterial occlusions. In the chapter on general diagnosis, the available examination procedures—history, physical findings, oscillometry, arteriography, etc.—are briefly discussed. The chapter on general therapy includes modern concepts of fibrinolysis. The larger part of this book describes the clinical forms of acute arterial occlusions in the extremities: arterial embolism, acute arterial thrombosis, acute spastic arterial occlusion (pseudoembolism). Each of these topics is didactically divided into etiology, pathophysiology, clinical manifestations, therapy, and prognosis. The thromboembolic processes occurring in infectious diseases, cardiac decompensation, hematological disorders, and infections and degenerative arterial diseases, as well as those resulting from trauma and freezing, are dealt with in this fashion. A noteworthy feature is the appendix showing the arterial collateral channels of the extremities. The bibliography is extensive and includes 631 references, mainly from the European and American literature.
This monograph offers a worthwhile addition to the library of students and clinicians, both internist and surgeon alike, who are interested in the field of angiology.
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