CONTROLLED HYPOTENSION*

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Hypotension has long been considered synonymous with shock, and hence its occurrence during surgical procedures is looked upon with fear or apprehension. However, blood pressure readings below normal need not be associated with shock. In recent years arterial hypotension has been utilized in attempts to reduce the loss of blood during operations. By inducing hypotension, it has been possible to perform certain operations more easily, more quickly, and with less risk, and to undertake others that would be impossible without this technique.

If it is true that a fall of blood pressure on one hand means shock, and on the other can be induced intentionally with little danger to the patient, it seems that there must be a fundamental difference between these two types of hypotension.

It will therefore be necessary to consider now some points concerning the factors that maintain blood pressure and to discuss the mechanisms that come into play when hypotension supervenes either because of shock or in consequence of deliberate action on the part of the physician.

PHYSIOLOGY OF BLOOD PRESSURE

Arterial blood pressure is maintained through a number of factors, the chief ones of which are: (1) cardiac output; (2) peripheral resistance; (3) amount of blood in the arteries; (4) blood viscosity; (5) elasticity of the arterial walls. Of these the first four factors contribute in regulating systolic blood pressure, while the last factor is chiefly instrumental in the maintenance of diastolic pressure. Any change in any one of these factors, sufficiently great or sustained to overcome the compensatory powers of the organism, will effect a rise or fall of blood pressure.

In circumstances that produce shock, such as for instance hemorrhage, the immediate reaction is a constriction of the arterioles, or more precisely of the metarteriolar sphincter mechanism. This represents an attempt at compensation which tends to maintain the blood pressure at its previous level. Trying to maintain the circulating volume at an adequate level, fluid

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is next shifted from the interstitial spaces into the blood stream, thus increasing the total plasma volume. When this takes place, one talks of "compensated" or better "incipient" shock. However, if hemorrhage continues without adequate replacement or if anoxic or stagnant hypoxia is superimposed, then arteriolar and capillary dilatation and stasis will develop. The consequence of this is a reduction of venous return and a fall of cardiac output with subsequent failure of compensation, hypotension, and the development of "frank" shock. If shock is not promptly corrected, the hypoxia increases until finally the integrity of the capillary wall is lost with such irreparable hypoxic damage as to make the shock "irreversible." The phenomenon of capillary stasis is demonstrated clinically by the increase of capillary refill time. The mechanism of hypotension as described pertains to cases of so-called "oligemic shock."

Another mechanism by which hypotension may be produced is one in which primary dilatation of the arteriolar bed occurs. Thus, by augmenting grossly the vascular bed and by decreasing peripheral resistance, a fall in arterial blood pressure ensues. In this kind of hypotension, capillary circulation should remain adequate, as can be demonstrated by very prompt refill time. Hence, venous return remains satisfactory and cardiac output is only moderately reduced. This is the mechanism acting in the rather badly termed "neurogenic shock," and is encountered clinically, for example, during spinal anesthesia.

It is, therefore, the adequacy or inadequacy of capillary circulation that determines whether or not the blood pressure fall is or is not a true shock state.

From the foregoing it is seen that hypotension of the first type associated with tissue hypoxia and leading to tissue changes cannot be tolerated for very long, and must be corrected by all the means at our disposal. Hypotension of the second type, on the other hand, has few deleterious effects, provided capillary circulation remains adequate and sufficient oxygenation of the blood is maintained. If these two prerequisites are followed, no damage will result to such vital organs as the brain, heart, kidneys and adrenals. This has been demonstrated both clinically and experimentally. Admittedly, filtration of urine probably ceases if the systolic pressure in the renal arteries falls below 75 mm. Hg (although this may be too high a value according to the latest investigative work). None the less, the nephron remains undamaged with pressures probably as low as 45-50 mm. Hg and may be even less, so that formation of urine is resumed when the filtration pressure is again reached. Let it be emphasized that this applies only with good capillary circulation and adequate oxygenation. With regard to the coronary arteries, even in individuals with coronary insufficiency, attacks of angina pectoris do not occur if arterial hypotension is induced by arteriolar dilatation, and if arteriosclerotic changes have not reduced the elasticity of the coronary vessels. If we consider the brain, we are more uncertain, as no one knows the minimum pressure necessary to maintain adequate nourishment of this sensi-