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A CLINICAL VIEW OF SHOCK*

BY

V. ZACHARY COPE, M.S., F.R.C.S.

Senior Surgeon, St. Mary's Hospital

Many words whose significance is well understood are difficult to define. This remark applies to the word "life" itself, and it is not surprising that shock, which threatens life and is the very shadow of death, should escape precise elucidation and definition. The difficulty is greater because similar symptoms may be produced by so many different agencies. The conditions induced by traumatic shock can be simulated by the effects of severe haemorrhage, by sudden loss of body fluid (as in cholera), by the introduction of foreign protein into the body, by severe burns, by high-voltage electric currents, by acute toxæmias, by some cases of visceral perforation, and even by severe mental stress.

Some critical observers have been so impressed by the absence of definite and easily measurable criteria of shock that they will accept no definition, and prefer to describe it by the enumeration of symptoms. Others have suggested different terms for the condition, such as "depressant syndrome," "traumatic hypodromia," or "exaemia," which give some indication of the pathological process involved. Shock is, however, a clinical state, and any definition must allow for the varied clinical picture which every clinical state may from time to time present. In any definition it is unwise to specify any particular symptom, for the first thing the clinician should learn is that there is hardly one symptom of shock which is constant; yet by a careful consideration of all the symptoms it can almost always be decided whether or not shock is present. It is generally agreed that shock is associated with impending or actual failure of the peripheral circulation, and we might perhaps adopt as a safe working definition that "shock is a condition following the application of harmful stimuli, or the rapid depletion of the body fluids, in which there is a serious and clinically demonstrable depression of the vital processes of the body, particularly of the circulation."

In connexion with trauma it is customary to speak of primary and secondary shock—the former coming on immediately and the latter at an interval after the injury. Perhaps the terms "immediate" and "delayed" would be clinically preferable to indicate the immediate and delayed onset of shock. Immediate shock may last for only a short time unless the causative factors continue to act.

Clinical Symptoms

What are the main clinical symptoms of the shocked condition? A *persistent reduced blood pressure* is regarded by most investigators and clinicians as an essential feature of shock. They say, "No fall of blood pressure, no shock." Yet 30 years ago Parsons and Tyrrell Gray showed that clinical symptoms of shock might appear with a blood pressure that was still high; and in the last war Bayliss showed that a high blood pressure might persist with a small blood volume owing to powerful vasoconstriction. Recently Grant has called attention to the same phenomenon, and Meakins has emphasized the same point in a clear manner. The latter states: "After the introduction of the sphygmomanometer our thoughts were slowly, ever so slowly, directed to the belief that a falling

blood pressure and shock were synonymous. This is to some degree correct, except that the real condition of shock has been initiated and has travelled well along its course before this spectacular and exact physiological disturbance occurs. It should be considered as one of the later findings to indicate the condition. It occurs after all compensatory mechanisms to maintain the circulation have failed."

Before we can tell whether the blood pressure has fallen we must know what it was before the injury, and there should be no need to be reminded that the average blood pressure for a given age may not be the normal for the patient. There are cases, also, in which the failing circulation is shown more by the rise in diastolic than by the fall in systolic pressure. A low pulse pressure is probably a better indication of shock than a lowered systolic pressure. Even while the blood pressure remains high and before any serious symptoms have appeared shock may be present in a latent form. This *latent shock* corresponds to the time at which the compensatory vasoconstriction barely maintains the intravascular pressure; collapse may be precipitated by any further strain on the circulation—even turning the patient over may cause serious collapse. This clinical condition of latent shock must have been noted by most experienced surgeons, but there is still need to emphasize its occurrence and its deceptive nature.

In well-developed shock it will be agreed that its severity can best be measured by the amount of fall in blood pressure. If the systolic falls below 80 the condition is serious, and a pulse pressure of under 25 is of bad prognosis.

A *small, weak, and rapid pulse* is regarded as a necessary accompaniment of shock, yet it is now well recognized that serious shock may be present without any increase in the pulse rate. Many years ago Malcolm pointed out that the pulse rate might remain slow in shock but get gradually smaller until it failed altogether. Cuthbert Wallace in the last war noted a class of shocked men whose pulse remained slow, and the same phenomenon has been observed in the shocked air-raid casualties of this war by Kekwick and others. From the operating surgeon's point of view I have confirmed that shock and a lowered blood pressure may coexist with a slow pulse. The testimony of so many observers must convince everyone of the not uncommon occurrence of shock accompanied by a slow pulse.

A *subnormal temperature* is a common symptom of shock; indeed, I have come to regard this as one of the most constant features of the condition. Kinnaman's experiments on animals showed that a fall of temperature was a better guide to shock than a fall of blood pressure. Observation of air-raid casualties led Kekwick and his fellow workers to say that "the general temperature of all severe cases is invariably subnormal." The drop in temperature may be down to 95° or lower. This fall is not dependent upon the fall in blood pressure, for the metabolic processes of the body are not seriously affected until the blood pressure has fallen below 80 mm. Hg, whereas it is common to get a temperature of 95° F. in cases of immediate shock in which the blood pressure may be lowered but little or not at all. It may be that there is a definite inhibition of metabolic processes in shock. The immediate shock which may

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follow the perforation of a peptic ulcer frequently results in a temperature of 95° F. with pallor and cold extremities even though the blood pressure may be about normal or only lowered 10 or 15 mm. Hg. I think that those who deny that shock is often present in cases of perforated peptic ulcer take too narrow a view of the condition.

In cases of shock *the skin is usually cold and pale or cyanotic, and sweating is often profuse*. It is frequently the onset of these two symptoms which indicates to the surgeon that shock is present. If the cheek feels cold to the back of the observer's hand and if small beads of moisture appear on the forehead one knows that the patient on the operating table has had enough. Latent shock is becoming patent.

Mental dullness is a variable symptom in shock. It is quite true that many shocked patients are dull or stuporous, but others are mentally alert. It was said of the wounded at El Alamein that the mental state was usually one of alert interest even when the blood pressure was very low. It is difficult to account for these differences. It is important that we should diagnose shock before all the textbook symptoms have appeared. Shock may be indicated by a subnormal temperature with a low blood pressure although the pulse rate may be within the normal limits; it may reveal itself by pallor, sweating, subnormal temperature, and a small rapid pulse, while the blood pressure may remain approximately normal; and some cases occur in which the appearance and mental alertness give no indication of the serious state of the circulation which the sphygmomanometer may demonstrate. When a person has sustained injuries which are such as to be productive of shock one must assume its presence in a latent form even though no obvious symptoms are observed. The clinician must look at the whole picture before he pronounces judgment.

Pathology of Shock

Can the clinician throw any light on the vexed question of the pathology of shock? He is at a disadvantage with the experimental worker in that he cannot standardize his cases or furnish accurate controls; but clinical cases deal with human beings and usually present the important factor of pain in the causation of shock. Though Moon asserts that "conditions accompanied by prolonged and intolerable pain such as renal or biliary colic do not cause shock," most clinicians would consider this at the least too dogmatic a statement. Indeed, few clinicians could doubt that severe pain can initiate, accelerate, or aggravate shock.

Of the many theories put forward there are some which have little clinical support. Hyperventilation and acapnia could not apply to the majority of cases; acidosis is more likely to be the result than the cause of shock, and could not account for the immediate variety; the same remark could be made about the increase of blood potassium, which some, especially Scudder, regard as an important causative factor of shock. Fat embolism may produce symptoms almost indistinguishable from those of shock, but clinically it cannot be supported as a common cause of the condition. The suprarenal gland seems to play a part in causation of the symptoms, but as a rule those symptoms do not become evident until serious circulatory changes have already occurred.

All experimentalists agree that in cases of shock there is a serious diminution in the volume of blood in effective circulation, or, as Moon puts it, the volume of blood is relatively inadequate to fill the vessels. This fact is daily verified by the clinician, who constantly sees that a rapidly diminishing blood volume caused by severe haemorrhage will produce symptoms of shock, so that he is often in doubt whether he is dealing with haemorrhage or shock, or a mixture of both. Now, the volume of blood in effective circulation may be diminished in one or more of three ways: first, by gross internal or external haemorrhage; secondly, by escape of plasma or the fluid part of the blood directly into the tissues (as in trauma) or on to the surface (as in burns), or indirectly by the loss of fluid externally by the secretions (sweat) or excretions (cholera and dysentery); and, thirdly, by loss of blood to the effective circulation owing to its stagnation in dilated capillaries. This capillary dilatation might be induced by reflex nervous influence, by local trauma, or by the action of toxins on the capillary walls. The second and third methods would account for shock when unaccom-

panied by haemorrhage. It is indeed impossible to account for the different clinical types of shock without invoking both vasodilatation and transudation of fluid. Immediate shock must be the result of an immediate cause, and it is difficult to avoid the view that this is due to a nervous reflex producing vasodilatation in some part of the peripheral circulation—perhaps in the flaccid muscles, as suggested by Starling. How quickly the reflex may act is well seen in the speed with which the cheek may be mantled by a blush. The importance of this nervous reflex has been minimized by many, yet there is little doubt that strong nervous afferent stimuli will of themselves cause shock. Crile's view that this was due to exhaustion of the vasomotor centre is no longer tenable, but we must assume some inhibition or incoordination of this centre to account for the onset of symptoms of immediate shock.

Can the clinician help to decide the vexed question as to the way in which the volume of blood in effective circulation is diminished in secondary or delayed shock? Is it due to the loss of fluid into the tissues of the damaged part, as asserted by Blalock; does it depend partly upon a nervous reflex induced by the barrage of nervous impulses ascending from the injured part, as claimed by O'Shaughnessy and Slome; or may it be due to absorption from the injured part of substances which may injure the epithelium of the capillaries and permit serious transudation of fluid from the peripheral circulation? The clinician must surely answer that the whole truth does not lie with any one of these views. There is no doubt that much blood and much plasma are lost into the tissues of a traumatized area, but severe shock may occur in cases in which it would seem impossible for enough fluid to have escaped to cause serious symptoms; and there is no reason why constant and powerful afferent stimuli from the injured part should not reflexly contribute to shock as they appear to do in cases of immediate shock.

The view that toxins from the breaking down of tissues in the injured part might produce shock was widely held during the last war but gradually lost favour and received little experimental support. Recently, however, H. N. Green obtained a shock-producing substance from fresh voluntary muscle; he found that it was rapidly destroyed in the muscle after death and therefore might easily be missed. Given intravenously, this substance had a powerfully depressant action on the blood pressure. Clinically one can judge little as to the likelihood of shock-producing substances in muscle, but no one who has seen the occasional rapid development of shock following the relief of obstruction of a coil of small bowel will be likely to doubt that toxins absorbed from the small gut may occasionally have a shock-producing effect. How such a toxin may produce shock is a subject for speculation.

It is a matter of common clinical experience that some cases of shock fail to respond to treatment or respond only for a time. The pathological process goes so far that it cannot be reversed. Mere haemorrhage can be rapidly made good by transfusion, but with some cases of severe shock no amount of blood or other fluid infused into the vessels will maintain pressure: either the fluid constantly transudes from the vessels, or the vasomotor centre cannot maintain the pressure in the vessels.

Conclusion

Our clinical scrutiny of the pathology and symptoms of shock leads us to conclude, therefore, that there are several mechanisms which may produce the same result—failure of the peripheral circulation—and this failure may be indicated by several differing clinical pictures.

A fifth edition (1944) of the Orthoptists' Section of the *National Register of Medical Auxiliary Services* has now been published by the Board of Registration of Medical Auxiliaries, B.M.A. House, Tavistock Square, London, W.C.1. Orthoptists whose names are included have qualified according to a standard approved by the Board. The Board also maintains registers of physiotherapists, radiographers, dispensing opticians, chiropodists, and speech therapists; and a register of dietitians is being prepared. The Board is anxious that the fullest possible use shall be made of all sections of the *Register*, and copies of any or all sections will be supplied free to medical practitioners, hospitals, local authorities, etc., on application to the acting secretary.