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Traumatic shock

Robert E. Barton
University of Nebraska Medical Center

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TRAUMATIC SHOCK

by

Robert E. Barton

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PREFACE

Because of its mysterious onset and nature, traumatic shock has long suggested problems of unusual clinical and scientific interest. Its frequent occurrence especially in association with severe wounds and extensive surgical operations renders a solution of these problems particularly important. Notwithstanding the large amount of attention which it has received and the resulting voluminous literature devoted to this subject, the conception of the ultimate nature of the phenomenon of shock at the present time stands not in universal agreement.

It would be of little practical value to examine in detail all of the literature available on the subject, for many of the older ideas relevant to the nature of traumatic shock have been definitely disproven. Furthermore, much of the literature shows extensive recapitulation. However, reference will be made to some of the more notable attempts to solve the problem by certain prominent investigators. As a result of observations made during the World War by members of the Shock Commission, a number of facts stand out to disprove

many old theories and provide a basis for the development of new ones. Outstanding among these researchers was Cannon, professor of physiology at Harvard, whose brilliant work at Bethune perhaps contributed more toward the modern conception of traumatic shock than that of any other investigator.

It is the purpose of this thesis to set forth, first of all, the clinical features of shock; secondly, associated basic factors; thirdly, theoretical ultimate factors; fourthly, the resultant of the numerous clinical and experimental observations, i.e., the modern conception of traumatic shock; and finally fifthly, rational therapeutics as pertains to this condition.

I - CLINICAL PICTURE

For many years, surgeons have noted a startling clinical picture which appears in patients following severe injuries. Many names have been given to this syndrome, adding further to the confusion of its understanding, but notwithstanding many objections the term shock has been almost universally adopted and was first used by Latta in 1795 to designate this condition.

Shock appears typically in patients after extensive surgical operations and in victims of burns, war wounds, and industrial and transportation accidents. The common occurrence of this complication in the World War led to the formation of a commission of surgeons and physiologists to study its nature. This effort which is described in Cannon's monograph on "Traumatic Shock" was productive of experimental and clinical evidence that went far toward the elucidation of the problem.

Clinical accounts of shock through the years have varied somewhat since earlier observers applied the term loosely to diverse conditions associated with sudden weakness, fainting, unconsciousness, and sudden death. On the other hand, there were some very accurate early descriptions of shock that are in accord with our

present-day clinical recognition of this condition.

The following is an abbreviation of an account given by Fischer (6) in 1870. The patient, a strong and perfectly healthy young man, was struck in the abdomen by a pole of a carriage drawn by runaway horses. After careful examination, there was nothing found to indicate an injury to any of the internal organs. Nevertheless, the grave symptoms and the alarming look which he still presents made their appearance immediately after the accident. He lies perfectly quiet and pays no attention whatever to events about him. The pupils are dilated and react slowly to light. He stares purposelessly and apathetically straight before him. His skin and such parts of the mucous membranes as are visible are as pale as marble, and his hands and lips have a bluish tinge. Large drops of sweat hang on his forehead and eyebrows, his whole body feels cold to the hand, and a thermometer indicates a degree and a half Centigrade in the axilla and a degree in the rectum below normal. Sensibility is much blunted over the whole body, and only when a very painful impression is made on the patient does he fretfully make a wry face and then a languid defensive movement. If the limbs are lifted and then let go, they immediately fall as if dead.

The urine is scanty and dense but free from any traces of sugar and albumen. The pulse is almost imperceptible and very rapid. The patient is conscious, but replies slowly and only when repeatedly and importunately questioned. On being thus questioned, he complains of cold, faintness, and deadness of the extremities. His voice is hoarse. His respiration is characterized by long, deep, sighing inspirations alternating with very superficial ones which are scarcely visible or audible. While being brought to the hospital, he vomited several times, and nausea and hiccoughs still remain.

A typical case of wound shock as described by Cannon (6) may be briefly summarized. A man belonging to the garrison was wounded by a bomb which partly shattered the forepart of his foot and sprinkled his neck and shoulder with tiny fragments. His chum, standing by his side, was killed. The blood pressure, which was 110/70 mm Hg a short while previously, was still the same immediately after he was wounded. It was a cold night with a chilly wind and as the man was carried along shallow trenches winding over a hill, he became colder and colder. At this time, too, there was occasional enemy activity. By the time he reached the aid post, an hour and a half later, he was pulseless. Two hours later, when examined at the next post, he was still pulseless,

and a serious view was taken of his condition. He was hurried on to the casualty clearing station where he arrived with a blood pressure of 80/65 mm Hg and no palpable pulse. After being warmed up in bed, the pulse soon returned and the shock passed off in the absence of any heroic measures of treatment.

When this characteristic syndrome occurs shortly after trauma or during the course of an operation, it is designated as primary shock; when the symptoms are delayed until several hours after an injury or operation, it is designated as secondary shock. The primary type is relatively transient and resembles the nervous phenomena of fainting or is associated with so extensive destruction of tissue as to make continuance of life impossible. Since such cases either recover without treatment, at least temporarily, or are hopeless, primary shock does not constitute as important a clinical problem as does secondary shock. Consequently, to this latter type, laboratory and field investigators have chiefly directed their researches. It is important to mention here that Blalock (3), a recent investigator, emphasizes the fact that fundamental physiologic disturbances associated with primary and secondary shock may

co-exist.

In order to clarify the course of events in primary and secondary shock, Cowell (7) has represented graphically the blood pressure changes that may be seen in cases of one or other of the two types. Refer to fig. 1, p.8 and to fig. 2, p.9.

PRIMARY WOUND SHOCK - ONSET AND COURSE

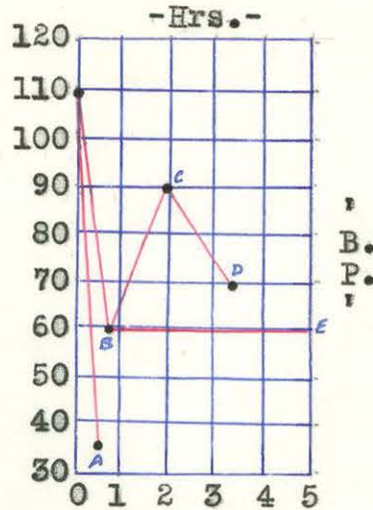


Fig. 1.

Following the receipt of a severe injury, such that death must ensue or life can be saved only by prompt surgical intervention, instant shock appears. This may be fatal in a short time (A). If all precautions are taken in the careful transit of the patient, the blood pressure may rise enroute (C) or remain level without further drop (BE). After lapse of a few hours, the condition of primary wound shock may merge into that of secondary wound shock (D or E).

SECONDARY WOUND SHOCK - ONSET AND COURSE

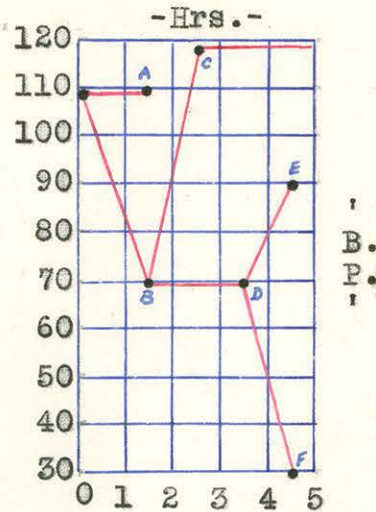


Fig. 2.

In many wound cases, the blood pressure will remain level (A). In others, as a result of hemorrhage or exposure to cold, there will be a drop of blood pressure with the establishment of secondary shock (B). If the patient is at this stage well cared for and the wound not too severe, the pressure may rise during the next stage of the journey (C) or remain stationary (D) and improve after admission to the casualty clearing station (E). In the absence of favorable circumstances, the pressure goes steadily down and the case terminates fatally, usually in from 12 - 24 hours (F).

The salient clinical features of secondary shock, as shown in Cannon's monograph, in Blalock's writings, and in others, may be summarized:

1. Appearance: pale, perspiring, apprehensive.
2. Subnormal temperature, cold extremities.
3. Rapid thready pulse, heart sounds often imperceptible.
4. Rapid respiration - shallow or deepened.
5. Declining blood pressure.
6. Reduction of blood volume with hemoconcentration
(increased red and white counts and elevated hemoglobin, increased viscosity and specific gravity and elevated blood nitrogen).
7. Other - reduced oxygen consumption and lowered basal metabolism; reduced alkali reserve; concentrated urine.
8. If progressive - apathy with blunted sensibility, pupillary dilatation with sluggish reaction to light, general decline of reflex excitability, prostration, coma, bulbar collapse, and death.

It is upon these symptoms and findings, when accounted for neither by hemorrhage or late infection with toxemia, the diagnosis is made.

Clinical differentiation between shock, hemorrhage, or the two combined is often difficult. The following points are at times helpful however:

1. If the pulse rate increases far out of proportion to the blood pressure fall, this is more characteristic of hemorrhage.
2. Blood volume is reduced both in hemorrhage and in secondary shock; but, within 24 hours, in the case of the former, hemodilution occurs, and, in the latter, hemoconcentration.
3. Hemorrhage, of course, when visible is confirmative of the same; when concealed, symptoms and findings characteristic of hemorrhage in certain regions of the body are suggestive.

II - MECHANISM: BASIC FACTORS

THE LOW BLOOD PRESSURE

There is no more important aspect of the complex of established shock than the state of the circulation as indicated by the sphygmomanometer; reduction of blood pressure is probably the most characteristic and essential feature. Furthermore, the degree of reduction of pressure may be regarded as a fairly satisfactory index of the degree of shock which is prevailing.

In a series of 93 cases of shock and hemorrhage, about half of them examined at Bethune by Cannon (6) and others reported to the English Shock Committee by Bazett (6) and by Keith (6), the distribution of the average systolic pressures, diastolic pressures, and pulse pressures was as follows:

14 cases	- Av. sys. p.	95	- dias. p.	63	- pulse p.	32
16		87		57		30
26		76		49		27
17		66		41		25
14		58		36		22
6		47		30		17

Brechet and Claret (6) have reported that the pulse pressure is of prognostic value. If it remains above 25mm., according to them, the prognosis is favorable; at 25mm., it is questionable; and below

25mm., the outlook is poor.

Although plans had been made for securing observations on venous pressure in shock cases, the ending of the War before these plans could be realized rendered them futile. However, Cannon (6) made the observation on numerous occasions that when drawing blood for tests in shock victims, in most instances, the veins were collapsed or contracted. Experimental evidence pointing to reduced venous pressure in shock has been offered by Morison and Hooker (6) who produced shock experimentally by intestinal injury and noted a progressive fall of pressure in the vena cava; by Wiggers (6) who noted fall of venous pressure repeatedly in experimental shock; and by Erlanger, Gesell and Gasser (6) who induced shock by intestinal manipulation and noted a fall of jugular pressure.

BASIC FACTORS WHICH MIGHT AFFECT BLOOD PRESSURE

The most important central problem of shock is that of discovering the reason for the lowered arterial pressure. The various theories which have been suggested to account for shock are all directed toward the solution of this problem in particular.

Normal arterial pressure may be reduced if the heart becomes inefficient, that is, if it contracts ineffectively or feebly. There is,

consequently, a possible cardiac factor.

Arterial pressure may be low because of diminution of peripheral resistance. Normally, the arterioles are held in a state of tonic contraction by impulses delivered from the vasomotor center. If the vasomotor center becomes impaired, there will be such a relaxation of the smooth muscle in the walls of the arterioles as to permit the blood to pass readily from the arterial into the capillary and venous areas with an attendant drop in the pressure level. A second possible factor, therefore, is a diminution or loss of vasomotor tone.

A third factor which would result in lowered pressure is an insufficient blood volume. Up to a certain limit, the blood vessels are capable of compensating by contraction for a loss of blood volume. Beyond this limit, any further reduction in blood volume results in diminished pressure.

Another way in which the relation between the volume of blood and the capacity of the vessels may become disturbed is through enlargement of the capacity of the vascular system, particularly of one portion such as the veins, The blood may then accumulate there and consequently be returned in so slight

a stream as not to permit the heart to develop a normal pressure within the arteries. The disturbance of the normal relation of the volume of circulating fluid to the capacity of the circulatory apparatus presents, therefore, another mode by which the circulation may become defective or fail.

THE QUESTION OF A PRIMARY CARDIAC FACTOR

It has been observed repeatedly that the heart characteristically beats faster than normal in shock or after hemorrhage. Hence, the heart might be looked to as a possible primary factor in shock.

Exhaustion of the cardioinhibitory center as a primary role:

In the course of experimental work in which operations on the brain were performed, Howell (6) noted that there was produced a marked increase in the pulse rate. Some cases showing an extraordinary increase in rate exhibited only slight lowering of arterial pressure, leading Howell to think of shock in two forms, i.e., cardiac and vascular. He assumed that in cardiac shock the most important factor is a partial or total suspension of activity of the cardioinhibitory center. The condition manifested

itself in his experiments as a sudden or progressive increase of rate and diminution of amplitude of the heart beat. What Howell calls vascular shock, i.e., a more or less complete loss of arterial tone, he never observed independent of cardiac shock; the latter always accompanied or preceded the fall of pressure.

The suggestions offered by Howell have been tested by both Crile and Mann. Crile (6) reported that he had noted reflex inhibition of the heart in animals which were in a serious degree of induced traumatic shock. In addition, he noted that shock is as readily produced in normal animals as in those in which the heart is isolated from the central nervous system. Mann (6) found that shock did not develop any more quickly under experimental conditions when the vagi had been cut than when these nerves were intact. He also noted that reflex inhibition of the heart by stimulation of the central end of a divided vagus trunk could be obtained even in the most extreme degrees of shock. He was able to induce vagal slowing of the heart repeatedly in shocked animals by increasing intracranial pressure, such slowing disappearing upon section of the vagi. The experiments of Crile and Mann, therefore, seem

to prove conclusively that in traumatic shock, the cardiac inhibitory mechanism is not exhausted and that it may be called into action by usual means.

Tonically contracted heart as a primary role:

Boise (6) has reported experiments in which he noted diminution of the extent of cardiac contractions associated with a lowered arterial pressure in shocked animals. He interpreted this as being due to tonic contraction of the cardiac muscle. Boise regarded this increased cardiac tonus as a primary factor.

Henderson (6), on the other hand, who observed the same phenomenon in similar experiments interpreted this tonic contraction as secondary to vascular or volume changes and the mechanism to be as follows:

Primary vascular or volume changes
 ↓
 Reduced volume of venous return to right heart
 ↓
 Reduced cardiac volume with retraction of the heart
 muscle (or increased cardiac tonus)
 ↓
 Reduced cardiac output
 ↓
 Decline of arterial pressure (after limitation of
 vasomotor compensation has been reached).

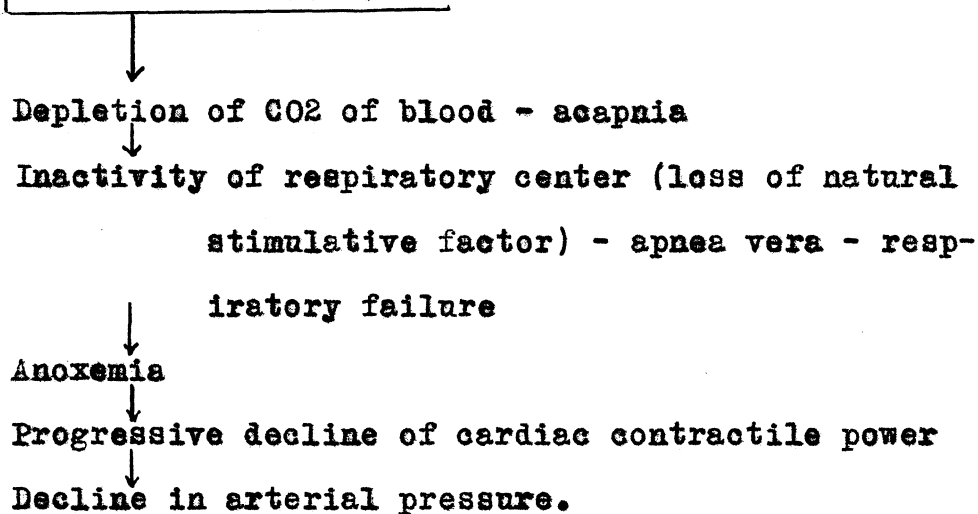
Increased cardiac tonus, therefore, can be accounted for as a result of diminution of the volume of venous return to the right heart. There is no evidence to show this factor to be primary. Further evidence that increased cardiac tonus, such as would lead to diminished cardiac output, is not the primary cause of shock is found in the state of venous pressure. Venous pressure would rise if the heart were suffering from a diminished capacity to transfer blood from the venous to the arterial side. But this is not the case; moreover, clinical and experimental evidence has indicated that venous pressure often falls as the arterial pressure falls during the development of shock. There is no evidence, therefore, that a tonic contraction of the heart plays any primary role in shock.

Impaired contractile power as a primary role:

Impairment of the inherent contractility of cardiac muscle results in an insufficient transfer of blood from the venous to the arterial side of the circulation, such impairment frequently being the cause of death in cardiac disease. Henderson (6) has argued that cardiac failure may occur in consequence

of excessive respiration. His experimental findings in animals with regard to this together with his interpretation may be outlined as follows:

- #1. Vigorous artificial respiration for 30 min.
- #2. Continued stimulation of sciatic nerve resulting in hyperpnea.



Wiggers (6) has produced by sciatic stimulation greatly increased depth and rate of respiration, but in no instance, even after two hours of stimulation, did permanent apnea or death from respiratory failure occur. Cannon (6) states that whether Henderson or Wiggers is correct with regard to the effects to be expected from excessive respiration, acute weakening of cardiac contractions as a result of oxygen lack do not duplicate the events which take place in the course of shock. Pain might evoke rapid and deep respiration but clinical experience

with shock proves that it is not attended by conditions which Henderson showed experimentally. Commonly, respiration ceases before the final heart beats, but cessation of respiration is not preceded by intense hyperpnea due to painful stimulation. Cannon concludes that it cannot be considered correct that failure of cardiac contractility as a result of apnea due to excessive breathing is the cause of death.

Condition of the heart in shock:

Mann (6) has reported after extensive study of shocked animals that, when the animals are allowed to die from shock itself, the heart continues to beat, though often feebly, after respiration has ceased and the blood pressure is practically zero. Cannon (6) has frequently observed the same sequence of events at death in cases of shock in human beings. In a series of experiments, Crile (6) observed that after animals had been reduced to a presumably fatal degree of shock and the blood pressure was then raised much higher than normal, the heart quickly recovered its capacity to do its normal work, this probably occurring on the basis of improved coronary flow with subsequent restoration the contractile stimulus, i.e., intracardiac

tension.

There has been no convincing evidence that either the heart muscle or the nervous mechanism controlling it has a primary role in lowering arterial pressure in shock.

Cardiac tamponage as a primary role:

Blalock (3), a recent investigator, has pointed out that traumatic hemopericardium resulting in cardiac tamponage with progressive myocardial weakness may induce the shock syndrome. There is a venous distension in contrast to the collapsed veins seen in peripheral circulatory failure.

THE QUESTION OF A PRIMARY VASCULAR FACTOR

Vasomotor exhaustion as a primary role:

The best known support for the view that shock is due to vasomotor exhaustion has come from the experiments of Crile (6). He stated that this might be due either to exhaustion of the smooth muscle in the vessel walls or to exhaustion of the vasomotor center; but, since adrenalin caused as great a rise in blood pressure in shock as it did in normal conditions, and since adrenalin produces its effect by stimulating the arteriolar smooth

muscle, Crile concluded that fatigue of the blood vessels themselves must be excluded and that shock is due to an exhaustion of the vasomotor center.

Porter (6) disproved Crile's work. He found that, even when an animal is in extreme shock, both pressor and depressor reflexes still occur.

Cannon (6) states that vasomotor exhaustion cannot be the basic cause of the low blood pressure in shock but that it may occur as a result of persistent lowered blood pressure which brings about a deficient blood supply to the bulbar centers.

Studies by Pike, Guthrie and Stewart (6) have revealed that the vasomotor center is more capable of withstanding the adverse influence of anemia than any of the other vital bulbar centers, including the respiratory, cardioinhibitory, etc., this indicating the vasomotor center to be an agent whose functions are extremely stable.

The condition of the vasomotor center in shock:

Experimental studies of shock by Erlanger, Gessel and Gasser (6) have brought out the distinction between the state of the vasomotor center in early and in late stages of shock. They determined directly the variations in peripheral resistance as shock

progressed. They concluded that in early stages of shock, vasomotor tone is most often augmented, probably as a compensatory phenomenon; later, as the arterial pressure falls and goes beyond a critical level, the peripheral resistance falls. Up to the time of death, however, the vessels preserve some tone and the vasomotor center retains slight capacity to react.

Clinical evidence for maintained vasomotor tone in human cases has been offered by Wallace, Fraser and Drummond (6), World War surgeons who performed hundreds of abdominal operations on patients in all degrees of wound shock without observing a single case of primary splanchnic congestion. Malcome (6) states that the more profound the degree of shock the paler the tissues become, and that the pallor is noted even when very little blood is lost. Further, Cannon (6) observed in shock cases that when the pulse could not be felt at the wrist, it could be felt easily over larger vessels as the carotid. Fraser (6) repeatedly noted, while operating on shocked men, such strong contraction of outlying arteries that no bleeding occurred when the vessels were cut. Ducastaing (6) has reported that in two cases of shock when the pulse was weak or wholly

imperceptible, the wave became quite readily palpable after the administration of amyl nitrite, a vasodilator. This, Ducastaing argued, is further evidence that in shock peripheral vessels are constricted.

The vasomotor factor as pertains to primary shock:

Evidence thusfar cited in part II has been with reference to secondary or delayed shock in particular. The majority of investigators have agreed that the vasomotor factor probably does not have a primary role in secondary shock. Whether or not this factor plays a primary part in primary shock is still a question, but modern opinion is in favor of this view.

Primary shock, it will be remembered, is a state of collapse in which characteristic symptoms and the low blood pressure appear immediately on receipt of some injury. Moon (15) records that an early investigator, Goltz, observed that repeated stroking of the frog's abdomen caused a marked vasodilatation, notably in the splanchnic area. Blalock (3) in his series of experiments has shown, that in cases of primary shock, peripheral vasodilatation, probably resulting from influences acting through the nervous system, occurs.

Cannon (6), in considering syncope and collapse in relation to shock, pointed out the following facts and his ideas regarding same. Syncope and collapse is the result of cerebral anemia which is usually associated with a depressed vascular tone throughout the body. Such an event is probably on a reflex nervous basis and may result from some psychic factor as, for example, sudden bad news; this, Cannon states, may be labeled as emotional shock. Again, this condition may result from violent stimulus applied to certain regions of the body as, for example, the epigastric region or the testicle, from rupture of an abdominal viscus, etc. It is characteristic of syncope that the individual on lying down or having the feet raised or the abdomen compressed regains consciousness and does not suffer subsequently. The symptoms of shock and collapse are practically identical. Collapse has been described as a sudden great fall in blood pressure such as may be seen after severe hemorrhage or after vasomotor paralysis arising from violent afferent impulses. It seems possible that primary shock and collapse may be regarded as practically indistinguishable, Cannon concludes.

Phemister (17) has shown that primary

shock may occur from psychogenic stimuli, a simple form being that seen in a person who faints at the sight of a severe accident. The blood pressure falls, the pulse rate slows, pallor develops and unconsciousness supervenes; the patient falls to a recumbent position which restores cerebral circulation, and consciousness returns. Phemister has also shown that primary shock occurs not infrequently during operations when the peritoneum is opened, also during operations on the stomach and biliary tract, probably as a result of stimulation of the autonomic fibers of the vagus nerve. If the operative manipulations are not too severe or prolonged, the blood pressure will return to its previous level; or the condition may continue and eventually pass into secondary shock.

(3)
Blalock refers to the mechanism of primary shock as neurogenic, the modus operandi being a reflex vasomotor collapse particularly in the splanchnic area. This splanchnic vasodilatation is probably the most satisfactory explanation of primary shock.

THE QUESTION OF A PRIMARY BLOOD VOLUME FACTOR

Attention is now turned again to secondary shock, and the question of a primary blood volume factor will be considered.

The evidence thus far brought forth shows that at first, in most cases of shock, the vasomotor center retains its efficiency and the heart is capable of assuming any reasonable burden which may be placed upon it. The only other possible factor which might lead to a persistently low blood pressure is the reduction of blood volume.

The essential condition, when the circulation fails through an inadequate return of venous blood to the heart, is a discrepancy between the volume of circulating fluid and the capacity of the circulatory system. There may be, on one hand, too great a capacity; but since, in most cases of shock, this is probably not primarily the result of relaxation of the arteries and arterioles, some investigators have assumed that the difficulty lies in the veins - that there is some derangement of some venopressor mechanism. On the other hand, the discrepancy may arise from a reduced blood volume. There are advocates for each of these views.

Venous atony as a primary role:

There is little or no available evidence indicating venous atony to be a basic factor in the production of shock. However, Cannon (6) believes

that it is probable that venous atony plays a secondary role after the onset of shock thusly:

Prolonged low blood pressure
 ↓
 Exhaustion of vasomotor center - or centers
 ↓
 Arteriolar atony - venous atony.

Reduced blood volume as a primary role:

The question of reduction of blood volume was considered by Keith (6) in his study of a series of 29 human cases during the World War. The method employed consisted essentially of introducing into the bloodstream a known amount of vital red, a non-toxic non-diffusible dye, and later comparing the color of the plasma with a known standard. In the series of cases Keith studied, each patient was weighed before the test was made, and values for total blood volumes and for plasma volumes were compared with those of normal men of the same weight. Total blood volumes were found to be constantly reduced in soldiers suffering from wound shock. Further, the diminished blood volume was recognized as bearing a definite relationship to the severity of the patient's clinical state. In 27 of the 29 cases of wound shock, the estimated blood volumes ranged from 52 - 85% of the normal, and there was a corresponding reduction of plasma volumes which

ranged from 62 - 90%. In the remaining two cases, direct determinations were not made, but there was other evidence to indicate a reduction of blood volume.

On the basis of clinical observation and the degree of reduction of blood volume, Keith found that the cases of wound shock fell into three groups. Refer to fig. 3, p.30.

SECONDARY WOUND SHOCK - THREE GROUPS

Group I - Compensated cases.

Group II - Partially compensated, borderline cases.

Group III - Uncompensated cases.

Grp.	General condition	Pulse rate	Sys.B.P. mm. Hg.	Blood volume %	Plasma volume %
I	good	90 - 110	100 ⁺	80 ⁺	85 ⁺
II	fair	120 - 140	70 - 80	65 - 75	70 - 80
III	poor	140 ⁺	80 ⁻	65 ⁻	60 - 65

Fig. 3

Keith pointed out that regardless of the presence or absence of hemorrhage in these cases of wound shock blood volumes were consistently reduced. Referring again to fig. 3, it will be noted that in group I, the reduction of blood volume was less than the reduction of plasma volume; in group II, the reduction of blood volume was about equal to the reduction of plasma volume; and in group III, the reduction of blood volume was greater than the reduction of plasma volume. This is of significance as will be later seen in part III.

Experimentally, Gasser, Erlanger and Meek (6) induced shock in animals and observed quite constantly a reduction of blood volume. Robertson and Bock (6), in their clinical observations which comprised a series somewhat smaller than that of Keith's, noted that with a blood pressure below 95 mm Hg. the blood volume is under 70%, and that with a blood pressure below 80 mm Hg. the blood volume is under 60%.

By this evidence, it is concluded that secondary shock is basically due to a diminished volume of circulating fluid, the phenomena of shock and hemorrhage being placed on similar planes.

III - MECHANISM: ULTIMATE FACTORS

Many suggestions have been presented in the past to account for the low blood pressure of shock and for its general phenomena. Some of the views are untenable in the light of modern knowledge of bodily function, and they will not be considered in this treatise. Theories that have been set forth to account for the development of the persistent low blood pressure of secondary traumatic shock are in relation to the basic factors heretofore mentioned in part II.

Theories in which the heart is regarded as the primary factor in the development of shock may be discounted in view of evidence in part II that has shown that neither in the action of the cardiac muscle nor in the nervous control of the heart is there a defect which would account either for the prompt reduction of pressure in primary shock or for its gradual reduction in secondary shock.

Theories in which reduced vasomotor tone is regarded as the primary factor in the development of secondary shock may be reviewed briefly, since many investigators in the past have looked upon

shock from this point of view.

The Exhaustion Theory:

According to Crile (6), a primary progressive exhaustion of the vasomotor center causes a general dilatation of peripheral arterioles which results in an accumulation of blood in the veins of the splanchnic area and a fall of arterial pressure. In this manner, the blood trapped in the mesenteric and portal veins fails to return to the heart and, by reducing its filling and discharge, closes the vicious circle by causing a further fall in arterial pressure.

The Inhibition Theory:

Meltzer (6) concluded that the injuries bringing on shock do so by favoring the development of the inhibitory side of reflex functions, for example, in the case of the circulation, converting pressor to depressor reflexes. This conception, like that of the preceding, involves depression of the peripheral vasomotor mechanism and pooling of blood in the viscera.

The Fatty Embolism Theory:

Porter (6) has advanced the conception that shock is due to embolism of the vasomotor center. He pointed out that fatty embolism of the pulmonary vessels is a frequent cause of death after fractures and after injury to fatty tissue as well as a sequel to operations; he also pointed out that the lungs do not act as perfect restrainers of fat entering the venous system. These facts are not disputed. The chief evidence reported by Porter consists in the fact that not only intravenous but also intra-arterial injections of fat emulsions cause, providing the fat reaches the medulla, a rapid failure of the vasomotor center. Other investigators have not been able to demonstrate this. It might be added that Porter emphasized that the viability of the vasomotor center remains unimpaired until the end (part II), and, in almost self-contradiction, he postulates that primary failure of the vasomotor center resulting from fatty embolism is responsible for the circulatory failure of shock.

The Acidosis Theory:

The demonstration that cases of low blood

pressure due to shock often have a low alkali reserve suggested the possibility that acidosis might be a fundamental cause of shock by bringing about peripheral vasodilatation. The suggestion, however, proved to be short lived, for experimental tests have shown that acidosis by itself does not cause shock. Dale and Richards (6) have shown that a large amount of acid may be injected into the bloodstream and the alkali reserve thereby reduced to a low degree without any fall of blood pressure to a shock level. They conclude, therefore, that the concomitant fall of blood pressure and alkali reserve in shock should not be regarded as indicating that a cause of shock is to be found in the lessened alkali content of the blood.

The Acapnia Theory:

Henderson (6), in his acapnia theory, maintains that as a result of pain or stimulation of afferent sensory neurons, an increase in respiratory volume occurs, thereby decreasing the CO₂ in the alveolar air and blood. According to Henderson's conception, this directly reduces the tone of smooth muscles and thereby diminishes the vascular support,

and, this, in turn, causes a stagnation of blood.

The Adrenal Exhaustion Theory:

On the basis that chromaffin tissue as well as adrenalin content of the adrenals is decreased in shock, it has been suggested that the removal of this secretion from the circulation results in peripheral dilatation of blood vessels, this being productive of shock. Short (6), who used a very delicate test, reported that the adrenalin content of the adrenal glands in fatal cases of shock is not noticeably reduced; furthermore, others have reported that the adrenalin secretion remains unaltered during shock.

The six preceding theories may be refuted and be concluded to be inadequate in view of evidence set forth in part II. Their chief and common defect is that they fail to account for the occurrence of a reduction of blood volume. Sufficient evidence has been presented in part II that a primary reduced vasomotor tone does not occur in secondary shock but that on the contrary a compensatory increased peripheral resistance is the rule. This

alone disproves the basic idea around which these conceptions have arisen. Furthermore, there is considerable clinical evidence, as cited in part II, that pooling of the blood in splanchnic areas does not occur but, as reported by World War surgeons, as a rule the splanchnic organs during secondary shock appear extremely pale.

Finally, theories in which reduced blood volume is regarded as the primary factor may be considered. There have been theories put forth notably by Malcom, Starling, and Erlanger and Gessell as reported by Cannon (6) that postulate a primary vasoconstriction with a consequent capillary congestion and reduction of blood volume, but these theories fail in that they do not suggest how a vasoconstriction capable of bringing about a reduction of blood volume would occur.

The Theory of Adrenal Cortex Deficiency:

Since death following adrenalectomy presents a form of circulatory failure and blood volume changes which are similar to those found in traumatic shock, and since animals practically moribund as a result of profound surgical shock could be revived

by intravenous injection of cortical hormone, Swingle (10) and his associates advanced the conception that the adrenal cortex is deranged in shock and that shock is caused by a deficiency of cortical hormone. Wiggers (6) states that it is scarcely likely that an insufficiency of cortical hormone could operate as acutely as the speedy development of surgical or traumatic shock would require.

THE PROBLEM OF THE "LOST BLOOD"

Since evidence of reduced blood volume has been found in both clinical and experimental shock without any indication of external hemorrhage, the question of the location of the "lost blood" remains - one of the most difficult to answer in the shock complex. Cannon (6) states that there are no indications that the missing blood is in the heart or lungs; it must be, therefore, in systemic arteries, capillaries, or veins or in the tissues.

It has been sufficiently proven that the lost blood is not in the arteries, Cannon states. With an efficient vasomotor center and a capable heart an adequate amount of blood in the arteries would be accompanied by satisfactory arterial pressure, this, of course, not being the case.

The view commonly held in the past has been that, in shock, blood is stagnant in the large venous reservoirs of the chest and abdomen. Cannon (6) states that this view has been based largely on experimental evidence which has been rather uncritically accepted. For many years, the most certain way to produce shock in a lower animal was by exposure and manipulation of the intestines, resulting in stagnation of visceral vessels notably in the mesenteric veins. Cannon pointed out that such a state is not actually seen in human shock cases. The testimony of surgeons who have had extensive experience in the recent war, notably Wallace, Fraser, and Drummond (part II) is opposed to such an idea. They testify that on opening abdomens of shocked humans they failed to find any evidence of splanchnic congestion.

Blood concentration in the capillaries:

In clinical studies made by Cannon (6), the first typical characteristic of the blood which was noted in secondary shock was a high capillary red count. In a series of 27 cases, hemorrhage was, as a rule, a complicating factor. This factor, of course, had the tendency, in itself, to reduce the red count. The increase in red count, therefore,

in virtue of the presence of associated hemorrhage is more striking. By his observations, Cannon concluded that in secondary shock a concentration of the blood, at least in superficial capillaries, is a typical occurrence. Whether or not the concentration found in capillary blood is true of all the blood was determined by counting capillary and venous samples taken simultaneously, such procedure revealing a more or less marked discrepancy between the two counts. The capillary samples were taken from widely separated parts of the body and the venous samples from an arm vein. Cannon plotted his observations on 16 cases as follows: Refer to fig. 4, p.41.

CAPILLARY AND VENOUS RED BLOOD CELL COUNTS
IN SECONDARY WOUND SHOCK

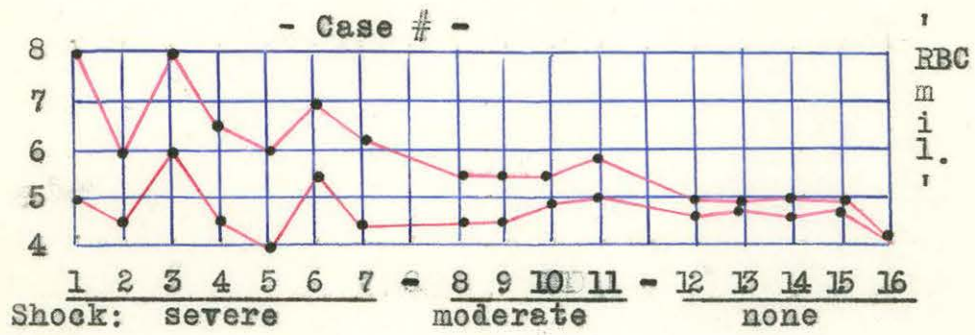


Fig. 4

Control observations made on normal individuals did not reveal greater differences than 3% between capillary and venous counts, and comparative samples of capillary and venous blood drawn before rising from bed in the morning proved that the discrepancy was not due merely to inactivity. Cannon pointed out that the difference between capillary and venous red counts varies roughly with the degree of shock and since the venous count is approximately at the normal level or below it, the difference must be due to concentration of the blood within the capillaries. Hematocrit determinations of volume % of corpuscles as well as hemoglobin determinations of capillary and venous samples confirmed observations made by counting. Observations of concentration of blood in the peripheral capillaries have been confirmed by Taylor (6) and also by Robertson and Boek (6). Cannon states that when shock is complicated by marked hemorrhage, the capillary red count becomes low but when compared with the venous red count the discrepancy between the two at once appears, the venous red count being lower still.

Transudation of plasma:

The concentration of blood is commonly accounted for by assuming that there is a passage of fluid from the vessels into the tissues. Careful studies made by Gasser, Erlanger and Meek (6), however, have shown that, in shock, the protein content of the plasma undergoes no marked change during the process of concentration of the blood. This indicates that the reduced blood volume is due to an escape of plasma as a whole.

In the normal individual after hemorrhage, there soon occurs a dilution of the blood due to the passage of fluid from the tissues into the bloodstream, a compensatory mechanism to maintain an adequate volume of circulating fluid. The mechanism of this is explained by Starling (6) as due to such reduction of blood pressure in the capillaries that the filtration pressure from within them no longer offsets the greater osmotic pressure of the plasma as compared with the lymph, and consequently water passes into the bloodstream. It is one of the unexplained features of shock that with the low arterial and venous pressures this process does not occur. Instead the plasma as a whole makes its escape through the

vessel walls. It seems probable that plasma transudation is not compensable by influx of tissue fluid into the bloodstream as in hemorrhage because of the fact that in the former condition osmotically active proteins leave the bloodstream and enter the tissues. Though in both shock and severe hemorrhage, blood volume is reduced, the processes occurring in the capillary region in the early stages of the two conditions are probably opposed; in shock, fluid passes outward through the capillary walls causing hemoconcentration and a reduction of plasma percentage; in hemorrhage, fluid passes inward to the bloodstream, compensating for the lost blood and reducing the concentration of corpuscles. With regard to shock, it is not known whether increased capillary permeability is uniform throughout the body or increased only in certain areas.

The Theory of Traumatic Toxemia:

The theory of secondary shock which has had, of late, an especially strong support is the traumatic toxemia theory of Cannon (6). This theory was developed as a result of the practical observation that secondary wound shock is frequently associated with extreme damage of the muscle tissue and occurs frequently after operations involving large muscle

masses.

Cannon postulated that a toxic factor produced at the site of injury in the damaged tissues is the ultimate causative factor initiating secondary shock. It operates by paralyzing capillary walls resulting in their dilatation and pooling of blood in the traumatized zone and by increasing their permeability resulting in marked plasma transudation, corpuscular concentration in the capillaries, and local edema. It is further postulated that the toxic factor by its absorption from the traumatized zone exerts a similar but less defined influence uniformly throughout the body. By considerable plasma transudation, there is a diversion of large quantities of fluid from the vascular tree resulting in reduced venous pressure and return to the heart, reduced volume of cardiac systolic discharge, and finally reduced arterial pressure, the latter proving fatal when falling below the level essential for nourishment of body tissues. Cannon emphasizes that the toxic factor is not usually working alone to bring about the shock state but also other factors, notably hemorrhage, cold and exposure. Bayliss states, as reported by Cannon, that though 25% of the blood may be lost with no permanent fall of pressure, this

loss, when combined with injury may bring about promptly the signs of shock.

Clinical Evidence:

1.

Secondary shock does not appear immediately after injury. Consequently it is not of the nature of a nervous effect. Furthermore, the state is usually well established before infection and therefore it is not of bacterial origin.

2.

Secondary shock is characteristically observed in association with extensive damage of muscles or with multiple wounds. The increase of urine nitrogen and of blood N.P.N. in shock cases may be accounted for as a consequence of absorption of material from the traumatized area and also, perhaps, as the effect of tissue damage done by circulating toxins.

3.

Everything that favors absorption at the region of injury is favorable to the development of shock. The development of shock is most severe when the region of damage communicates with the exterior by only a small orifice. The negative aspect of this evidence is presented by cases in which a large fleshy

mass, along with the skin which covers it, is carried away by a missile; in such cases, shock is slight or wholly absent.

4.

Anything that delays or checks absorption from the injured region delays the development of shock; but if there is a sudden removal of the check, serious results follow.

5.

Suppression of the injured region, i.e. removal of the damaged part from the body, if not too long delayed, causes shock to disappear.

Experimental Evidence:

1.

Crushing of hind limbs of animals produced shock and circulatory failure after lapse of 20 - 30 minutes.

2.

The objection that the fall of pressure after traumatization might be regarded as due to loss of blood and lymph into the damaged tissues since there is always a considerable swelling of the injured region was refuted by the following test:

Cannon removed the hind limbs of the experimental animals as symmetrical units, one normal, the

other injured, and weighed them (post mortem). The difference in weight which in some instances was only 10% of the estimated blood volume would not represent enough extravasated blood to account for the fall of pressure. On the other hand, Cannon admits that loss of blood by extravasation even when slight may play a role in the subsequent development of the low blood pressure.

3.

The objection that the circulatory failure might be due to nervous impulses aroused by trauma which on passing to the central nervous system might so effect it as to produce profound depression of nerve cells or reflex inhibition of the vasoconstrictor center as has been suggested by theories heretofore mentioned, was refuted in the following manner:

Cannon repeated experiments wherein the hind legs of animals were crushed but preceded the tests by cutting nerves supplying the region or by transectioning the cord above the lumbar roots and obtained the same results. Thus Cannon showed that there is no essential relation between the production of secondary shock and an excessive stimulation of the central nervous system.

4.

The objection that local injury would permit a sufficient amount of fat to be liberated to cause the blood pressure to fall was refuted thusly:

In a number of cases, post mortem examinations of the experimental animals by expert pathologists failed to reveal any fatty accumulations in the lungs.

5.

The absence of hyperpnea for sometime after injury in shocked experimental animals, although the fixed CO₂ capacity of the plasma was falling, rules out acapnia as a cause of the lowered blood pressure.

6.

Cannon postulated that a toxic factor produced at the site of injury is the ultimate causative factor in initiating secondary shock. He tested this idea in the following manner:

On a number of occasions, he ligated the iliac artery and vein in animals; following this he crushed the limb muscles supplied by the artery and vein that was tied. For some time after the average period for development of secondary shock

in animals whose vessels are left untied, there was no fall of blood pressure. As soon as the blood flow was restored, however, the pressure promptly fell to a low level.

Evidence supporting the inference that damaged tissue itself induces shock has been obtained by Mc Iver, as reported by Cannon. He established a crossed circulation between two animals; he crushed the muscles of the hind limbs in one and after an interval of 20 - 30 minutes, the blood pressure began to fall in both. Delbet and Karajonapoulos (6) found that injection of autolytic products of crushed muscles into experimental animals proved to be extremely toxic.

Dale and Laidlow (6) called attention to reduction of blood pressure induced by injection of extremely minute amounts of histamine intravenously into experimental animals. Histamine is derived from the amino acid histidine by the removal of CO₂. Later, Dale, Laidlow and Richards (6) called attention to the shock-like character of the reaction following histamine injection. The arteries are constricted, there is oligemia, an increased ratio of

corpuscles to plasma, and failure of the cardiac output. If the chest is opened, the heart is seen beating regularly, but its chambers contain little blood. Moreover, by the use of vital red, Dale demonstrated that a considerable part of the blood passes out of currency. Dale and Richards (6) have further analyzed the action of histamine. They point out the low blood pressure is apparently due to a series of changes in which dilatation of the capillaries and pooling of blood within them, poisoning of their endothelial walls so that they are abnormally permeable, escape of plasma through these walls into tissue spaces, and consequent concentration of the corpuscles are the main features.

Abel and Kubota, as reported by Wiggers (21), have demonstrated that histamine or histamine-like substances are formed when tissues are injured or proteins partially digested, and that these substances are present in the intestinal mucosa and many glandular secretions. The final link was completed to formulate a toxemic theory of shock which satisfactorily explains not only the onset of various forms of secondary surgical and traumatic shock, but which may be extended to include shock following intoxications and infections as well as anaphylactic

shock.

Lewis, as reported by Wiggers (21), states that since essentially similar responses are evoked by a great variety of irritants - mechanical, thermal, chemical, electrical, and nervous - since injections of minute quantities of histamine cause similar reactions, and since histamine or related substances are known to be formed by more extensive destruction of tissues, the triple response following any irritation may be assumed to be the result of the local formation of histamine-like substances. The primary capillary dilatation and increased permeability is thus logically referred to direct chemical action of the histamine-like substance, and the antidromic (axon reflex) arteriolar dilatation surrounding the zone of irritation may be attributed to stimulation of sensory nerve endings by this substance.

Cannon (6) states that the significance of these observations is that the action of histamine may reasonably be regarded as typifying the action of a large class of poisonous protein derivatives, i.e. products of partial digestion, of bacterial action, and of tissue extraction.

Dale (6) states that since traumatic shock is probably due to action of toxins arising in injured

tissue, places it in the same general category with anaphylactic shock, for that likewise is probably the consequence of poisoning by protein cleavage products. The shock-like condition produced by toxic substances arising from intestinal obstruction which Whipple (6) has done much to elucidate becomes related to traumatic shock in Cannon's opinion. Again, there is much to indicate, according to Cannon, that shock following burns is closely related to traumatic shock.

More recently Blalock (7) and his co-workers, in a long series of experiments, have presented some very interesting and significant evidence as pertains to the ultimate mechanism of shock.

Blalock emphasizes that the mechanism of the production of all types of shock is not the same and that they cannot all be satisfactorily be explained by one theory. He points out that the group of findings characteristic of the shock syndrome may be encountered under a variety of circumstances: following extensive burns, trauma, operation, internal or external hemorrhage, following perforation of a hollow viscus, complicating the infectious fevers,

in acute pancreatitis, in pulmonary embolism, associated with continued vomiting or diarrhea, in heat-stroke and in many other conditions. He points out the close relationship of shock and hemorrhage and groups them both under the heading of acute circulatory failure. Blalock believes the central problem in the study of shock should be the determination of agencies responsible for the decrease in venous return to the heart.

Experimental Findings of Blalock and Co-workers:

1.

Blalock repeated the experiments of Cannon which contributed the main evidence for the toxemia theory. In Cannon's experiments, it will be remembered, after one of the extremities of an experimental animal was traumatized, there was not found sufficient bleeding into the traumatized area to account by itself for the reduction in blood pressure; it was then assumed that the continued fall in blood pressure following trauma was produced by the absorption of some depressant substance. Blalock observed that the swelling which followed the injury was not limited to the area directly traumatized but extended into the loose tissues of the groin and flank. Cannon, in comparing

the weights of the traumatized and non-traumatized limbs, in attempt to determine the amount of local loss of fluid into the injured part, performed their amputations by symmetrical cuts across the upper thighs. Blalock, in his experiments, amputated higher and above the site to which the swelling extended in the traumatized limb, and the difference in the weights of the injured and non-injured parts was determined. This difference indicated the loss of approximately 50% of the total blood volume into the injured area, and a comparison with experiments in which death was produced by uncomplicated hemorrhage showed that it was sufficiently great by itself to account for the decline of blood pressure. Parsons and Phemister confirmed these experimental observations.

(16)

2.

In another series of experiments, Blalock burned about one half of the body surface area of deeply anesthetized animals. This resulted in the escape of a sufficient volume of blood plasma into the injured tissue spaces to account for, by itself, the decline of blood pressure which ensued after a few hours. Marked hemoconcentration was noted.

3.

Shock associated with trauma is characteristically accompanied by hemoconcentration and capillary congestion, while uncomplicated hemorrhage from a large vessel is characteristically accompanied by hemodilution and capillary anemia. But Blalock (4), by evidence obtained from one group of his experiments, has attempted to show that findings in shock due to hemorrhage are not necessarily different from those in shock due to trauma.

Blood pressure was gradually reduced to a low level in a number of dogs by slow withdrawal of blood from the femoral artery. Local anesthesia was used. Some of the animals died after a short period of time, and a few of these showed hemoconcentration and capillary congestion. In cases where death was delayed by transfusion, the blood pressure being kept at low level for several hours, all died despite the fact more blood had been introduced than removed. In these cases, hemoconcentration, capillary congestion, and hemorrhages in many of the tissues were consistently noted. From these observations, Blalock concludes that hemorrhage alone may present the same findings as shock due to trauma if the blood pressure remains

at a low level for a considerable time preceding death. These findings are not observed in patients succumbing to uncomplicated hemorrhage, Blalock states, because death follows rather rapidly usually without a prolonged period of low blood pressure.

4.

Evidence as to the mechanism of the production of different types of shock was obtained in another group of Blalock's (13) experiments in which the cardiac output and blood pressure were determined frequently during shock development in the experimental animals used. Briefly, the following points were noted:

- (a). Slow withdrawal of blood was associated with an early decline in cardiac output and a subsequent decline in the blood pressure.
- (b). Trauma to an extremity was followed by alterations similar to those in (a).
- (c). The injection of histamine was associated with an early decline in blood pressure and a subsequent diminution in cardiac output.
- (d). The injection of procaine in excess intraspinally was followed by alterations similar to those in (c).

These observations have been confirmed by Roome, Keith, and Plemister (18). With regard to shock mechanisms, Blalock assumes that in both (a) and (b) there was a primary decrease in blood volume as a result of loss of whole blood, obviously, in (a) and as a result of local loss of whole blood and of local plasma transudation in (b), the latter assumption being opposed to the toxemia hypothesis. With a gradual primary decrease in blood volume, the sequence reduced venous return to the heart - reduced cardiac output - declining arterial pressure is readily understandable. Blalock further assumes that in both (c) and (d) there was a primary peripheral vascular dilatation, that in (c) this was due to direct action of histamine on the vessel walls, that in (d) this was due to indirect action through the nervous system, and that (c) and (d) may be considered as forms of primary shock. With a sudden widespread vascular dilatation, the sequence fall in arterial pressure - reduced venous return to the heart - reduced cardiac output is understandable.

Blalock has classified peripheral circulatory failure from a physiological point of view

thusly:

1. Hematogenic Shock

Hematogenic shock is the typical traumatic shock in which the important primary change is a reduced blood volume. This is followed by reduced venous return to the heart and reduced cardiac output and finally by a decline in arterial pressure. A compensatory vasoconstriction usually immediately follows the primary change and may maintain the blood pressure near the normal level. If the blood volume continues to diminish, the blood pressure declines in spite of vasoconstriction, and if the process continues beyond a certain point, the vasoconstrictor mechanism fails and vasomotor collapse results. At this stage, many other factors produce a vicious circle in which the inadequate circulation further increases the severity of the condition. Conditions associated with hemorrhage and with trauma to muscles are examples of this type.

2. Neurogenic Shock

Neurogenic shock includes that group of conditions spoken of as primary shock or collapse. A primary peripheral vasodilatation occurs as a

result of a diminished constrictor tone due to adverse stimuli from the nervous system. The arterial pressure falls immediately; this may be restored soon if the condition is not serious or may continue resulting in diminution of venous return to the heart and cardiac output. Neurogenic shock may be due to action on the nervous system directly, as by spinal anaesthesia, or it may be reflex, as from a severe blow to the epigastrium or testicle.

3. Vasogenic Shock

Vasogenic shock is basically similar to neurogenic shock, i.e. the primary change is a peripheral vasodilatation, but this change is brought about by direct action of substances on the vessel wall. Histamine may produce its characteristic effect in this manner.

4. Cardiogenic Shock

Shock occurring as a result of primary disturbance of the heart is rare. The rapid accumulation of fluid in the pericardial cavity, as from traumatic hemopericardium, may cause this effect and is known as cardiac tamponade. There is a venous

distension in contrast to the collapsed veins seen in peripheral circulatory failure.

5. Multigenic Shock

Many instances of shock in man are complicated and involve combinations of the above forms as causative to its initiation.

Blalock concludes that in all cases of shock there is a disproportion between the volume of blood in active circulation and the capacity of the vascular bed. This might be due to a primary change of either of the two or both.

IV - PERSONAL REFLECTIONS

In the main, conflicting opinions as to the nature of shock appear to-day to be centered about the questions as to whether or not, in secondary traumatic shock, toxic absorption from the site of injury with local and generalized plasma transudation as maintained by Cannon in his traumatic toxemia hypothesis, or local extravasation at and about the site of injury by itself as maintained by Blalock is the existing initiating mechanism.

I have in my mind a series of experimental tests that might be tried which, perhaps, could be of some value in affirming or denying the theory of traumatic toxemia as pertains to secondary traumatic shock. I am taking the liberty, at this point, to outline in brief what my plan of approach would be, and, at the same time, I invite my readers' inevitable criticism.

The work would necessitate the use of three dogs for one complete experiment, and, in event of significant results in the first experiment, three dogs for each successive confirmative experiment. The three dogs of any given group preferably would be

members of the same litter and each of about the same size and weight.

A. Experimental Procedure

Preliminary Work:

Select two dogs of a group of three, match each dog's blood against the other's for any possible agglutinative interaction, and, if negative, mutually transfuse the same two dogs by a direct method and with volumes approximately equal to the estimated blood volumes of either animal, (both animals should be of approximately the same body weight). The blood in either dog then should consist of a good admixture of the bloods of both. Observe the animals closely for about one hour following transfusion and at intervals for a period of 24 hours in attempt to detect possible transfusion reactions.

Experiment Proper: - after 24 hours,

Part I:

Anesthetize dog #1 (one of previously transfused animals) with evipal rectally. This anesthesia has been used, of late, in certain experimental investigations at the University of Nebraska, College of Medicine, Department of Physiology with satisfactory

results. As soon as animal is well under the surgical degree of anesthesia, i.e. when good relaxation is obtained, arrange blood pressure equipment to take a continuous carotid arterial tracing. When this is in order, crush the muscles of both lower extremities with some heavy blunt object. If, in accordance with previous experiments of this nature, the onset of secondary shock should be from 15 - 30 minutes after injury as evidenced by declining arterial pressure. Significant signs as respiratory alterations, mucus membrane pallor, etc. should be noted. About ten minutes after the onset of shock (or less if the circulation seems to be failing rapidly), doubly ligate one upper extremity high and tight enough to completely impede all circulation, withdraw as much blood as possible by artery and vein, replace with an equal volume of 10% argyroll, amputate between ligatures and lay extremity aside. When the dog's respiration begins to fail, withdraw as much blood as possible from the animal by artery and vein, add oxalate crystals in sufficient quantity, and measure the volume obtained.

Part II:

Anesthetize dog #2 as in part I, transfuse

blood obtained from dog #1 into dog #2 by femoral vein and at the same time withdraw an equal volume of blood from the opposite femoral vein. A continuous blood pressure tracing should be taken as in part I. If secondary shock supervenes, approximately ten minutes after its onset (or less if the circulation seems to be failing rapidly) repeat upper limb procedure as in part I.

Part III:

Anesthetize dog #3 and repeat upper limb procedure as in part I.

Part IV:

Remove hair from the three amputated extremities and have prepared three frozen sections taken transversely through the soft tissues at corresponding levels of each of the three limbs. Examine the nine sections microscopically for relative quantities of extravasated silver.

B. How Possible Results Might Be Evaluated

1.

If dog #2 should develop secondary shock, this would indicate, I believe, that secondary traumatic shock is, at least in part, due to toxic absorption from the

site of injury, such toxic factor probably exerting a widespread influence on the vascular bed tending to bring about a disproportion between the blood volume and the capacity of the vascular tree by either peripheral vasodilatation or an increase in capillary permeability with plasma transudation or both. A possible transfusion reaction simulating shock would be improbable in case of negative results in preliminary work.

2.

If considerable quantities of silver were found to be extravasated in the sections from amputated extremities of dogs #1 and #2, and none or a negligible amount in dog #3, this, I believe, would suggest that a generalized increased capillary permeability with plasma transudation accompanies secondary traumatic shock.

If positive results could be obtained in this experiment and in repeated ones, it could be concluded that secondary traumatic shock is probably, at least in part, on a toxemic basis and that a generalized increased capillary permeability with plasma ~~plasma~~ transudation is at least part of the modus operandi.

If results were found to be repeatedly negative, it could then be assumed that secondary traumatic shock

probably is mainly initiated by local extravasation
at and about the site of injury.

V - MODERN CONCEPTION

As a means of summerizing the salient facts and inferences heretofore presented, it is a good plan, I believe, to attempt to bring together in resumé the various lines of evidence as to the nature of traumatic shock. This may best be done, I believe, by graphic correlation of essential points both factual and theoretical.

TRAUMATIC SHOCKINITIATION:TRAUMASECONDARY OR HEMATOGENIC SHOCK - delayed and progressiveI
HEMORRHAGE - local

(secondary compensatory hemodilution on basis of reduced capillary hydrostatic pressure which then fails to offset the osmotic pressure of the plasma which is greater than that of the tissue fluids)

II
Liberation of histamine-like toxin in damaged tissue

1. Local capillary paralysis and dilatation, increased permeability and stasis with pooling of blood in traumatized zone and PLASMA TRANSUDATION
2. Toxic absorption with generalized increased capillary permeability and plasma transudation (?)

(secondary hemoconcentration in the capillaries on basis of plasma transudation)

I, II, or Both

OLIGEMIA with chlorid loss

CIRCULATORY FAILURE WITH DECLINING BLOOD PRESSURE and pallor

MORBID TREND:

Increased Sympathetic Activity:

1. Rapid Thready Pulse (rapidity on basis of carotid sinus and arch reflexes; smallness on basis of reduced intracardiac tension)
2. Compensatory Vasoconstriction (on basis of above reflexes)
3. Elevated Blood Sugar (probably on basis of adrenalin-hepatic mobilization)
4. Increased Sweat Secretion

PRIMARY, NEUROGENIC OR VASOGENIC SHOCK - sudden transient or fatalI
REFLEX NERVOUS REACTION
as from rupture of abdominal viscus - or direct nervous reaction as from spinal cord injuryII
DIRECT ACTION ON VASCULAR BED by toxic histamine-like substancesCARDIOGENIC SHOCK
(uncommon)

Ex. Traumatic Hemopericardium

VASOMOTOR COLLAPSE

CARDIAC INSUFFICIENCY

Peripheral Vascular Damage (vicious cycle)

(B.P. below critical level of 80mm Hg.)

DEFECTIVE METABOLISM AND PROGRESSIVE TISSUE DEVITALIZATION

1. Subnormal Temperature
2. Subnormal Alkali Reserve with acidotic tendency
Hyperpnea of Hypercapnia
3. Terminal Stages:
Bulbar Collapse:
Respiratory Failure
Final Vasomotor Collapse
Cardioinhibitory Release with Cardiac Dilatation
General decline of reflex excitability, coma, death.

Fig. 5

VI - MANAGEMENT

A. PREVENTION

The best treatment as pertains to traumatic shock, of course, is prevention, but this is routinely possible as a rule only in those cases of elected surgical trauma. However, in cases of accidental trauma, early and proper treatment does much to minimize the severity of shock and at times probably prevents secondary shock as has been pointed out by Cannon (6) in regard to wound cases during the War.

Blalock (3) points out that the incidence of shock associated with surgery has markedly declined in recent years and this has been due chiefly to better hemostasis, gentler handling and less exposure of tissues, and to advances in anesthesia. Prior to an operative procedure, Blalock states, patients should be given ample fluids except in grave emergencies and fluids should be administered during the operation if same is protracted. Maddock and Collier (14) have pointed out the great fluid loss during and after operations. They found that the sick surgical patient needs 2000cc of water to replace that lost by vaporization, 1500cc to replace that lost in the urine, and additional

quantities if vomiting and diarrhea are present or in cases with hemorrhage, transudation, or exudation. A patient particularly dehydrated before operation should be given even larger quantities. Other important precautions include insuring adequate liver glycogen by administration of dextrose when indicated, transfusion in cases of anemia, preoperative sedation, proper choice of anesthetic, absolute bedrest warmth and control of pain after operation, and careful observation of the pulse and blood pressure during the recovery period for any evidence of impending shock.

In cases of accidental trauma, early and proper treatment may prevent fatal shock. Elimination of factors that contribute to the production of shock other than the injury itself is important according to Cannon (6), notably exposure to dampness and cold, thirst and starvation, and exhaustion. Control of hemorrhage, splinting of fractures, early surgical treatment when indicated, control of pain and restlessness, and conservation of body heat are important first aid aspects.

1. Control of Hemorrhage:

Repeatedly, Cannon (6) noted that during the War individuals who have been severely injured

and are in an unstable condition may be readily reduced to shock by relatively slight hemorrhage. Furthermore, in association with serious wounds there is likely to be a considerable loss of blood and therefore an urgent need for hemostasis.

The handiest method of checking hemorrhage when a limb has been wounded is by the application of a tourniquet, but this procedure is at times attended by a certain degree of danger. Cannon has cited cases in which long exclusion of the circulation from a part of the body will result in the production of shock when the flow is restored to the anemic part. In most cases of hemorrhage incident to trauma the consensus of opinion is that direct compression to the wound itself is adequately effective in most cases in stopping hemorrhage permanently or temporarily until ligature of the bleeding point itself is performed. Wallace and Fraser as reported by Cannon state that if an artery is tied it should be tied proximal to the injury; that the use of the tourniquet should be limited to cases in which a limb is so badly mangled that it cannot be saved and when amputation is performed it should be proximal to the tourniquet to protect the patient from absorption of toxic material in the traumatized

area; and that very occasionally the tourniquet may be used in less serious wounds when there is marked uncontrollable bleeding but release of the tourniquet at intervals and attempts to stop bleeding by direct compression over the area is advisable.

2. Splinting of Fractures:

With the War came plentiful evidence that the agitation of a broken bone in damaged tissue results in a sharp fall of blood pressure - an effect which, according to Cannon (6), may be accounted for by further traumatization of the soft tissues resulting in the liberation of more toxic material. Experience during the years of the War proved to British surgeons, according to Cannon, that the use of the Thomas splint while transporting the wounded did more, perhaps, than any other agent to reduce the incidence of shock. The precaution of splinting when there is serious bone injury is especially important in fracture of the femur because of the large muscle masses surrounding this bone. In addition to minimizing further destruction of the soft parts, splinting lessens the occasion for pain.

3. Early Surgical Treatment When Indicated:

When occasion arises that necessitates a traumatized patient to be operated upon, this constituting

a definite added hazard, the operation should be done as promptly as possible after injury and before advent of secondary shock. In 1917 Santy as reported by Cannon (6), observed 340 cases of non-transportable wounded. In 79 of these the time between the reception of the wound and the surgical treatment was known, and in this same group the wounds were of similar severity. The observations are tabulated as follows:

<u>Hrs. Intervening</u>	<u>- No. Cases</u>	<u>- % Mortality</u>
1	10	10
2	9	11
3	8	12
4	11	36
5	9	33
6	12	41
8	8	75
9&10	12	75

It will be noted in the above table that as the number of hours intervening between the time of injury and surgical treatment increases the percent mortality increases, such being due to the advent of secondary shock with lapse of time following injury. Santy's observations were confirmed by Gatelier, as reported by Cannon, who treated 13 serious cases without waiting by limited excision of injured tissues or by amputation and had no deaths.

If secondary shock is already established when the patient is brought under surgical care, there is general agreement, according to Cannon, that simple measures such as warmth rest and fluids should be applied in attempt to improve his state before operative interference is begun except, of course, in cases with uncontrollable hemorrhage. The principle involved in the operative treatment of fully developed secondary shock is the same as that employed for prophylaxis against its development, i.e. there must be suppression as soon as possible of the traumatized area.

With regard to frank shock cases, and also those cases where shock is a probable pending factor, in whom surgical treatment is indicated a proper choice of anesthetic is important. Clinical evidence has indicated, according to Cannon (6), that a patient in shock may be anesthetized with nitrous oxide and oxygen and suffer no appreciable drop in blood pressure, whereas with ether or chloroform the drop is likely to be considerable and possibly highly dangerous. During his extensive experience as anesthetist in a casualty clearing station in Flanders, Marshall, as reported by Cannon, found in a large series of very severe cases that gas-oxygen anesthesia was followed

by no increase of shock whatever. Marshall's observations were confirmed by Bazett, as reported by Cannon, who likewise had abundant opportunities to make careful observations. It has been observed that high ratios of nitrous oxide to oxygen are productive of harmful effects but that a ratio of three parts nitrous oxide to one of oxygen is a mixture that can be used with safety.

4. Control of Pain and Restlessness:

Whether or not morphine analgesia and narcosis is beneficial in pending and frank shock cases, as far as the shock itself is concerned, is not actually known. Pain and restlessness is considered by many as a contributing factor in shock production and, in this light, morphine analgesia and narcosis probably helps. On the other hand, morphine's tendency to inhibit respiration and to reduce general metabolic activity is obviously detrimental. Marshall, as reported by Cannon (6), has testified that the severely wounded when deeply morphinized make an unsatisfactory recovery after operation. Cannon states that the drug should be given in quarter and half grain doses which, as a rule, suffice to make the patient comfortable and quiet.

5. Conservation of Body Heat:

Cannon (6) stresses the urgency of taking every precaution to conserve the store of heat which the body has and to give back to the body which it may have lost. He states that if the patient cannot promptly be placed in bed, ~~he~~ should be wrapped with blankets. More blankets are needed under the body than over; the reason for this is that the blanket protects against heat loss by the air which it holds enmeshed in its fibers; the weight of the body lessens the air space in the fabric and consequently reduces the amount of protection. If possible heat should be applied to the body in the form of hot water bottles placed preferably in positions whereby maximum effect is secured, for example Cannon states that one could be placed on the abdomen and the hands placed over it, another between the feet, etc. Heat should not be provided in a degree which will induce sweating which will tend to further deplete body fluid. Hot drinks are beneficial; not only do they help to restore body heat but they relieve the distressing thirst which is almost constantly complained of by the wounded.

B. TREATMENT

The treatment of cases of frank shock entails many of the same principles heretofore mentioned under shock prevention, i.e. control of hemorrhage, control of pain and restlessness, conservation of body heat, etc. In addition to these measures, it is essential to attempt by every rational means to raise the patient's blood pressure, for if there is a prolonged insufficient volume flow of blood certain definitely harmful effects ensue as previously emphasized. Three therapeutic approaches will be considered and evaluated, viz. postural change, medication, and restoration of blood volume by parenteral fluids.

1. Postural Change:

The head-down position for the patient in shock for the purpose of improving cerebral circulation is not likely to have an important influence in most cases according to Cannon (6) and to military surgeons during the War who tested this procedure in numerous instances. Furthermore it proved to be rather disturbing to the patient. On the other hand, Cannon states that postural change in primary shock may at times be serviceable. This procedure and bandaging of the extremities as well are used quite generally to-day and are

thought by many to have a beneficial effect.

2. Medication:

Whether or not beneficial effects result from various forms of medication in shock is questionable. Adrenalin and pituitrin (or pitressin) have been used for many years, according to Cannon (6), to improve the circulation in shock by their vasopressor effect. Cannon and others have maintained that a temporary rise of blood pressure as manifested after the administration of these two drugs is of practically of no benefit. Furthermore, damming the blood on the arterial side does not improve the volume flow in the capillaries which is the ultimate end sought for.

Blalock (3) is in agreement with this view and states that these drugs are useless particularly in shock of the hematogenic type. Digitalis is not indicated since it has been shown that the heart is rarely a primary factor in shock initiation. The value of caffeine and other stimulants is questionable.

3. Restoration of Blood Volume by Parenteral Fluids:

Since most cases of traumatic shock are basically due to an actual loss of blood volume by plasma transudation or hemorrhage or both, the treatment has been directed largely toward the restoration of blood

volume. The lost volume is best replaced by the introduction into the circulation of a fluid that will remain there, increasing the blood volume and raising the blood pressure. Experience and experiment have shown that a most important loss to be replaced is the plasma proteins since they constitute the osmotically active factor in holding fluids in the bloodstream. This has been stressed by Cannon (6) and by Johnson and Blalock (12). Replacement of lost chlorides is important. Since patients in shock rarely can take fluids by mouth, fluids are usually given parenterally and most often by vein.

(a) - Whole Blood:

By far the most satisfactory substance used has been whole blood; this has been pointed out by numerous workers during the War and has been confirmed repeatedly by many up to the present time. But since whole blood for transfusion must be typed, is expensive, and is not always readily available, other substances have been substituted, these usually attended, however, by less therapeutic success.

(b) - Blood Plasma - Lyophile Serum:

When there is hemoconcentration due to plasma transudation in the main rather than strict hemorrhage, the ideal fluid, according to Blalock (2), is blood

plasma. Harkins (11) has evidence that plasma transfusion in combating experimental shock is of even more therapeutic value than whole blood transfusion.

Methods for the preservation of normal blood serum in desiccated form have been developed by Elser, Thomas, and Steffen (9) and by Flosdorf and Mudd (10). Essentially, this procedure is one of rapid freezing at a very low temperature and rapid dehydration from the frozen state under high vacuum, leaving in dry form all the solid elements of the serum. The proteins appear to be unaltered and their antibody properties are preserved in full titre. In this form, serum may be preserved for extended periods and is readily dissolved in water to make an isotonic or hypertonic solution. Because of its rapid solubility, this product is called 'lyophile'. For intravenous use, serum must be processed twice by this method with an intermediate filtration to remove fat particles. Bond and Wright (5) in a series of experiments using lyophile serum intravenously to treat dogs in which the blood pressure was markedly reduced by intestinal manipulation, trauma to an extremity, or by acute hemorrhage, observed that the blood pressure was raised and maintained for at least several hours by this procedure. They conclude that the immed-

iate availability of lyophile serum, its theoretic suitability, and its action upon shocked animals suggest its use as a valuable treatment for clinical shock and hemorrhage.

(c) - 6-7% Gum Acacia in .9% Sodium Chloride;

Gum-salt solution has been and is considered of value by many authorities. Bayliss, as reported by Cannon (6), was the first to advocate this substance after careful analysis of various colloids. Among the group of materials studied by him, he found that gum acacia alone is free from serious objection and capable of replacing blood plasma and its proteins. A solution of 6-7% of it in .9% sodium chloride has the same viscosity as whole blood and the same osmotic pressure as blood plasma. It is chemically inert, can be sterilized without chemical or physical alteration, and it does not induce anaphylactic reactions when repeatedly injected. De Kruif, as reported by Cannon (6), has subjected gum-salt solution to very thorough tests as to its toxicity with negative results. On the other hand, Studdiford (19), a later investigator, has pointed out that occasionally gum acacia has toxic properties, notably upon the liver and upon the gaseous exchange of red blood cells. At no time has gum acacia been so thoroughly tested

clinically as it was during the War when its use was first advocated. Although many reports highly favorable to the use of gum-salt solution were made at and since that time, a few strong opinions have been expressed against its use. Cannon (6) states that both in the American and British armies, gum-salt, when used early in cases of shock and moderate hemorrhage, had excellent effects as a rule. On the other hand, when the wounded were brought in after prolonged exposure to cold and dampness, especially during the winter months, gum-salt had less or often no effect. However, it was found that under such conditions, blood transfusion also was often quite as ineffective as the artificial fluid in restoring the circulation.

(d) - Other Agencies:

Glucose and salt solutions in various strengths and mixtures of same have been widely used, but their diffusibility is so great, especially in face of increased capillary permeability, that the increase of the volume of circulating fluid is transient, and there is danger, especially if large quantities are introduced rapidly, of an added hazard, i.e. edema. Furthermore, Blalock (2) has shown that not only do these substances diffuse rapidly, but, because they dilute the remaining plasma

proteins, they further reduce the effective osmotic pressure. Blalock concluded that intravenous salt or glucose is usually not objectionable, however, except in excessive amounts and are often of some value as emergency agents particularly in cases of shock due to hemorrhage. Deitrick (5) has advocated the use of 5% sodium bicarbonate solution intravenously in cases with impending acidosis.

In cases of frank shock or of impending shock, it is of considerable importance to follow closely the clinical course of the patient. Prognostic guides of value, as emphasized by Cannon (6) and others, are repeated observations of the pulse and blood pressure; frequent determinations of the hemoglobin, red count, or volume% of red cells; and watchfulness as to the clinical appearance of the patient. A slower more forceful pulse, a progressive rise in blood pressure, and progressive hemodilution (subsequent to concentration) are indicative of progressive recovery. Recovery fails to occur as a rule, according to Deitrick, (7), if the blood volume has been reduced by as much as one half of the patient's normal blood volume.

In conclusion, it may be stated that the best treatment for shock is prevention when possible by proper and immediate first aid in accidental cases or by rational precautions in cases of elected surgery. In cases of frank shock, the best therapeutic approach has been shown to be restoration of the blood volume, plasma proteins, and chlorides by parenteral fluids, the relative value of same being in the order mentioned: (1) whole blood or blood plasma, (2) 6-7% gum acacia in .9% sodium chloride, and (3) glucose and salt solutions.

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