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President of the Ulster Medical Society

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Presidential Opening Address

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RECENT WORK ON SHOCK AND HAEMORRHAGE.

LADIES AND GENTLEMEN, My first duty, on assuming the Presidential Chair of this Society, is to thank the members for the honour they have done me in electing me to this important post. I crave your patience and indulgence for prospective shortcomings, and I promise to use my utmost endeavours to uphold the high traditions of this office, and to further the interests of this Society to the best of my ability.

During the year that is past the hand of death has been laid on two of our members.

One of our seven honorary Fellows – Dr. Robert Esler – has passed away at the ripe age of eighty-three years. Qualified in 1875, he soon began to exert an influence on the affairs of this Society, and in 1887 the members showed their appreciation of his worth by electing him to the office of President. Dr. Esler was closely identified with the professional life of this city for fifteen years. At the end of that period he went to London and built up a large practice in one of its important suburbs. He was a leading figure during the meeting of the British Medical Association held in Belfast in 1884, and published, for the use of visitors to that meeting, a Guide to Belfast and the North of Ireland, which was much appreciated. He was one of the founders of the Ulster Hospital for Women and Children, which was housed at that time in Chichester Street. He was a man of wide sympathies and varied attainments, and his name awakens pleasant memories in the minds of the older members of the Society.

Dr. J. B. Logan, of Ballynure, was called away in the prime of life. He qualified in 1903, and held, first, the post of Resident Medical Officer at the Union Infirmary, and later, that of House Surgeon to the Royal Infirmary, Bradford. He had not long started practice in Ballynure before he was overtaken by death, to the great regret of his friends and colleagues.

During the coming session we shall be, as usual, chiefly concerned with scientific business, but

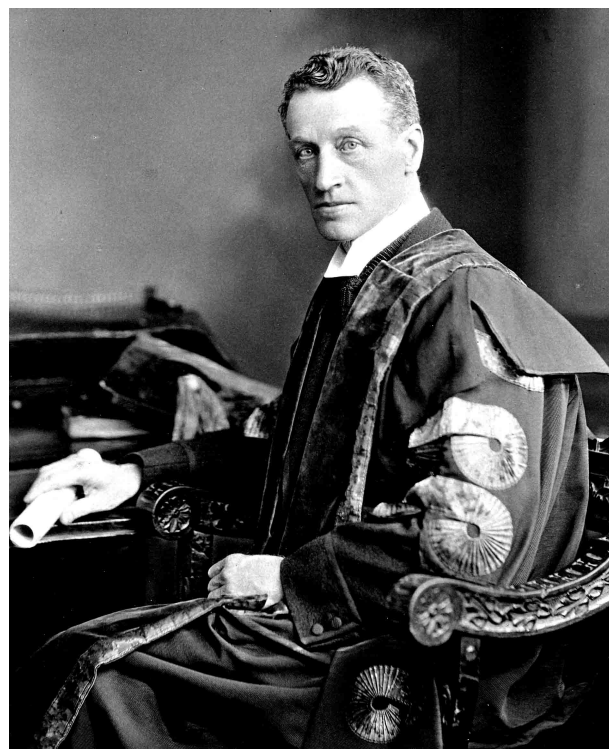


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it appears to me that the medico-political aspect of our work should not be entirely ignored. At the moment everything is in a state of flux, and our profession is entering upon a period of reconstruction. Just how that reconstruction will affect us in the North of Ireland it would be difficult to predict. I feel certain that, in the past, we have not sufficiently emphasised our position as a profession in directing the counsels of those who are responsible for the health of the community. It may be that during the coming session some suggestions may be made to help us to take our proper place as an advisory body on matters of public health.

There is another question that should be discussed by this Society in conjunction with the Ulster Branch of the British Medical Association, and that is the question of suitable remuneration for professional services. Under present conditions, medical fees are much too low. The drop in the value of the sovereign has hit hardly the members of our profession. While salaries and wages in general have risen, we alone have taken no steps as a body to obtain adequate remuneration for our members,

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though the work of the medical practitioner has improved enormously in the last twenty years. The struggling practitioner, under present conditions, will find it difficult, or impossible, to make suitable provision for his family, and will find himself compelled to work well into old age if he is to continue to live in a manner becoming his position. The young medical man just starting practice is severely handicapped by the rise in the cost of house-rent, furniture, instruments, drugs, dressings, &c., and he may be so harassed about ways and means that he cannot give adequate attention to those things that make for good and efficient work. The remedy is an all-round increase of fees, and the public are quite prepared for this. I would suggest –

- (1) That a joint meeting of this Society and the Ulster Branch of the British Medical Association be held to draw up a revised scale of fees.
- (2) That announcements be made in the public Press that a certain percentage, to be agreed upon by the joint meeting, has been added to the present rate of fees.
- (3) That this should be done at once so that a scheme may be ready for the beginning of the new year.

And now, Ladies and Gentlemen, I come to the subject of my presidential address. After much consideration I have decided to discuss with you some of the recent work on shock and haemorrhage, based largely on the experiences of the late war.

In August, 1917, the Medical Research Committee invited a Special Investigation Committee “to undertake the co-ordination of inquiries into surgical shock and allied conditions, with a view to the better correlation of laboratory and clinical observations.” This committee included British and American physiologists and clinicians of the highest repute. The Research Society of the American Red Cross in France also did useful work on shock. Reports were made to the Medical Research Committee and the Research Society of the American Red Cross in France, respectively, by medical officers (including pathologists and surgeons) in the forward area, so that the condition of the wounded man was thoroughly investigated from the aid post to the clearing station. The result of this co-ordinated work was a large increase in our knowledge of shock and the indications for treatment. In this address I shall avail myself of the work done by men whose opportunities for observation, and whose special training, well qualified them to undertake research. What follows is, to a large extent, a résumé of the

work done by these investigators.

At the very outset we are faced with the difficulty of determining what is the cause of the phenomena associated with the condition known as shock. We shall see presently that the outstanding feature of shock is a diminished blood pressure, but we are in the dark as to how this is initiated in the first instance. Several factors may be in operation, among the most important being loss of blood. It has proved very difficult to assess the exact role of haemorrhage, injury, exposure to cold, fatigue, terror, toxæmia, &c., in the production of the group of symptoms well recognised and universally described as shock. It has been found almost impossible to distinguish by symptoms, shock, haemorrhage and the toxæmia of gas gangrene.

Surgical shock, traumatic shock, or, as Cowell has called it, wound shock, may be defined as that depressed condition of the bodily functions which occurs when a patient receives a severe injury or undergoes a severe surgical operation. We now recognise primary shock and secondary shock. A patient, when first wounded, may suffer from primary shock, which may pass off, or be merged into, or succeeded by, secondary shock. On the other hand, a patient with such a severe injury as, say, a compound fracture of the femur, may show very little shock at first. During his transit, however, from the front line to the clearing station, he may gradually develop secondary shock, even when no further blood has been lost during his journey down. The nature of this secondary shock has been carefully investigated during the late war.

Cannon, of the United States army, summing up in the Reports of the Medical Research Committee, gives an excellent résumé of the work done, and the bearing of the results on previous views of the nature of shock. I cannot do better than quote freely from his report.

PREVIOUS THEORIES OF SHOCK.

1. ACAPNIA. – Yandell Henderson advanced the theory that shock was due to a reduction of the carbon dioxide of the blood by the forced breathing which, he says, insupportable pain always produces. This hyperpnoea involved a far greater ventilation of the lungs than normal breathing affords. CO₂ is the normal stimulant of the respiration.

According to this theory, breathing stops because there is not enough CO₂ left in the blood to excite the respiratory centre to activity. To this condition of diminished CO₂ in the blood Mosso gave

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the name “acpna,” from the Greek “a” – without – and “kapnos “ – smoke. This theory pre-supposes that a wounded man breathes vigorously as the result of pain. Observations on wounded men, however, in the front line, failed to support this idea of vigorous respiration. In fact, during shock, respiration is usually rapid and shallow, and not of such a character as to cause increased ventilation of the lungs. Cannon says that from the evidence in hand it appears that painful wounds are not directly associated with a hyperpnoea which would produce acpna.

If I have read recent literature aright it would appear that the question cannot be so easily disposed of, and one awaits with interest further debate thereon. Benjamin Moore, a distinguished alumnus of this school, in a recent paper (*Lancet*, September 13th, 1919), referring to excessive respiration as an antecedent to shock, says the balance depends on the relative rate of working of the circulatory and respiratory systems. “If,” he says, “as a result of a primary shock the circulation is only working at one-third of its usual speed, and the respiration is going on at the usual rate, the amount of carbon dioxide produced will be only one-third of the normal, while elimination proceeds at normal rate. The result must be that the alkalinity of the blood increases. It is thus seen that hyperpnoea need not necessarily be an antecedent factor to surgical shock, and that in the main the condition depends upon relative rates of circulation and respiration, although other conditions such as toxic products from wounds and muscle injury, and fatigue of nerve centres, undoubtedly play a part.”

2. ADRENAL EXHAUSTION. – Since removal of the supra-renal glands results in lowered arterial pressure (low blood pressure being the chief phenomenon of shock), and since secretion, or injection of the extract of the medulla of the gland, increases the pressure, the idea has been advanced that in shock there is adrenal exhaustion and consequent hypotension. Short (*Lancet*, March 14, 1914), however, found that the adrenin content of the glands in fatal cases was not notably reduced; and Mann (*Jour. Am. Med. Assoc.*) reported that total excision of the adrenals did not reproduce the phenomena of shock.

Cannon (“Bodily Changes in Pain, Hunger, Fear and Rage”: New York, 1915) brings forward experimental testimony that painful stimuli and asphyxia increase both the secretion of adrenin and the percentage of sugar in the blood, and that the sugar percentage does not rise if the adrenal glands are not co-operative. He showed that in shock cases

there was no lack of sugar in the blood (Paper III., Reports to M.R.C.). The high percentage of sugar found in his series of shock cases indicates that the adrenal glands are, if anything, over-active rather than exhausted. To make clear the connection between adrenal activity and increased sugar in the blood, reference should be made to Cannon’s book mentioned above. In well-fed animals the liver contains an abundance of glycogen or animal starch. The injection of adrenin can liberate sugar from the liver to such an extent that glycosuria results. Adrenin secretion is discharged in pain and strong emotional excitement, and plays a role in producing glycosuria under such conditions. The emotional origin of some cases of diabetes and glycosuria may thus be explained.

3. EXHAUSTION OF NERVE CENTRES. – The best-known exponent of the theory that shock is due to exhaustion of nerve centres is G. W. Crile. Crile says that shock is the result of the excessive conversion of potential into kinetic energy in response to adequate stimuli. According to him, the essential lesions of shock are in the cells of the brain, the suprarenals and the liver. The histological changes may be produced by emotion alone, by physical injury alone, by haemorrhage alone, by starvation alone, by insomnia alone; or these changes may be started by emotion, carried on hither by muscular exertion, another step by physical injury, another by haemorrhage, and so on until the cells are destroyed; or all of these factors acting simultaneously may produce the same result. According to this theory, all forms of shock are caused by overstimulation and consequent exhaustion. The most vital effect, according to Crile is impairment of the vaso-motor mechanism.

As the outcome of Crile’s researches he developed a new principle of operative surgery, to which he gave the name of anoci-association, or the exclusion of all nocuous or harmful physical and psychical stimuli from the brain.

“By an assuring pre-operative environment; by the definite dulling of the nerves through the administration of a narcotic; by a non-suffocating, odourless inhalation anaesthetic; by a local anaesthetic to cut off all afferent impulses during the course of the operation; by a second local anaesthetic of lasting effect to protect the patient during the painful post-operation hours; by gentle manipulation and sharp dissection – by the combination of all these methods the patient is protected from damage from every factor excepting those which exist in the diseased condition for which relief is sought.” – Crile

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and Lower. Anoci-association.

There can be no question that the work of Crile has contributed greatly to the comfort and well-being of a great mass of suffering humanity, though his conception as to the causation of the phenomena of shock has been much criticised. Evidence has been brought forward to disprove the theory of exhaustion of the cells of the brain, liver and adrenal glands. We have seen that the adrenals are not exhausted. Cannon criticises the theory of primary exhaustion of nerve cells as follows:—

“A lowering of arterial pressure is not proof that the vaso-motor centre is inactive or exhausted, for arterial pressure may be too low in consequence of haemorrhage — that is, when only a small volume of blood is delivered to the heart for each contraction.”

Furthermore, it has been found (Porter, *Am. Jl. Physiology*, 1907), that when an animal is in extreme shock, both pressor and depressor reflexes still occur. The occurrence of depressor effects proves that some tonic activity of the vaso-motor centre is still present, for otherwise its action could not be depressed; and the pressor responses show that the centre is still capable of increased action when stimulated. It has been shown (Pike, Guthrie and Stewart, Jr. *Exp. Med.*, 1908), that the vaso-motor centre is more capable of withstanding the adverse influences of anaemia than any of the other vital bulbar centres — the respiratory, the cardio-inhibitory, or the swallowing mechanisms. Its capacity to function is the last to disappear in total anaemia and the first to reappear when the blood flow is restored. In cases of profound shock it is observed not infrequently that when a pulse cannot be felt at the wrist, it can be felt where the arteries are larger. This is explained by the complete obliteration by contraction of the peripheral arteries, owing to their emptiness.

The evidence from functional disturbance that parts of the nervous system other than the centres controlling the circulation are exhausted in any strict sense of the term is, according to Cannon, meagre. Even when the blood pressure has been much reduced the intelligence remains clear, the patient may be restless rather than somnolent, and often exhibits surprising muscular power.

The evidence for exhaustion which has been advanced by Crile and his co-workers is mainly histological, and is based on examination of nerve cells taken from shocked animals. The suggestion is reasonable that any cell alterations that may occur in shock are the resultant of the low blood pressure rather than its cause.

THE CARDIAC FACTOR IN SHOCK.

We have seen that the low blood pressure of shock is not due to exhaustion of the vasomotor centre. Is it due to exhaustion of the cardio-inhibitory centre, or to failure of the heart's muscle?

Mann found (*Jour. Am. Med. Assoc.*) that in the shocked Animal the cardio-inhibitory centre is responsive to reflex stimulation and also to increase of intracranial tension. The nervous check on the heart, therefore, is not impaired. The rapid cardiac beat with low blood pressure is what is to be expected according to the reciprocal relation which commonly prevails between heart rate and arterial pressure.

That the heart muscle is not defective has been shown experimentally. Raising the arterial pressure to a high level does not overwhelm the heart; when properly supplied with blood it meets the situation and contracts with vigour (Cannon). Low arterial pressure, if prolonged, may, however, cause impairment of the heart, and the latter may beat less energetically. Thus the efficiency of the organ may diminish in time, though no primary defect has been present.

THE LOST BLOOD IN SHOCK.

In shock as well as in haemorrhage the low blood pressure is due to diminished volume of blood in circulation. It is easy to see how this occurs in haemorrhage, but the explanation in shock is not so obvious. The vasomotor centre is not at fault, and the heart is capable of assuming any reasonable burden placed upon it. We must, therefore, assume that in shock there is not enough blood in circulation to fill adequately the blood vessels. Where is the lost blood?

IT IS NOT IN THE ARTERIES. — With an efficient vasomotor centre and a capable heart an adequate amount of blood in the arteries would be accompanied by high arterial pressure. That the pressure is low signifies that the heart is not supplied with enough blood to fill the arterial system.

IT IS NOT IN THE VEINS. — 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, and especially in the splanchnic area. The shocked man is said to “bleed into his own abdominal veins.” This view arose from a method which is commonly used to produce shock in animals — exposure and manipulation of the intestines. Under these circumstances the mesenteric veins stand out prominently, blood gathers in the intestinal walls and becomes more concentrated there, and the structures that have been freely handled appear as if

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inflamed. Such a condition is not seen in natural shock. Surgeons at casualty clearing stations who have performed many hundreds of abdominal operations on patients in all degrees of shock have not found any primary congestion (Wallace, Fraser & Drummond, *Lancet*, 1917, p. 727).

The method employed to produce shock in animals has given rise to misleading inferences as to what occurs in shock brought on by wounding other regions than the belly. As Cannon says, if the lost blood were in the systemic veins it should be possible promptly to remedy this state of affairs by placing the patient's body in a slanting, head down position, bandaging the limbs and compressing the abdomen, all of which methods have been extensively tried with results which do not indicate that the blood which is out of circulation is stagnant in the large venous channels. There are no observations that the veins are even slightly dilated in shock (Cannon).

IS IT IN THE CAPILLARIES? – If in wound shock the lost blood is not in the arteries and probably not to a great amount in the veins it must be mainly stagnant in the capillaries. Cannon, Fraser and Hooper made observations in "Some Alterations in the Distribution and Character of the Blood in Shock." Their observations were based on records from ninety-eight cases of shock and haemorrhage, and on fourteen control cases. The routine examinations consisted of a count of the red blood corpuscles, estimation of the haemoglobin, and determination of the percentage of corpuscles in the blood. In discussing the blood changes it was found desirable to distinguish between cases of severe or extreme shock and those of a moderate character. The severe cases had a systolic pressure of not over 70 mm. of mercury, and the moderate a systolic pressure of not over 90 mm. of mercury.

The first noteworthy characteristic of the blood in shock was a high capillary red count. In the majority of the cases the count was six million per cubic millimetre or over, and in some it was more than seven million corpuscles. When haemorrhage, as a complicating factor, tending to reduce the blood count, is considered, these high counts are striking. They indicate that in shock a concentration of the blood occurs at least in the superficial capillaries. Capillary and venous samples were taken at the same time and a more or less marked discrepancy between the two was revealed. The capillary samples were taken from widely separate parts of the body, from the lobe of an ear, from a finger or from a toe; the venous samples from an arm vein. This striking discrepancy between the capillary and venous red

counts in the severe cases was reduced in the moderate cases, but even in them the difference was nearly a million corpuscles per cubic millimetre. Control observations made by these observers on normal individuals did not reveal greater differences than three per cent, between capillary and venous counts. It is clear, therefore, that the difference between capillary and venous red counts varies roughly with the degree of shock, and since the venous count was approximately normal the difference was due to concentration of blood or stagnation of corpuscles in the capillaries.

In all probability the low temperature typical of patients in shock is an important factor in producing the increased corpuscular content of the capillaries. It is known that blood drawn from a cold finger contains a larger number of corpuscles in a given volume than that drawn from the same finger after it has been warmed. The capillaries of the mouth, less exposed to loss of heat than those of the skin, contained blood nearer the venous blood in concentration than did those of the skin, but still indicated stasis.

When the capillary stagnation has become established it does not promptly disappear. Cannon, Fraser and Hooper saw a patient who had recovered from severe shock, but whose hands from wrist to finger tips, in spite of being warmed, were still bluish-grey with stagnant blood.

Haemocrit determinations of the volume per cent. of corpuscles in capillary and venous blood were also made. The results confirmed the discrepancy found in the counts. The capillary corpuscle volume was greater than the venous by amounts ranging from 12 to 23 per cent.

The difference was further confirmed by haemoglobin determinations. The capillary haemoglobin readings exceeded the venous by amounts ranging from 6 to 29 per cent.

The influence of cold in producing stagnation or concentration of blood in capillaries has been mentioned. It is in the capillary region that the corpuscles are most exposed to contact with the vascular wall – i.e., in this region and in the finer arterioles friction is greatest. When arterial pressure is low, as in shock, there is naturally a tendency for the blood corpuscles to gather in the place of greatest resistance, and if these channels are differentiated by cold so that some (the warmer) offer easier courses, and others (the cooler) more difficult courses for the blood to take, the accumulation of corpuscles, especially in capillaries of the skin and limbs, may reasonably be accounted for as a partial stasis. The blood thus checked in capillary areas would be out of

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currency, and by failure to return to the heart would contribute to lowering of arterial pressure.

The blood counts of haemorrhage showed several distinctive features. The capillary red count was usually much lower than that of shock alone, but by no means so low as the pallid appearance of the patient might lead one to suspect. Another feature of haemorrhage which was found fairly constantly was a relatively low haemoglobin reading. Cannon says that although the discrepancy between the capillary and the venous count in shock is more marked in the superficial areas, a noteworthy difference is found even in the deeper regions. For this condition of loss of blood from currency he suggests the term "Exaemia."

It would appear that the only way to account for the ability of the circulatory system to accommodate itself to injection of large amounts of blood is to assume the utilization of capillaries not ordinarily filled (Worm Müller). The distensibility of the capillaries should also be considered, as well as the concentration of the capillary blood, and, according to Cannon, the conclusion is apparently warranted that the capillary capacity is sufficient to contain the lost blood in shock, and that the chances of it doing so are greater the more concentrated the lost blood becomes.

THE VISCOSITY FACTOR IN SHOCK.

The viscosity of the blood consists of the internal friction of the plasma, the friction of the corpuscles with the plasma and with each other, and the frictional contact of the corpuscles with the vessel walls, especially in the capillaries. Concentration by increasing the number of corpuscles per unit volume causes the viscosity to rise. A fall of temperature has a similar effect. The incidence of shock is greater in cold weather, especially when the cold is accompanied by rain, so that the clothing of the wounded man is wet through.

ACIDOSIS AND SHOCK.

A good deal of attention has been paid to the role of acidosis in producing shock, and in France for some time it was thought to be an important factor to be dealt with by alkaline drinks and intravenous injections. At one time a great many intravenous injections of bicarbonate of soda were given, but the results were disappointing. After witnessing the results of experiments at the Lister Institute in February, 1918, Drs. Bainbridge, Bayliss, Cannon, Dale

and Richards accepted the demonstration as deciding in favour of the view that reduction of the alkali-reserve does not necessarily by itself cause shock in the otherwise normal animal but may favour the shock-producing action of other factors which have yet to be explored.

Wright, Crile and Cannon have written much on the question of acidosis in shock, and Milroy has shown that haemorrhage may be a cause of acidosis.

Cannon briefly reviews the subject, and the following abridged account is taken from his report to the Medical Research Committee.

In all probability the lowering of the alkali reserve in cases of shock and haemorrhage is due to a fixed union of the alkali with acids which, unlike carbonic acid, do not pass off in the lungs. The production of such acids is known to occur when oxidation is interfered with. A low blood pressure, with slow circulation, cold, and corpuscular stagnation would all co-operate to check the normal oxidative processes of the body and to increase the production of acid metabolites. Cannon continues:—

1. There is evidence that acid or a change in the blood in the direction of acidity may have depressive effects on the blood pressure. On the other hand, lack of oxygen, increase of carbonic acid in the blood, or injection of weak organic acids, all of which increase the H-ion content, stimulate the vasomotor centre and cause a rise of blood pressure.
2. Increase of the carbonic acid of the blood affects the cardiac contraction. Patterson has shown (Proc. Roy. Soc., Lon., 1915), that when the H-ions are increased by this means cardiac muscle relaxes to a greater extent in diastole and contracts less in systole.
3. Increase of carbonic acid increases the viscosity of the blood. This would tend to produce stagnation of corpuscles in capillaries.
4. It has been noted by Hamburger that the size of the corpuscles is increased by the action of carbonic and other acids.

All these effects of increasing the H-ions of the blood would be, according to Cannon, favourable to a continuance of low blood pressure.

Bayliss (Intravenous Injections in Wound Shock) is unable to confirm the statement that the viscosity of the blood is increased by acid, and says, with regard to the swelling of the red blood corpuscles described by Hamburger, that the diameter is not increased, though they become more globular in shape. This fact, he says, may explain why no increase in viscosity is produced. Bayliss thus sums

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up: "We finally arrive at the conclusion that such an acidosis as is sometimes present in wound shock is innocuous in itself and may even be beneficial in increasing the supply of oxygen by greater pulmonary ventilation. The use of alkaline injections is unnecessary."

TRAUMATIC TOXEMIA AS A FACTOR IN SHOCK.

During the later stages of the war attention was directed to traumatic toxæmia as a factor in shock.

Dale, Laidlaw and Richards presented in 1919 a Report on the Action of Histamine: its bearing on Traumatic Toxaemia as a Factor in Shock. The shock with which they were concerned was what has been called secondary shock. For primary shock, the condition which Crile and his followers have distinguished as collapse, they think that a sufficient explanation seems to have been found as long ago as 1870 when Goltz demonstrated that a blow on the exposed mesentery of a suspended frog caused reflex inhibition of the heart through the vagus, and reflex dilatation of the arteries in the splanchnic area, in which the blood tended to collect by gravitation. Two conceptions were current, however, when the Special Investigation Committee began their work: Crile's theory, which has already been mentioned, and the theory in which defect of the volume of blood in effective currency was the central feature. According to this view, the arteries were constricted rather than dilated in shock, the vaso motor centre attempting to compensate for the reduction in blood volume. This defect of volume was partly but apparently not wholly due to loss of plasma from the blood, as indicated by an increase in the corpuscular element. Mann found that the volume of blood recoverable by bleeding the shocked animal out through the large arteries and veins showed a deficiency over and above that accounted for by concentration. The experience of surgeons in France is definitely in favour of the latter conception as correctly describing the condition of shock which they observed, but no adequate theory as to the mode of its causation was available. In several ways the condition is anomalous. It presents the paradox of arterial constriction associated with low blood pressure. The low arterial pressure, again, was associated with concentration of the blood, whereas, normally, the blood takes up fluid from the tissues when the arterial pressure falls. Dale and Laidlaw have been working for some years on the action of a substance called "histamine," which presented very similar anomalies. Injected in certain

doses into the blood stream of the cat, dog or monkey, it caused a typical shock-like failure of the circulation, with arterial constriction, oligæmia, concentration of the blood, failure of cardiac output. Dale and Richards, in 1918, obtained positive evidence that histamine, and, by implication, the group of substances having the same type of action, owe their effect on the circulation to their power of obliterating the normal tone of the capillaries. It will be obvious how this conception of histamine shock, as due to a poisoning of the capillary epithelium, furnishes also an explanation of the passage of plasma from the blood into the tissues, which characterizes the shock produced by sudden injection of a large dose. The effect of histamine in producing shock was found to be enormously increased by previous hæmorrhage and by anaesthetics – ether being that used in the experiments. In commenting on this work Dale says some caution is needed in applying these results to the condition of traumatic shock. Nevertheless, he says, the analogy with the experience of military surgeons can hardly be missed. The special liability to shock of patients whose wounds have been complicated by hæmorrhage is almost a truism. There is a suggestive analogy, again, between the greatly increased liability to histamine shock produced by ether and the experience that patients who were previously, in moderate good condition may pass into a dangerous condition of shock after the administration of anaesthetics of this class. It is fair to assume that one factor in the reduced tolerance to histamine produced by ether is a weakened resistance of the capillary epithelium to the poisonous action which renders it abnormally permeable. The fact that anaesthesia with nitrous oxide and oxygen is practically free from this danger arouses the expectation that it will prove to have little or no sensitizing action for the effect of histamine. As a matter of fact, recent actual experiment has shown this to be the case. With these researches on histamine shock in our minds we shall be able to appreciate more fully the bearing of the following observations made by Cannon and Bayliss.

1. EXPERIMENTAL INJURY OF MUSCLES. – These observers used the flexor group of muscles in the thighs of a cat. The muscles were struck with a hammer so that they were ruptured and severely bruised. The skin was rarely broken by the blows. In a few instances the femur was fractured but this had no consequence different from that resulting from muscle injury alone. At autopsy varying amounts of extravasated blood were found, but in no case was there sufficient bleeding into the wounds to account

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by itself alone for the effects observed. The blood pressure as a rule fell sharply during the traumatization. This fall was followed sometimes by a slight temporary rise. The pressure then continued at a fairly even level for a time, and then a secondary fall occurred. The promptness of the first fall suggested that it was reflex in nature. It occurred, however, after the cord had been cut in the upper lumbar region. It failed to appear if the aorta was clipped above the iliacs during the traumatization, although the nerve conduction would not be impaired during this short anaemia. It may have been due to a combination of dilatation of blood vessels in the injured area, together with some extravasation of blood, with the rapid setting free of a chemical substance acting as a powerful depressant on the blood pressure. The secondary fall may be explained by the continual absorption of the depressant substance and by the establishment of a vicious circle in consequence of low blood pressure. This was associated with a steady fall in the bicarbonate reserve of the blood. A remarkable effect on the respiration usually usually to be seen. The rate increased from about 30 per minute to as much as 180. The depth was shallow.

As the state of the animal became worse the rate decreased steadily until death. The rate of the pulse followed a similar course. *Post-mortem* – The appearance of the muscles reminded the investigators strikingly of the shell wounds seen in France.

2. MUSCLE INJURY IN CASES OF SHOCK IN MAN. – When a large jagged piece of shell, moving at high velocity, breaks the humerus, or the femur, or the ilium, it smashes and tears extensively the overlying muscles. And just as in animal experiments, the injured muscle would produce metabolites which on being absorbed into the blood stream, would indicate their presence by a decrease in blood pressure, with other signs of shock. It is of interest to note that massage of damaged muscles, unless a nerve happens to be pinched, results in a further fall of blood pressure. This phenomenon has probably some significance in relation to the increase of shock when the limb is not adequately supported during transit to the casualty clearing station.

Crile has stated that the Thomas's Splint is one of the most effective means of preventing shock that this war has developed (Research Society Reports, 1918, Medical Bulletin). Bayliss, speaking of the nature of the depressant substance, says lactic acid is not the active agent, nor other acids *qua* acids. Some other substance produced by tissue disintegration must be responsible, and we naturally think of histamine or

related compounds whose action has been investigated by Dale, Laidlaw and Richards.

Cannon, continuing experiments begun with Bayliss, found that the initial fall of pressure observed in the earlier experiments was not often seen when muscles were crushed in pincers. The initial fall, therefore, is not an essential factor in the establishment of shock pressure.

That the "shock" was not due to nervous stimulation was confirmed not only by obtaining, after previous severance of the cord in the lumbar region, the usual fall of pressure from trauma; but also in other cases after severance of the nerves to the limbs at the point of their emergence from the pelvis. Furthermore, the shock was not produced when the nerves were intact but the blood vessels tied. The experiment just mentioned indicated clearly that some substance given off from the injured tissue was the cause of the drop in the pressure, for the permanent fall occurred only when the blood was permitted to flow in and out of the damaged region thirty-three minutes after the traumatism. Further, in a number of instances in which the femur was broken, the fall of pressure was accelerated by agitation of the broken bone in the tissues. This observation is significant in relation to the benefits which are derived from carefully splinting broken limbs during transport.

I might mention here that Crile and Lower (Anoci-association, 1914, p. 73), in their book published before the war, produced evidence which they said proved that the cell changes which they describe as present in shock are not produced by any gaseous change in the blood or any noxious products of metabolism resulting from trauma.

The circulation of two dogs was so anastomosed that the blood streams intermingled freely, and one animal only was traumatised. The functional impairment and the brain cell changes were limited to the animal which had received the injury, although the blood of the traumatised dog flowed freely through the brain of the dog whose body had not been traumatised. Clinical evidence, however, coincides so closely with the experimental evidence adduced by Cannon and Bayliss that the opinion of surgeons is in accord with the views expressed by the latter observers.

In placing before you in a somewhat concentrated form this account of the recent work done in connection with shock, I fear I have run some risk of confusing your minds as to the salient points. In order to emphasise certain conclusions justified by the evidence in hand it may be of help to summarize

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as follows:—

WOUND SHOCK may shortly be defined as that state of depression of the bodily functions which is produced by wounds received in warfare. In civil life, injuries and surgical operations may be taken to represent “wounds.”

SHOCK may be divided into Primary and Secondary Shock.

PRIMARY SHOCK, the nature of which is as yet undetermined, but may be nervous in origin, comes on immediately after injury.

SECONDARY SHOCK, a later development, comes on gradually, the degree depending to some extent on the care of the injured man during transit.

Shock is not due to

EXHAUSTION OF NERVE CENTRES. The theory that it is due to exhaustion of the vasomotor centre must, therefore, be abandoned.

It is not due to

Cardiac Weakness.

It is not due to

Suprarenal Exhaustion.

It is not due to

Acapnia. (See, however, Benjamin Moore, *Lancet*, September 13, 1919.)

It is not due to

Acidosis.

There is evidence in favour of its being produced by absorption of metabolites from injured tissue, especially muscle.

Low BLOOD PRESSURE is one of the outstanding features of shock.

This is due to (1) loss of blood by haemorrhage, or (2) loss of blood by stagnation and concentration in the capillary area.

Contributory factors in the production of Shock are:— deprivation of water, hunger, pain, anxiety, fear, rage, fatigue, insomnia, cold and depressing conditions of all kinds, amongst which may be included anaesthesia.

Bayliss gives the following general conclusion:— In attempting to form some conception of the nature of wound shock, so far as is possible from the data at hand, we arrive at something of this kind. Various causes in combination, some nervous, some chemical, each associated with a reduction of arterial pressure, and all exaggerated by haemorrhage result in a state of collapse whose symptoms seem to be sufficiently accounted for by the effects of a more or less prolonged low blood pressure. Along with haemorrhage the most serious of these collateral causes is the absorption of toxic products from injured tissues, especially muscle. These products

appear to have a dilator action on the capillaries, an action allied to that of histamine; blood is thereby withdrawn from circulation and held up by stasis. If the low blood pressure has lasted for more than a certain time, which depends on the degree of anaemia, the nerve centres become paralysed. In the early stages this paralysis may be recovered from, if means are taken to improve the circulation as quickly as possible. After a longer period of anaemia, no recovery is possible by any method yet devised, and Mott (1918) has found structural changes in the nerve cells at this stage.

TREATMENT OF SHOCK.

PREVENTATIVE — In the injuries of civil life much can be done to prevent the onset of shock by gentle handling, immediate immobilization of injured parts, care in transit, and warmth. Thomas's leg and arm splints should be kept in readiness in all our shipyards, engineering works, foundries, mills, factories, &c., and skilled first aid should be available on the spot. To prevent the onset of shock during and after operative procedures, much also may be done. Before operation we should endeavour to secure sleep for our patient, to allay his pain, anxiety and fear, to promote for him as far as possible a calm and restful state of mind, and to avoid starvation, undue purgation and deprivation of fluids. The recognised methods of preparation for operation are calculated to predispose to shock, and some modification should be adopted. The patient who is violently purged, starved for hours before operation, deprived of fluids, and often strapped to the table before anaesthesia is established has received a poor preparation for the ordeal he is to undergo. Added to this, struggling under the anaesthetic, rough handling of the tissues, “carnivorous” surgery, inefficient haemostasis, undue exposure and prolongation of the operation all tend to initiate a condition of shock which is liable to continue or become aggravated even after the patient returns to bed. The deprivation of fluid is a serious drawback. If shock is due to diminished volume of fluid in circulation, and if the patient has no fluid upon which to draw while his circulation is still capable of taking it up, he is in a condition which predisposes him to shock. The abstraction of fluid by purgation tends to the same result. It is sound practice to give a patient who is to undergo a serious or prolonged operation salines by the rectum or subcutaneously either before or during the operation. By this means we may help to prevent concentration and stagnation of blood in the capillary area.

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On the field of battle it was not always possible to avoid cold, hunger, thirst, fatigue, anxiety and insomnia before the reception of wounds. But the careful splinting of patients by Thomas's splints lessened to an enormous degree the incidence of shock in wounded men. This is attributable to the relief of pain and to the fact that immobilization prevents further damage to muscle, and thus lessens the production of those toxic metabolites which, when absorbed, cause secondary shock. Primary shock, unfortunately, could not be so prevented.

The treatment of the established condition is the same in civil surgery as in the surgery of warfare. It is essential that it should be prompt. If shock be allowed to continue for long, a time comes when methods of treatment which might have availed if used in the early stages will fail because of the damage caused to important organs by insufficient blood supply.

Recognising that toxic products from injured tissues, specially muscle, can produce a state of shock, it is imperative that dead and badly damaged tissue should be removed as early as possible.

The main object in the treatment of shock is to restore the blood pressure and thereby ensure an adequate supply of blood to the various organs of the body.

The most successful agents are *warmth* and *rest*.

There is no need to elaborate the methods by means of which *warmth* may be supplied to the patient. They will suggest themselves to anyone of ordinary intelligence. The warming up of the patient was looked upon in France as the most important measure in the treatment. This was done by spirit lamps, stoves, hot bottles, hot drinks and hot fluids per rectum.

Rest was produced by moderate doses of morphia and by careful splinting and gentle handling of the patient during transit. Large doses of morphia were found to be injurious but moderate doses composed the patient's mind and allayed anxiety (especially during shelling on the way down), relaxed his muscles and relieved pain.

The *administration* of *stimulants* found very little favour, and was soon abandoned for more efficient methods. Civil surgeons will almost universally endorse this experience.

Strychnin has long been abandoned in the treatment of shock. Crile and Lower (loc. cit.) said, in 1914: "A research was made upon the effect of strychnin, and it was discovered that this stimulating drug not only did not improve the condition of the

animal after shock, but actually made it worse. At the end of that research, therefore, we concluded that shock could be produced by strychnin alone, and that it was as logical to treat traumatic shock by strychnin as it would be to treat strychnin shock with trauma."

Vasoconstrictors like adrenalin and pituitrin were extensively used, but failed to establish a reputation in treatment of shock. As Bayliss says: "The end to be attained is not the height of the blood pressure in itself, but a better supply of blood to the tissues. The increase of peripheral resistance by drugs which constrict arterioles is bad in shock, as, although the driving pressure in the main arteries rises, the effect on the supply to an organ is more or less counteracted by the constriction of the arterioles in that organ."

Glucose Solutions have been recommended as an addition to fluids for intravenous injection on the ground that there may be carbohydrate starvation in wound shock. As has been pointed out, the blood sugar has been found above the normal in shock. Bayliss sees no object in adding glucose to solutions for intravenous injections in shock (loc. cit.).

TREATMENT DIRECTED TOWARDS INCREASING THE BLOOD VOLUME.

If we accept the theory that the low blood pressure of shock is not due to heart failure nor to exhaustion of the vasomotor centre, but to diminished blood in currency, our endeavours will naturally be directed to measures which aim at increase of the volume of blood in circulation.

FLUIDS BY MOUTH AND RECTUM. — Before resorting to more heroic measures, a trial should be made of administering fluids by mouth or by rectum. In the milder forms of shock, in which grave failure of the circulation has not taken place, satisfactory results may thus be obtained.

SUBCUTANEOUS SALINE. — The administration of saline by subcutaneous injection in the graver degrees of shock proved a disappointing measure in France. During the later stages of the war one very seldom saw this method employed.

INTRAVENOUS INJECTIONS. — Those who are interested in the subject of intravenous injections in shock should read Bayliss' book on the subject. It is full of valuable information. I am indebted for much of the substance of this address to his admirable work. As chairman of the Special Investigation Committee he has, with his co-workers, done much to place the treatment of shock on a sound basis.

1. SIMPLE ISOTONIC SALT SOLUTIONS. — For many

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years past it has been the custom to treat shock by the injection of isotonic salt solutions such as Ringer's solution. In the earlier days of the war this practice was followed, but the results were very disappointing. It was found that such solutions were useless in restoring a low blood pressure in wounded men. Blood pressure readings were taken, among others, by Fraser and Cowell, whose verdict was as follows:—"We have been greatly disappointed with the results obtained, and blood pressure readings confirm the clinical disappointment. After injection of these fluids there is a temporary rise followed by a fall to a point sometimes below that registered before injection. Within half-an-hour or so the blood pressure falls to, or below, its original level." It would seem as if the fluid ran out of the vessels almost as quickly as it was introduced. The fluid leaves the capillaries at such a rate that oedema of the tissues may result. The risk of producing oedema of the lung by copious intravenous saline injections is well recognised. Bayliss says:—"The final result of injecting saline solutions is usually a fall of blood pressure below the value at the moment of injection. This is probably due to the progressive effect of the deficient supply of blood to the brain and other tissues."

2. HYPERTONIC SALINE. — On account of the success of hypertonic solutions in cholera, in which, as is well known, concentration of blood occurs, it was thought that these solutions might be of use in shock. They had a very extensive trial in France, but it was found that their effect was hardly less transient than that of isotonic saline. Hypertonic saline soon followed isotonic saline into oblivion.

3. CALCIUM SALTS. — Calcium has been supposed to decrease the permeability of the blood vessels. If this were so, escape of fluid from the vessels might be prevented. Bayliss, to test this, compared the rate of escape from the blood of ordinary Ringer's solution with that of a similar solution to which excess of calcium chloride had been added. He was unable to detect any difference. He does not consider that the addition of calcium salts to a solution for intravenous injection has any beneficial effect whatever.

4. ALKALINE INJECTIONS (BICARBONATE OF SODA). — Bicarbonate solutions were tried on the assumption that acidosis was a potent factor in the production or maintenance or wound shock. A solution of 4% was used. Bayliss says that after careful examination of the evidence he has been compelled to come to the conclusion that the importance of this state has been greatly exaggerated, not only in wound shock, but in general. The use of bicarbonate solutions is

unnecessary. Haldane, in a recent paper (*B.M.J.*, July 19th, 1919), is more emphatic still. He says:—"It seems to me a gross mistake to treat the supposed acidosis of anoxaemia due to gas poisoning, 'shock,' and other conditions, by the administration of alkalis. The body is calling for both oxygen and acid."

Benjamin Moore (*loc. cit.*) says:—"It is entirely wrong treatment to administer alkalies. What the organism requires is oxygen, carbon dioxide and warmth, until oxidation in the tissues begins to approach its normal level. The organism has breathed off in excess its balancing carbonic acid in its attempt to get enough oxygen; it has thereby alkalisied all its cells, and this alkalinity by its action on the state of aggregation of the molecules of the bioplasm has disturbed the functions of heart and nerve centres."

5. INJECTIONS OF SOLUTIONS WHICH DO NOT LEAVE THE BLOOD VESSELS. — When it was found that solutions containing salts or other crystalloids had only the most temporary effect, physiologists set themselves to find substances which would remedy the defects of these solutions. These defects are

1. Their viscosity is too low.
2. They contain no colloid with an osmotic pressure. Of these the second is the more important.

Proteins suggest themselves, but Bayliss points out that they are unsuitable (1) because foreign proteins affect the kidney so that they appear in the urine and (2) these produce anaphylactic sensibility so that if at any future time another similar intravenous injection were made severe anaphylactic shock would almost certainly occur.

Two substances remain: gelatin and gum. According to Bayliss these substances in concentration which have the correct osmotic pressure have a viscosity almost identical with that of blood. These are 6% for gelatin and 7% for gum arabic when the osmotic pressure is measured in the presence of the salts of Ringer's solution. Both these substances were subjected to extensive trial in the laboratory and as the result of experiment Bayliss decided in favour of gum acacia. He gives the following main objections to gelatin, which might seem the more natural as its precursor collagen is a constituent of animal tissues.

(1) Owing to the source of most of the best commercial gelatin, namely, calves' feet, it is liable to contain the spores of tetanus bacilli which are difficult to kill.

(2) Gelatin causes intravascular clotting and has been used in the treatment of aneurism for this property.

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(3) In cold weather the setting to jelly in the tubes and cannulae, if a block occurs, may become inconvenient.

Fortunately, gelatin has no properties of importance for our purpose that gum does not possess. The osmotic pressure of any of the colloids mentioned is not in itself high enough to prevent haemolysis of red corpuscles. The colloids must be dissolved in 0.9 per cent. sodium chloride.

BAYLISS'S SOLUTION. — A solution of 6 to 7 per cent, in 0.9 per cent, sodium chloride has the viscosity of blood and the osmotic pressure of its colloids. Hence such solutions do not leave the blood vessels. Gum is chemically inert and quite innocuous. Dale tested it in rabbits, and Bayliss in cats. No ill effects were noticed. Extensive trials were made in man by Drummond and Taylor and many other surgeons in France. In a few cases ill effects were attributed to its use, but the balance of opinion was that it was harmless. The solutions are easily sterilised without loss of viscosity. The temperature of a steam sterilizer is probably sufficient, but it is safer to use an autoclave.

The solutions have no haemolytic or agglutinating properties when tested with human blood.

The gum used was that sold as "Turkey elect."

Mucilage of acacia of the British Pharmacopoeia may be used if properly diluted and made to contain 0.9 per cent, of sodium chloride.

Gum arabic is a mixture of polymerised anhydrides of galactose and of a pentose sugar (arabinose) in varying proportions.

FATE OF GUM IN THE ORGANISM. — Dr. Bayliss found the blood of a cat to give a positive reaction for pentoses twenty-four hours after an injection, but it was absent three weeks later. If it escapes by the urine it is very slow. It has been found after large quantities of gum had been injected. It is possible that gum or its hydrolytic products may be excreted into the intestine and utilised as food. It disappears from the circulation sooner or later, but it is not certain how this takes place.

The solutions may be made with freshly distilled water or with ordinary tap water.

They must be filtered through a rather coarse medium, such as flannel or Chardin paper, to remove bits of wood and such like.

A 6 per cent. solution is strong enough for routine use. The solutions may be injected through a sharp hollow needle into a suitable vein, or the latter may be exposed and the injection made by a cannula.

The solution ought to be run in at the rate of

one pint in fifteen minutes. The amount used will depend on the effect on the blood pressure, and the condition of the patient.

One pint may be used to begin with, to be followed by a second pint half-an-hour to an hour later.

These details are given by Bayliss in his book.

The blood pressure will be found to rise, and in favourable cases the rise is lasting. The solution must be used before a low blood pressure has had time to cause a loss of excitability of the bulbar centres.

GUM SALINE SOLUTION IN THE TREATMENT OF HAEMORRHAGE.

In cases of shock it is often difficult to determine to what degree the condition is due to haemorrhage. Most wounded men requiring resuscitation have lost a considerable amount of blood. As we have seen, shock is due to a combination of several factors, any one of which by itself might not produce any serious effects. The loss of a pint or more blood by a healthy man would not give rise to any untoward symptoms, but the same amount of loss in a man severely wounded or badly chilled might easily be fatal. Even a few ounces loss in a cold or badly wounded man, or in one who has had prolonged sepsis, might turn the scale against him. The researches of Bayliss, confirmed by the clinical experience of many surgeons in France, show that even in cases of shock associated with severe haemorrhage gum saline solution is capable of restoring the patient Oswald H. Robertson (Reports M.R.C., No. 4) quotes Rous and Wilson as having brought forward some recent data on fluid substitutes for blood. They were able to show that after rabbits had been bled to such an extent that they had lost three-quarters of their total blood volume, the blood pressure could be restored to normal by the immediate injection of an equal quantity of blood plasma freed from red corpuscles. The improvident was maintained and the animals remained in good condition, though anaemic. Six per cent. gum acacia solution worked as well in these experiments as blood plasma. Robertson, commenting on these experiments and their relation to wounded men, makes the obvious criticism that the addition of a large quantity of new blood in such a case would seem to meet the demands of the situation more effectively. Bayliss, referring to the wounded man, says it is possible that there may be cases in which gum is less effective than transfusion of blood or injection of preserved blood corpuscles; but so far as

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definite evidence goes the difference does not seem to amount to much. If, he says, there has been a very large loss of blood it is reasonable to make use of blood itself, but it may be effectively supplemented by gum and economy in blood attained. A gum injection before, during, or after an operation is frequently valuable if the blood pressure is low or tends to fall.

BLOOD TRANSFUSION. – During the latter half of the war blood transfusion was extensively used in France for haemorrhage and shock. We were at first slow to employ this method of treatment, and it was not till 1916 that an extensive use was made of it. The advent of the Americans in 1917 gave a fillip to it, and soon it had a great vogue. In some cases it is possible that less heroic measures might have sufficed. Nevertheless, the war has served to place this life-saving measure on a proper basis.

If the use of gum saline justifies the hopes raised for it by recent research and clinical experience in France, blood will be reserved for cases of severe haemorrhage or grave anaemia. Accepting the theory that in shock the lost blood is, so to speak, sidetracked and out of currency for the time being, the reasonable line of treatment would be to keep up the blood pressure by some substance such as gum saline solution until the circulation is sufficiently re-established to sweep the stagnant blood once more into the main current. Extensive loss of blood cannot thus be dealt with, and transfusion will remain our sheet-anchor in such cases.

In our earlier work in France we failed to attach sufficient importance to the possibility of the blood of the donor and the blood of the receiver being incompatible, and several untoward results followed. The procedure for the selection of suitable donors has been much simplified by the work of Moss, who found that all individuals fall into four groups as regards the haemolytic and agglutinative properties of their blood.

Moss's CLASSIFICATION.

Cells	I.	II.	III.	IV.	Percentage
Serum I.	–	O	O	O	8%
II.	X	–	X	O	40%
III.	X	X	–	O	10%
IV.	X	X	X	–	42%

X means agglutination. Agglutination always precedes haemolysis.

Roger Lee (B.M.J., Nov. 24, 1917,) showed that the only practical consideration is that the recipient should not agglutinate the red corpuscles of the

donor. The dilution which the donor's serum undergoes when introduced into recipient's blood diminishes its agglutinative power, which may then be neglected. In addition to this, the red blood corpuscles of the recipient are protected by their own plasma.

Individuals of No. I. blood group can receive from all four groups, but can only give blood to individuals of group I. They are called universal receivers. The members of group IV. can give blood to those of all four groups but can only themselves receive from those of group IV. They are therefore, called universal donors. Individuals of group II. can only receive from those of group II. and IV., and individuals of group III. from those of III. and IV.

It was the custom in France to group prospective donors and a number of lightly-wounded men, or men with some such disability as hernia, sprains, etc., who, on being tested belonged to group IV., were kept in readiness for emergencies. It was necessary, of course, to exclude donors who gave a history of syphilis, malaria, or other communicable disease. In civil practice there is usually time to test the serum of the recipient against the corpuscles of the donor, but it is not practicable to have in readiness a number of individuals of group IV.

Time will not permit of the discussion in detail of all the methods of blood transfusion in general use at the present time. A very short summary will suffice. I will confine myself to those used during the war by British and American surgeons in France.

I. DIRECT METHOD. – This is a method in which I was myself much interested in 1916. I was associated with Capt. Bazett and Prof. Dreyer, both of Oxford University, in working out the details of an apparatus by means of which blood could be transfused directly from the radial or ulnar artery of the donor to any suitable vein of the recipient. Silver cannulae, connected by thin rubber tubing, were used to effect a communication between the donor's artery and the receiver's vein. The apparatus was coated with wax inside and out, and was kept sterilised ready for use (*Lancet*, May 12, 1917). An objection to this method was the difficulty of estimating how much blood passed from donor to receiver. We judged by the appearance of the recipient and donor when sufficient had been received or lost.

II. INDIRECT METHODS, (a) *Paraffined tubes*. – Of these the best known are those of Vincent and Kimpton. These tubes have a capacity of about 400 to 500 c.c., and terminate in a drawn-out cannula of a size suitable for introduction into a vein. They are coated on the inside with hard paraffin, and are kept

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sterilised ready for use. The upper end is fitted with a rubber stopper, and an air tube leads off from near the top. The air tube may be fitted with a reversible rubber bulb for applying suction or for pumping out the blood, as may be required. The blood is received into the paraffined tube from a vein of the donor. As soon as sufficient has been drawn, the cannula end is removed from the vein of the donor and placed in that of the receiver, and the contents of the tube allowed to flow, or the flow may be aided by compressing the bulb attached to the air tube at the top of the apparatus. Several tubes should be at hand, and in septic cases the same tube should not be reintroduced into the vein of the donor after it has been in the vein of the receiver. The blood should not be introduced too quickly. If signs of distress occur the rate may be retarded until these pass off.

The above method is that now usually employed for transfusing unaltered blood. It is simple and effective. The direct method requires a little more surgical skill and greater finesse in the technique.

(b) *Syringe Methods.* – Bruce Robertson (B. M. J., July 8th, 1916) was the chief exponent of this method in France. He used Record syringes of 20 c.c. capacity and two needle cannulae specially devised by Lindemann. Six syringes are required. The cannulae and syringes are lubricated with liquid paraffin to prevent clotting. The cannulae are introduced into the selected veins of donor and receiver respectively, and are allowed to remain until the operation is concluded, unless clotting takes place. Used syringes are washed out with saline and again lubricated with liquid paraffin, so as to be ready for further use during the same transfusion. The method has proved useful in trained hands, but is not so generally practicable as the last mentioned.

(c) *Citrated Blood Transfusion.* – When sodium citrate is added to blood in sufficient quantity clotting will not take place. Blood can, therefore, be received into a solution of sodium citrate of suitable strength and without undue haste transferred to a recipient. The very simplicity of the method is perhaps one of its drawbacks. If care is not used, and too much time is consumed over the procedure certain changes will take place in the blood which may lead to reactions in the receiver. The blood must be quickly received and mixed with the citrate solution before clotting has had time to take place. Oswald H. Robertson (Reports M.R.C., No. 4) has devised a simple apparatus which answers all purposes. The blood is received into a solution of sodium citrate, 3.8 per cent. which is isotonic. Sharp needles are used to enter the veins of donor and receiver, and these are connected with the

glass jar containing the solution by suitable tubes. An indiarubber bulb is connected with a third tube which just projects into the jar through the rubber stopper. The bulb is reversible so as to exert negative or positive pressure on the contents of the glass jar. Fleming and Porteous (*Lancet*, June 7th, 1919), tested the recipient's blood before, immediately after, and again, twenty-four hours after transfusion with citrated blood, and found that the coagulation time was not altered. This meets the objection that the citrate used might have some effect in predisposing to haemorrhage. The citrate method of blood transfusion is probably the most fool proof, and, no doubt, will be extensively used in civil practice.

(d) *Preserved Blood.* – A good deal of interest has been evinced, especially among the laity, in what has been called "pickled" blood. The method is based on the work of Rous and Turner (*Jrn. Exp. Med. N.Y.*, 1916), and was first used in France by Oswald H. Robertson of the United States Army. Rous and Turner found that blood when taken into a mixture of dextrose and sodium citrate solutions and kept in the cold could be preserved for several weeks. The red cells remained intact and retained their vitality for a long period. Robertson used blood from donors of No. IV.

Group. The blood was received into a bottle containing a mixture of isotonic dextrose solution (5.4 per cent.) and isotonic citrate solution (3.8 per cent.). It was then intimately mixed with these solutions by a rotatory motion of the bottle. The latter was plugged and placed in an ice chest so as to allow the red blood cells to sediment. At the end of four to five days the blood was available for use. When it was desired to use the blood the supernatant fluid was drawn off. The blood was brought at least to its original volume by a diluting agent, and for this purpose a 2.5 per cent. solution of gelatin in normal saline was used. The mixture was filtered carefully so as to avoid damage to the red cells, and the bottle containing it heated by placing it in water at a temperature of 106°-107°F. The blood was then transfused by means of an apparatus similar to that used for citrated blood.

Robertson found that preserved blood could be transported for considerable distances without injury to the red cells. It could be kept for several weeks. In addition to Robertson a large number of workers in France have used preserved blood cell transfusions with results as good, apparently, as those obtained by other methods of transfusion.