GAS GANGRENE AS SEEN AT THE CASUALTY CLEARING STATIONS.

BY

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GAS GANGRENE is still a very striking feature in the surgery of this war. It has from its very frequency ceased to arouse very much wonder, and is, I think, a little in danger of being accepted as a necessary evil, and treated by routine measures.

Two very interesting papers have recently appeared by d'Este Emery and Kenneth Taylor.

d'Este Emery's views seem to be as follows: The toxin kills the leucocytes which are the natural protection of the body. To have a sufficient supply of these it is necessary that the circulation be intact. Therefore, tissue devitalized by trauma, by constriction of the limb, or by actual damage, will favour the disease. Why, however, does the disease not stop when healthy tissue is reached? The explanation lies in the fact that the toxin, when present in large amount, inhibits emigration and kills the leucocytes. If there is no free escape the toxin accumulates to such an extent that it soaks through into the healthy tissue and kills the defensive leucocyte, and so the gangrene spreads.

Taylor's view may be summarized as follows: The mechanical action of the pressure produced by the gas is usually, if not always, the most important part of the infection. It brings about the death of the tissues from (a) the resulting anaemia, (b) the actual mechanical fragmentation of the muscle substance, (c) the mechanical scattering of the infection. He shows in the earlier part of his paper that the experimental injection of the exotoxin produces softening of muscle and parenchymatous degeneration of the liver cells. His experiments *in vitro* show the same effect on pieces of muscle. He suggests that the rapid systemic intoxication is produced by the breaking down of the muscle substance, as injection of the exotoxin does not produce an effect comparable to that seen in the clinical course of the disease.

If these two theories of the disease are compared, it will be found that they differ mainly in the part played by the gas. Taylor thinks that it plays an important part; d'Este Emery denies this and believes that the bacteria toxins are the important factor. Taylor believes that the disease is mainly one of the muscles; d'Este Emery thinks that this is a mistake, and that it is only the fact of the bacteria being able to produce gas by the aid of the muscle sugar that leads to this assumption.

These views reflect two somewhat different conceptions of the disease, but it seems probable that both observers have some right on their sides.

I have thought that a statement of some of the features of the disease as it presents itself to me may be of interest. I will commence by quoting some cases that illustrate some points that I want to emphasize.

CASE I.

A soldier (under the care of Captain Anderson) was hit by a shell fragment which made a large wound involving the adductors and hamstrings and smashing the femur in the upper third.

The superficial and deep femorals were injured and had to be ligated. The wound, which was a large open one, was cleansed and drained in the ordinary way. The limb was fixed in a Thomas splint.

Within thirty-six hours the leg was dead but non-tympanic, the thigh was tympanic over all its circumference. It was still warm, as such infected limbs often are, about the seat of the actual injury. No crepitation could be obtained by palpation, but with a stethoscope distinct crackling could be detected on passive movement of the limb and on relaxing or increasing deep pressure; the stethoscope was applied over the vastus externus. The limb was removed through the fracture, but the patient died quietly in his sleep some twelve hours later.

Examination of the Ablated Limb.

The subcutaneous tissue appeared normally yellow in its fatty layer, and no gas could be demonstrated by pressure. The deeper layer immediately over the fascia lata was oedematous but not discoloured. On incising the fascia lata, which appeared normal, a similar oedematous condition of the areolar tissue over the vastus externus was observed. No gas could be demonstrated here. The vastus externus was normal in colour, and contracted in a lively manner when pinched or when cut with a knife.

The adductor longus was of a dirty brick-red colour, more opaque and less translucent than healthy muscle. It was non-contractile, and gave no sign of life when pinched. It was non-crepitant to the squeezing finger and cutting scalpel. The hamstring muscles were the same colour, non-contractile, and frankly gaseous and crepitant.

The areolar tissue was oedematous and infiltrated with blood, but not black.

Cultures were made from portions of the vastus externus and adductor longus by taking pieces from the heart of the muscle after searing the surface. Both, as reported by Captain Emrys Roberts, showed anaerobic bacteria.

Pieces of muscle taken from vastus externus, adductor longus and biceps were examined microscopically by Captain McNee, Captain E. Roberts, and Lieutenant Dunn.

1. *The Vastus Externus*.—The fibres were normal, and no bacteria were to be seen in the tissues.

2. *The Adductor Longus*.—The fibres had lost their transverse striation. The muscle substance was broken up in transverse plates or presented longitudinal markings, as if painted with a coarse brush. Between the fibres, which were obviously separated from one another, were bacteria in varying quantities in the interfibrillar spaces. There were some polymorphonuclear leucocytes.

3. *The Biceps*.—The appearances were similar to those seen in the adductor longus, but the separation of the fibres was more marked and there were more bacilli and leucocytes.

CASE II.

A man under the care of Major Cuff, who had been hit with a shell fragment in the middle of the leg. The anterior tibial was divided and was tied. (The man was also wounded in the liver and coeliotomy performed to stop the bleeding.)

About forty-eight hours after the receipt of the injury the leg became tympanic and died. The skin of the thigh was crepitant and the gas in the crepitant area could be displaced by pressure in the same manner that surgical emphysema can be.

An incision in the crepitant area showed normal looking fat without oedema, and the knife drawn over the deep fascia gave the typical hollow sound. The subfascia connective tissue contained gas. A culture taken from this locality remained sterile.

The leg was taken off through the knee. The thigh never gave any sign of infection, although it was crepitant. A good recovery followed.

CASE III.

Pte. B. was wounded in the thigh with a shell fragment. The femoral was exposed in the wound. The thigh became tympanic on the fourth day, and was treated with incisions, which arrested the gangrene. The foot died in the following night, and the leg was tympanic in the morning. Exploration showed that the femoral had become thrombosed. The leg was amputated at the seat of election.

Examination of the Ablated Part.

It was drummy, but not crepitant. The anterior tibial muscle group was of a dirty red colour and non-contractile. The posterior tibial muscle group was brick-red, but non-crepitant. The calf group was contractile and purple. The fascial planes between the muscles appeared normal. The subcutaneous tissue in the cut surface of the amputated leg was oedematous and greenish, but contained no gas.

Cultures from the subcutaneous tissue and the purple contractile calf groups both gave *B. aerogenes capsulatus*.

CASE IV.

A man was wounded on the flexor aspect of the forearm. He exhibited all the signs of "gas infection." The hand was warm but anaesthetic. Incisions through the deep fascia arrested the disease, but the superficial flexors sloughed; otherwise the man made a good recovery.

CASE V.

A man was shot through the flexor aspect of the forearm. The bones were intact. He was admitted with a tense swollen forearm. The hand was cold and insensible and showed a line across the back of the hand suggesting that the outer half of the hand was about to mortify.

Incisions into the flexor aspect of the forearm and into the thenar eminences showed the superficial muscles and those of the thenar eminences in a state of "red death." Next day the hand was warm and the circulation returning. The improvement was of short duration and the hand and flexor muscles were ablated. The extensor muscles were uninfected. The man made a good recovery.

CASE VI.

A man was shot through the middle of the humeral biceps. He developed all the classical signs of gas gangrene. The medical officer in charge thought that amputation would be necessary. The surgical specialist (Captain Neligan), however, made a long incision over the muscle, and found that the muscle was dead and gaseous in the middle at the site of the wound. The two ends of the muscle were still normal in colour and contractile. The whole muscle was ablated.

There was some doubt as to the state of the triceps, but an incision through the fascia showed that this muscle was healthy and contractile.

CASE VII.

A patient hit in the buttock suddenly developed gas gangrene in this region. It was freely incised. Before death, which occurred within a few hours, the upper arm became crepitant to the finger, though there was no wound at all in this region.

CASE VIII.

The case quoted by Captains Mullally and McNee in the *Lancet*, April 1st, 1916, where gas gangrene appeared at the sites of subcutaneous injections.

CASE IX.

A case quoted by Lieutenant-Colonel Gordon Watson where the opposite thigh to that injured became distended with gas shortly before death.

CASE X.

Many cases in which, after amputation, one muscle is found in the stump dead and gaseous and separable from the others if the insertion or origin is divided.

CASE XI.

A man was shot through the popliteal space. The leg was cold and dead, but was not drummy or crepitant. Amputation through the knee was performed. The popliteal space was full of blood. There was a large hole in the artery.

Dissection of the ablated leg showed that the muscles were non-contractile but purple in appearance.

Microscopical sections showed the normal striation of the fibres, which were not separated from one another.

My own impressions of the disease as seen at the front and gathered from the cases above quoted, and many others, may be briefly stated as follows:

1.

It is rare to meet gas gangrene without a muscle injury.

2.

It is chiefly a disease of the muscles, and is rarely dangerous unless muscle is involved.

It occurs, it is true, in retroperitoneal haematoma, but here there are wounded muscles in which it can at first obtain a lodgement, and there is, in addition, the blood clot and the tissues devitalized by the extravasated blood in which it can spread.

It has been said that one of the most remarkable features about the *B. aerogenes capsulatus* is the fact that a bacillus usually but little pathogenic becomes suddenly extremely virulent. Barger and Dale have shown that an allied bacillus—the vibrio-septique—becomes at once extremely toxic when grown on muscle.

Cannot, then, the difference in the toxicity of the *B. aerogenes capsulatus* depend on the nature of the medium in which it is implanted? Will not this account for the difference in the clinical course of many gunshot injuries? If inoculated into subcutaneous or areolar tissue it finds difficulty in establishing itself, but if it gains entrance to muscle it finds itself in favourable surroundings and is enabled at once to produce such toxins as will quickly destroy its host.

3.

The lesion in its early stages may be described as a longitudinal one, running up and down the wounded muscles from the seat of the lesion (see Case vi). Muscles and groups of muscles are involved while others escape.

4.

It is rare to find all the muscles of a segment of a limb involved, save in a segment distal to one in which the main blood supply has been cut off. Thus the whole leg dies and becomes gaseous when the femoral artery has been blocked in the thigh.

5.

The muscles affected are in the first instance the wounded ones. If the pressure caused by the disease is relieved, the gangrene will most probably be confined to these muscles, but if the pressure is not relieved the other muscles may so have their blood supply checked as to fall victims to the infection.

6.

Muscles contained in rigid compartments, such as the anterior tibial group, are especially prone to die if wounded.

7.

There is but little tendency for the infection to pass from one muscle to another. This is well shown in

amputation stumps, where one muscle dies and becomes gaseous, while the rest of the cut muscles remain healthy.

8.

The infection is further advanced in the muscles than in the intermuscular areolar planes.

In legs amputated for gas gangrene the only visible abnormality may be the brick-red colour of the dead muscles. So normal-looking may be the appearance of the cut surface of the limb that I have known people unacquainted with the peculiarities of the disease express unbelief in the infection of the limb until the bacilli were demonstrated in the non-contractile muscle. Again, the internal appearance of a limb affected with gas gangrene is quite different from that of such a disease as cellulitis of the neck, in which the areolar tissue is infected and the muscles normal.

9.

The muscles become resonant from the presence of gas long before they become crepitant to the finger, though this phenomenon may be perceptible at an early date by means of the stethoscope. This was pointed out to me by Captain Maybury.

10.

The presence of gaseous crepitation does not necessarily mean microbic infection (see Case ii).

11.

Crepitation is usually a comparatively late phenomenon, and is due to the escape of gas into the areolar and subcutaneous tissue.

12.

In an infected limb a vascular lesion will be followed by the death of the muscle or muscle group, which death would not have followed in an uninfected limb. It is believed that the pressure produced by the gas so raises the tension in the limb as to finally arrest the circulation.

13.

In an infected limb there are several conditions of the muscles:

(a) Normal purple red contractile muscle, which may or may not be infected, as judged by cultural experiments.

(b) Dead, non-contractile, non-crepitant muscle, which has a peculiar red colour and is less translucent than normal muscle.

(c) Dead, non-contractile, crepitant muscle, which has the same appearance as the last.

(d) Brown, black, or diffuent muscle.

(Muscle dead from the cutting off of the blood supply is a purplish brown and its naked-eye appearance quite different from (b) and (c).)

14.

The microscopic appearances of muscle dead from cutting off its blood supply are different from those of a muscle dead from infection. The striation is present in the former and absent in the latter.

15.

The bacteria are between the muscle fibres and not in them.

16.

Microscopical examination suggests that the gas may find its way between the muscle fibres in front of the bacterial invasion.

17.

In dead infected muscles the fibres are separated from one another. This separation is more marked in muscles that are crepitant than in those that have not yet reached that stage.

Death of a Muscle with an Intact Blood Supply.

A muscle deprived of its usual blood supply, or with a restricted supply, falls an easy prey to the gas bacillus. Muscles with an intact blood supply seem liable to die, but the method of their death is not quite clear. Cultures show that normal-looking and contractile muscle may be infected. What proportion of such infected muscles would ultimately die if left untreated is not known. Nor is the way in which the infection spreads known.

Case VI shows that the infection and death spreads in both directions from the seat of injury, for in this case the centre of the muscle on both sides of the wound was dead, but the extremities were still living and contractile. Small bomb or shell wounds which would not affect the main blood supply may be followed by gas gangrene; and experience shows that if left untreated such infection will spread rapidly and compass the death of the wounded muscle. Incisions will save this spread of the gangrene. It seems, therefore, that pressure is a great factor, but whether it wholly acts by cutting off the blood supply or by allowing the gas to penetrate the muscle and produce an anaemia of the fibres, or by favouring the penetration of the yet living muscle by the toxins derived directly or indirectly from the bacilli, is yet undetermined.

In the first instance the infection is possibly due to the fact that the local damage affords a medium in which the bacteria can grow and multiply. The extension is brought about in two ways.

1. *The Toxins Produced by the Bacteria.*

One of the most striking things seen in microscopic sections of muscles dead of gas infection is the loss of striation and the breaking up of the muscle-fibre substance. These appearances are quite different from those seen in uninfected dead muscle. There must be some cause for this difference.

In some microscopic sections of an infected muscle all stages of the change can be seen from normal striated fibres to the totally disorganized. The appearances are seen in sections in which the bacteria are scanty or absent and in which the amount of the separation of the fibres—presumably by gas—varies in great degree.

The change in the muscle fibre may, therefore, be due either to some toxin produced directly or indirectly by the bacteria.

Both d'Este Emery and Taylor seem disposed to credit some action to this source. Taylor is of opinion that the bacterial exotoxin is not powerful enough to account for the constitutional symptoms, and suggests that these are due to toxin produced by disintegration of the muscle substance by the bacterial toxin. He does not suggest it, but it seems possible that the toxic muscle substance produced in the traumatized portion of the muscle may be carried into the more distal parts of the muscle and cause its death.

2. *Part Played by the Gas.*

This may be divided into two categories:

- (a) The part it plays within the limb.
- (b) The part it plays within the muscle.

One of the most striking and most important signs of gas infection is the tense and tympanitic state of the limb and the rapidity with which this condition is reached.

There is nothing but the rapid evolution of gas that could bring about such a condition in so short a time. An incision into such a limb shows that the increased tension is caused by the swelling of the muscle due to the gas within it. There may be gas in the areolar planes, but this is not usually the case to any great degree. It has been pointed out that a muscle may be dead and full of gas and yet not crepitant to the finger; if, therefore, crepitation is taken as the criterion of gas infection, a mistake may be made and a gaseous muscle passed as non-gaseous.

When the gas is present in the areolar tissue it follows the path of least resistance and finds its way along the intermuscular planes and around the vessels. It will disseminate itself into the subcutaneous tissue a considerable way from the wound.

It has been stated above that in a segment of a limb that has been wounded it is only the wounded muscles that yield to the infection in the first instance. The injured muscles will induce a deleterious effect on the uninjured one by raising the intrafascial pressure of the limb and thus causing pressure on the blood vessels and consequent checking of the circulation. Even if the areolar planes are grossly infected, they cannot influence the pressure in the limb, since the muscle sugar is necessary for the production of gas.

Taylor is of opinion that the pressure of gas within the muscle itself leads to disintegration of the muscle fibre

substance. The pressure produced by the gas will in addition tend to drive the toxins into the circulation and thus be a factor in the production of the systemic poisoning and lowering of the resistive power of the body.

d'Este Emery, in combating Taylor's contention that the gas production plays an important part in the disease, says: "Gangrene may occur and the patient may die without the formation of any gas in the tissues." This statement is opposed to what is seen of the disease in the casualty clearing stations. The great sign on which reliance is placed is the swollen and tympanitic condition of the limb, usually preceded by constitutional symptoms. What can cause such sudden swelling unless it be gas? In such cases there is no crackling or crepitation to the finger palpating the skin. An examination of the muscles that have suffered the "red death" may even yet reveal no crepitation, but microscopic sections of such non-crepitant muscles show the fibres separated from one another, and the spaces often free from leucocytes and bacteria.

Men who die of the effects of gas gangrene in the casualty clearing stations die before there is any marked suppuration. It may be that the suppuration and involvement of the fascial planes are seen after the patients have left the front, being a later manifestation of the disease.

The Path of the Infection when an Unwounded Segment of a Limb from which the Blood Supply has been cut off becomes Infected.

When a muscle has been wounded the method of infection is obvious. The path of the infection is not so obvious when there is no wound. For instance, the leg below the knee will become infected when the femoral vessels are occluded, although there is no wound in the leg proper. There seem to be three possible avenues: (1) Direct extension; (2) extension along the blood vessels; (3) systemic infection.

1. *Direct Extension.*—The anatomical appearances do not favour this route. The only muscular tissue that crosses the knee is contained in the two heads of the gastrocnemius. It has been pointed out above that direct extension to an intact muscle is rare, and as a matter of fact the soleus and gastrocnemius are the last muscles to become infected and die. The appearance of the fascial planes does not suggest them as the path.

2. *Extension along the Blood Vessels.*—Possibly the infection travels inside the vessels, just before or after the circulation has ceased, the open ends of the vessels on the infected wound being the starting point. H. Dunn has found the *aërogenes* bacilli in the clotted vessels in the brain after a wound of the carotid artery.

3. *Systemic Infection.*—Mullally and McNee have pointed out that gas formation may take place at the sites of subcutaneous and saline injections, the slight trauma being sufficient to afford a resting place for the bacteria circulating in the blood. Case VII and Case IX, above quoted, point in the same direction. It is therefore possible that bacteria in the blood are implanted in the distal segment of the limb before the circulation is quite brought to a close. It may be that they first gain a footing in the muscles mostly affected by the cutting off of the main blood supply, and by the production of gas within them finally and completely arrest the blood stream.

CONCLUSIONS AS REGARDS TREATMENT.

(a)

The circulation should be helped in every way. It is unnecessary to insist on the bad effects of tight bandages and tourniquets—they are appreciated now by all. Tension in a limb due to blood effusion should be relieved, no matter how small the wound. Bleeding from a small branch may produce pressure on the main vessel, and even without this may cause such pressure as to embarrass the circulation. In cases where haemorrhage into a limb is continuing, there is no doubt as to the advisability of finding the bleeding point. If the artery is a main one an attempt should be made to suture, instead of to ligate, the vessel.

Suture also would seem to be worth trying when a main vessel is locally thrombosed from the effects of trauma. If there is difficulty in bringing the cut ends of the vessel into apposition a Tuffier's tube may be tried.

Injuries of the popliteal artery are so uniformly followed by gangrene, if the vessel is ligated, that every means should be tried to re-establish the lumen if only for a few hours.

In the case of circumscribed arterial haematoma accompanying a small wound of the limb, time alone can show if an attempt to suture the artery is good. One should most probably be guided by the state of the circulation in the distal portion of the limb.

(b)

In dealing with gas gangrene in a wounded segment of a limb and deciding on the advisability of amputation, it should be borne in mind that it is usually only the wounded muscles that become gaseous and that incision or ablation of such muscles is often sufficient to arrest the disease and stop the infection (see Case vi).

In the case of the thigh the ablation of muscles may present difficulties, but in the leg this part of the limb can often be saved by the ablation of the anterior tibial group, and the same applies to the muscles of the forearm. The brick-red colour and the non-contractility will show at once which muscles are past saving.

(c)

When gas gangrene occurs in a segment of a limb distal to the segment wounded it nearly always means that the main artery is blocked, and amputation of the gangrenous segment is the only course.

(d)

The presence of crepitation in tissues apart from other signs is of no special importance. A limb should not be sacrificed on account of crepitant skin (see Case ii). The state of the muscles and the number dead should be ascertained before amputation is performed, otherwise a limb or a very considerable portion of its length may be sacrificed unnecessarily.

The disease is the great bugbear of the surgeon at the front. It involves much labour and complicates the evacuation of the wounded. It occurs in all sorts of wounds, whether small or large, and with all sorts of projectiles. There is no wound, however insignificant, that one can feel happy about if not opened up. It is true that the disease is not so dangerous now as it used to be, because of the universal opening up of wounds, but the amount of trouble that it entails and the amount of disturbance that it is necessary to inflict on a grievously wounded patient makes one long for some remedy other than surgical interference.

THE DIRECT TRANSFUSION OF BLOOD:

*ITS VALUE IN HAEMORRHAGE AND SHOCK
IN THE TREATMENT OF THE
WOUNDED IN WAR.**

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THE direct transfusion of human blood from one individual to another is an operation of great antiquity, but in the history of medicine it has never been as extensively employed as it is to-day. From time to time transfusion of blood has been abandoned, largely because of technical difficulties in its performance, but of late years, as the result of the introduction of more perfect and less complex methods, the operation has become much more generally employed. Its value as a therapeutic measure in the treatment of many pathological conditions has in recent years been established on a scientific basis. Thus it has proved of great service in the treatment of haemorrhage, shock, illuminating gas poisoning, and of more doubtful value in pernicious anaemia and certain toxæmias.

We will not review the observations which have been made in recent medical literature regarding the value of transfusion in the multitudinous conditions in which it has been used; we propose to restrict our observations to the value of direct transfusion of blood in the emergencies of military surgery. There may be some question as to its efficacy in many of the conditions in which it has been

employed, but in haemorrhage and shock, more particularly in shock accompanied by haemorrhage, its value has been proved beyond a shadow of doubt. We may assert that we have no more efficient means at our command to save life in cases of severe haemorrhage than by the direct transfusion of human blood from one individual to another. This has been proved not only in traumatic haemorrhage (primary and secondary), but in haemorrhage from such conditions as gastric ulcer, typhoid ulcer, ruptured extra-uterine pregnancy, the haemorrhages of the newborn, and in haemophilia.

If we inquire into facts which are known regarding the effect of introducing human blood into the vascular system of an individual suffering from haemorrhage, we realize the difficulty and complexity of the problem involved in our effort to appreciate the full result of transfusion. As regards the transfused blood, we have no precise knowledge concerning the fate of the corpuscular elements, nor of the plasma. The possibility of the harmful effect of lysins or agglutinins in the blood of the donor or recipient has been feared, but this has been greatly exaggerated. A large amount of clinical evidence has accumulated to warrant us in assuming that no serious harm is to be anticipated from such sources. The laboratory tests for haemolysis and agglutination may be carried out if time and facilities permit, but they are not necessary and are by no means trustworthy. The laboratory findings in haemolysis, for example, have been found at times at variance with the actual results obtained in the living body. No fatal result from either haemolysis or agglutination has hitherto been reported.

The danger of the transmission of disease makes it essential we should inquire into the past record of the donor, particularly as to whether or not he has had syphilis.

Two results of transfusion stand out prominently and are unquestionably of enormous value in the treatment of haemorrhage. These are, first, it increases the power of coagulation of the blood, and second, it improves the local resistance of the tissues to infection. These facts have been recorded by a number of observers over a large series of cases and have been shown in the most striking fashion in the cases of secondary haemorrhage which we have personally treated by transfusion. It we stopped there and claimed no more for transfusion our contention in urging its employment in such cases would be justified. There are, however, other effects which are more difficult to analyse, and yet which we are warranted in believing are of great service. If we introduce, say, a litre of blood with all its constituent elements into the vascular system of an individual suffering from the direct effects of a severe haemorrhage, and if we have reason to believe the transfused blood takes on its normal function in the recipient, the ideal therapeutic measure has obviously been employed. This is admittedly a very complex problem, and we acknowledge our ground is not as sure in the matter of conclusive evidence as it is regarding the effect upon the coagulability of the blood and the increased resistance to infection. If, however, we employ all known tests after transfusion and find no evidence of haemolysis, we have strong ground for assuming that the donor's blood, in all its elements, circulates and functions in the recipient. If, for example, we find no evidence of haemoglobin in the urine, we certainly have no lacking of the red cells in the circulating blood. The possibility of phagocytic destruction of red cells in the liver cannot so readily be disposed of, and we have no means of determining this factor. The blood count does not help us to any extent, because we find, for example, that if a litre of blood is transfused into a patient who is exsanguinated, the blood count is not necessarily altered in spite of the fact that the red cells are low in our patient at the time. Thus, recently a patient with a red count of 3,000,000 received a litre of blood from a donor with a red count of 5,000,000. Two hours subsequently the red cells in the recipient were still 3,000,000, but his colour was markedly improved and his blood pressure and pulse were maintained at normal. Obviously the introduction of the more concentrated blood resulted in an osmosis which quickly restored the former equilibrium and resulted in a dilution of the mixed blood now circulating. There was no evidence of haemolysis and the result of transfusion was to increase

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