In April, 1918, another batch of 16 severe cases of mustard gas poisoning were admitted. These were sent by Captain Barber, R.A.M.C., No. 25 General Hospital; only one had to be evacuated to England owing to symptoms referable to the heart. This man was fit previous to being gassed. On admission he was very short of breath, and had a markedly dilated heart, the apex beat being $4\frac{1}{2}$ in. from mid sternum with a short apical systolic murmur. The remainder returned to duty without any symptoms of D.A.H.

Table VIII shows how 318 gas poisoning cases were discharged. Only 8 of them were evacuated to England, and 61.1 per cent. of the A men were discharged as A. How these gas cases were discharged is a good indication as to the permanence of their D.A.H. symptoms, especially if the following table be compared with the discharges from other causes in Table IX.

TABLE VIII.—Discharge of 318 Gas Cases.

Category.	How Discharged.
A 293	A 179 = 61.1% B 62 = 21.2% Hospital 20 England 8 Other dépôt 24 = 17.7%
B 23	B 21 = 91.3% Other dépôt 2 = 8.7%
Unknown ... 2	Discharged A... .. 1 Discharged B... .. 1
Total ... 318	318

I think, therefore, we may conclude that though gas produces during convalescence symptoms of the same nature as D.A.H., yet that these symptoms are neither serious nor lasting.

Age and various diseases other than those mentioned are the cause of a small percentage.

Lastly, those to whom it was impossible to attribute a definite cause accounted for from 15.7 to 27.4 per cent. This seems a large number, but in order to obtain figures of any value in working out the causation this could not be avoided.

CONCLUSION.

In conclusion, I would state that the strain, mental and physical, of warfare is the chief cause of the symptoms of D.A.H. That the causation of next importance is infective diseases, and of these the majority was due to those contracted in civil life, rheumatic fever being the worst offender.

Table IX shows how 6,269 cases of D.A.H. were discharged, and for comparison I have added a table giving the discharge of 790 cases which were due to rheumatic fever and 422 cases following P.U.O.

TABLE IX.

Discharge of 6,269 D.A.H. Cases.

Admitted as A men	4,991 cases.
Discharged as A men	48.0 per cent.
Discharged as B men	39.1 "
Discharged to hospital, O.C.D., England	12.9 "
Admitted as B men	1,278 cases.
Discharged as B men	89.0 per cent.
Discharged to hospital, O.C.D., England	11.0 "

Discharge of 790 Cases of Rheumatic Fever.

Admitted as A men	560 cases.
Discharged as A men	31.2 per cent.
Discharged as B men	44.5 "
Discharged to hospital, O.C.D., England	24.3 "
Admitted as B men	230 cases.
Discharged as B men	69.8 per cent.
Discharged to hospital, O.C.D., England	30.2 "

Discharge of 422 Cases of P.U.O.

Admitted as A men	364 cases.
Discharged as A men	58.0 per cent.
Discharged as B men	17.6 "
Discharged to hospital, O.C.D., England	24.4 "
Admitted as B men	58 cases.
Discharged as B men	86.2 per cent.
Discharged to hospital, O.C.D., England	13.8 "

My thanks are due to Colonel W. E. Hume, C.M.G., A.M.S., for permission to use the records from which the above figures are taken, and also to Lance-Corporal Dillingham, R.A.M.C., whose assistance in extracting the figures was invaluable.

THE PATHOLOGY AND TREATMENT OF STIFF KNEE IN RELATION TO COMPOUND FRACTURE OF THE FEMUR.

BY

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FIBROUS or bony ankylosis of the knee frequently follows compound fracture of the femur when the fracture is into the joint or when there has been prolonged suppuration near the joint.

There is, however, another class of "stiff knee" which follows compound fracture of the shaft of the femur in which the limitation of flexion is not due to articular so much as periarticular and muscular changes. This is the type which yields very satisfactory results with careful treatment, but is frequently dealt with in a very offhand sort of way, leading often to disastrous results. Stiff knee after compound fracture of the femur may be due to articular, periarticular, or muscular causes.

It is chiefly the changes in the quadriceps which concern us at the moment, and before we discuss the treatment it is useful to glance at the pathology of these, as our methods must be guided largely by it. We should keep it in mind that some degree of joint infection must occur in all cases of compound fracture, although in those we are considering the reaction of the joint tissues is generally sufficient to deal with this. The real obstruction to flexion is to be looked for in the very profound alteration occurring in the quadriceps as a result of sepsis and prolonged disuse.

In a typical example we find a markedly wasted and scaphoid quadriceps. On attempting passive flexion of the knee considerable pain is generally caused on account of periarticular adhesions, but if these are absent a very much shortened quadriceps prevents flexion for more than a few degrees. This means that the normal functions of extensibility and contractibility are absent; that in parts dense scar has replaced the muscle tissue; and that there are scars of healed sinuses reaching down to bone. This state of affairs has resulted from the original injury, the persistence of sepsis, and prolonged disuse.

Histologically the picture varies with the position from which the sections are taken. Those near the septic focus show absolute replacement of muscular tissue by scar, while other sections show decreasing amounts of scar as we get further from the site of injury.

Throughout the muscle, even in that portion most distant from the injury, there is considerable shortening of individual muscle fibres, which, in addition to showing marked contraction in size, are in a state of partial degeneration, and as a result of prolonged disuse have "forgotten" how to function. Their condition is in fact that of typical disuse-atrophy.

It is a well established pathological fact that cells in this state tend to be replaced by fibrous tissue, and this does occur to some extent just as in the same way such structures as the ductus arteriosus and thymus, when they cease to function, are gradually replaced by fibrous tissue.

But not only is the muscle substance shortened and partially replaced by scar; the individual fibres are surrounded by lymph spaces which drain into lymph capillaries, and it must be remembered that lymph is coagulable in virtue of the fact that it contains fibrinogen and its cellular elements thrombin. Coagulation will follow any of those incidents which cause clotting of the blood; among these are trauma, contact with harmful nucleo-proteins manufactured in the septic focus, and stasis. Nature attempts to remove this clot by phagocytosis, but as a result of the special conditions we are considering a certain amount of scar tissue remains, gluing the fibres together and forming intramuscular adhesions.

In opposition to the changes above enumerated there is a certain degree of regeneration going on as a result of

proliferation of sarcolemma nuclei, and muscle cell budding and splitting. The survival of the young muscle cells thus formed depends upon the degree of encouragement they receive to function; otherwise they run the risk of destruction before they reach maturity, either simply by disuse-atrophy or by condensation of the recently formed scar tissue. Their failure to survive only increases the amount of fibrous tissue formation.

During the treatment of compound fracture of the femur, then, we have all the necessary conditions for the formation of fibrous tissue in muscle—namely, trauma, sepsis, and prolonged disuse. Our picture of the quadriceps, therefore, is that of a muscle which has practically lost its contractility and extensibility and is pervaded throughout with fibrous tissue gluing the fibres together, and to a large extent actually replacing the muscle cells. In addition, in the neighbourhood of the fracture and about each sinus the muscle is riveted, as it were, to the bone by dense scar. Even with a normal joint such a shortened muscle forms a very considerable obstacle to flexion of the knee. Similar changes are found about tendon sheaths and in tendons and ligaments around the knee-joint due to the same causes.

The conditions to be dealt with, therefore, before we get movement are perimuscular and periarticular adhesions, intramuscular adhesions, contracted and imperfectly functioning muscle cells. Experience in the treatment of stiff knee, following the type of compound fracture we are considering, shows that the real block to flexion is not due to changes in and about the knee but in the quadriceps.

Treatment should be first prophylactic, by chemical sterilization, removal of all sequestra, and early secondary closure of wounds when possible. Secondly, any scarring which binds the muscle to bone and all intramuscular and periarticular adhesions must be broken down. Thirdly, the normal functions of contractility and extensibility must be restored and growth of young muscle cells encouraged.

As soon as the condition of the wounds permit the quadriceps should be treated by graduated contraction by means of the Bristow faradic coil. This loosens intramuscular and periarticular adhesions, and restores tone to the muscle, which begins to increase in size.

If the fracture is united and the patient out of the Thomas splint, a small sand-bag is placed beneath the knee as he lies in bed, the heel being off the bed. The weight of the leg is then sufficient to commence flexion. As the knee begins to bend the size of the sand-bag is increased, the patient being encouraged in the meantime to extend the knee voluntarily. In this way flexion of 20 to 30 degrees is obtained, and graduated contraction and exercises are continued until he is able to fully extend the knee from the flexed position.

The knee should then be further flexed under gas, the remaining intramuscular and periarticular adhesions being broken down in this way. It is most important that during this operation the knee should not be bent beyond the point of extreme muscular extensibility at the time. When this point (generally just short of 90 degrees) is reached the surgeon feels a characteristic jerk, which can only be recognized by experience, but which closely resembles the sudden stop felt on dorsiflexing the ankle of a patient with contraction of the Achilles tendon. The knee and thigh are then vigorously slapped (tapotement)—a procedure which tends to limit any effusion into the joint and deep tissues. The patient is put back to bed either in a suspended knee flexion splint, in which the thigh only is fixed, and which is bent at an angle beyond that obtained during the operation, or simply with the joint flexed over sand-bags as before described. The following day the knee may be slightly swollen, and the patient may complain of some pain on each side of the patella ligament, and where the muscle scarring is most dense. Gentle massage (effleurage) is comforting at this stage, while weak faradic graduated contraction undoubtedly hastens absorption of effusion from the tissues.

This treatment—massage and graduated contraction—should be continued for three days; at the end of this time any effusion will have disappeared and passive movements should be commenced in addition.

Two points will now be noticed: First, that flexion cannot be obtained to the same degree as in the theatre—the loss may be as much as 10 degrees—and secondly, that voluntary extension is only to about 165 degrees. This

range, particularly the extension, will increase from day to day under treatment by the Bristow coil and voluntary exercises until the patient is able to perform active movement from about 100 degrees to full extension.

At the end of a week or ten days, if the fracture is firmly united, the patient should be instructed in knee-bending exercises while standing at the foot of his bed. At least every other day he should be made to lie face downward on the bed, and the surgeon, holding the thigh down by grasping just above the knee, should flex the leg passively with his other hand in one slow, firm, continuous movement. By these means flexion beyond a right angle is generally obtained. Daily graduated contraction should be continued for four or five weeks, at the end of which time, if improvement is not satisfactory, the knee should be wrenched again.

There are several precautions to be taken, apart from those already mentioned, when wrenching a knee: First, if the site of fracture is tender to deep pressure, coaptation splints should first be applied. Secondly, there should not be too much anxiety to get too much flexion at one sitting. Overflexion may result in rupture of the quadriceps, injury to the patella ligament and capsule of the joint, or even fracture of the patella. Thirdly, whatever the degree of flexion, the patient is not ready for a second wrenching until he is able to extend his leg fully.

During the after-treatment the graduated contraction should not be too severe. If, following treatment by this method, the patient complains of stiffness in his muscle, it is probable, as pointed out by Major Bristow, that more harm than good is being done. An overworked muscle does not develop, it atrophies.

What has been said about the knee applies obviously, though to a lesser extent, to the other joints of the limb. During treatment, then, it is the duty of the surgeon to see also that the hip and ankle joints and the toes are not allowed to become stiff.

TREATMENT OF BILHARZIAL INFECTION BY TARTAR EMETIC.

BY

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TARTAR EMETIC, as advised by J. B. Christopherson,¹ must be accepted as a positive cure of bilharzial infection. I knew many of the cases on which his recommendation was based, and had commenced the treatment in the Egyptian Military Hospital, Khartoum, where the opportunities of proving or disproving its efficiency are many among the fellaheen recruits of the Egyptian army. Since then McDonough has reported that he had previously used the drug; and Major Strong of the Australian Medical Service tells me that he and one or two brother officers had worked with it in 1916, in fairly small doses and at intervals of not less than a week between the injections, but no one felt convinced of the possibility of curing the disease, nor had anyone the courage to adopt the truly heroic dosage which is necessary to affect the bilharzia worm and its ovum.

Our cases are divided into two series; the first is composed of twenty-six cases which were treated in the period from June 27th, 1918, to August 10th, 1918, and the second of thirty-six cases treated from August 12th, 1918, to January 15th, 1919.

The interval between these two series was longer than the actual dates seem to indicate, for there happened in August an outbreak of influenza in the ward where these cases were; the temperature in all the cases was considerable—never less than 104°—and one patient died whose organs showed most extensive fatty degeneration;² fearing the toxic effect of antimony in such cases, the treatment was discontinued in several patients whose urine still showed bilharzia, and the first three of the second series were started on injections of 10 c.cm. of mercury perchloride solution, 1 in 1,000, as has been tried by Madden of Cairo. But this solution failing to have the effect of clearing the urine of blood and ova, we again resorted to tartar emetic, and have used it invariably since.

Of the first series of 26 cases, 14 were discharged with the urine free of blood and ova, 11 were still infected but