keeping with conservative ophthalmology than to enucleate the eyeball if any sympathetic irritation is present. A few days ago at my home in Denver on the day I was preparing to start for Milwaukee, a gentleman came to my office who had an injured eye. The ocular injection was very great and inflammatory action had gone to a considerable extent. The well eye I noticed, on examination, was wanting quite a little in power to accommodate. He said to me: "I came to get some medicine for my eye." I examined it carefully and found that it had been caused by the introduction of a foreign body. I told him he was running a considerable risk in allowing the affected eye to remain in its orbit and assured him that he had a sympathetic irritation in the well eye, which was likely to produce total blindness unless attended to at once. The affected eye looked phthisical, was minus tension and the vision was entirely gone. I said: "I will enucleate the eye for you, if you want it done, otherwise I will do nothing." He went away but soon returned and informed me that he would take my advice and have the operation performed. On opening it I found the remains of a foreign body.

About three years ago lady came to my office and said: "Doctor, I have come to get something to allay this inflammation in my eye." It had never given her such trouble before. It was brought about by the introduction of a small piece of thorn having pierced the ciliary body. I told her I would enucleate the eye by all means, as she was beginning also to have sympathetic trouble in the well eye, and that I could not be honest in advising her to try to hold on to the injured eye longer, as it would be running a risk that I would not carry for a thousand dollars a day. She refused to abide by my advice and went away and the sequelæ will tell the result: in three months she returned, and I found that the injured eye had recovered, to a great extent, from the introduction of the foreign body, but on examining the other eye, alas! I found it too was in total darkness. Had I advised this woman at her first visit to me to have pursued the course she did, my conscience would haunt me to this very hour for not doing my duty. I do not say to do a wholesale enucleation for every foreign body—far from this; if sympathetic inflammation has manifested its presence I never fail to advise enucleation, and I have got the first case to regret for having taken such a course. I do not think we act wisely by attempting to carry these risks. This case is only one of a dozen or more that has come under my own observation with like results.

DR. HÖRZ—called attention to the fact that prognostically it made a vast difference whether the foreign body in the eyeball was lodged in the vitreous, or in the lens, or in the iris. General statements therefore on foreign bodies in the eye without mentioning the location must be misleading; for instance, when a gentleman said it was his rule to take any eye out in which he found a foreign body, he certainly did not wish us to believe he would enucleate eyes having a foreign body in the iris or lens; for these foreign bodies may be either left in situ or can successfully be removed and the eyeball saved. But conservative treatment can not be recommended for foreign bodies in the vitreous. The experience of all oculists, at all times, has been that foreign bodies in the vitreous are a prolific source of sympathetic inflammation. Such eyes are a constant menace to their fellows and should be removed; for we have no right to jeopardize the sight of thousands of eyes by a conservative treatment, because in some rare exceptions it has proved satisfactory. We must not be guided in our actions by what our experience of every day shows to be the usual course with such patients.

PROF. ZEHENDOR (by invitation of the chairman) called attention to the experiments of Kries and Haab with electro-magnets of unequal strength for the extraction of iron

and steel from the eye without entering the eyeball, but by magnetic traction through the tunics. He has observed two cases operated on in this manner by Dr. Schloesser of Munich. In both cases the extraction of the iron piece succeeded very well and quite easily, but in one of these cases the piece was so big, that in spite of the extraction the eye was destroyed. He also related an instance in his experience in which he observed at the time the entrance of a foreign body into the interior of the eye in a boy, which was followed by retinal detachment and atrophy of that eyeball, but had not caused any sympathetic trouble in the fellow eye up to the time of his death sixteen years later.

THE ACTION AND SAFETY OF CHLOROFORM.

Being an abstract of a report to Surgeon Lieutenant Colonel Edward Lawrie and the Government of His Highness, the Nizam of Hyderabad, India.

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To any one who endeavors to view the subject of chloroform anesthesia in an entirely impartial light, two objects raise themselves so high above all others that they form the peaks about which the smaller questions must cluster. The first object on which the judicial eye rests is the firm belief of many clinicians that chloroform may cause sudden cardiac death; the second object to be seen is the statement of the Hyderabad Chloroform Commissions that death from chloroform is never due to cardiac failure. In the support of the first belief we have not only wide clinical observation but also the experimental evidences of a number of investigations. In support of the second statement we have an array of experimental study, not equaled by any other research extant, associated with an enormous number of negative observations on man. Negative observations, because Lawrie alone in 25,000 cases has never had a death.

The controversy concerning the action of chloroform upon the animal organism has been waged so incessantly for many years, and has led to such extraordinary efforts for its elucidation and final decision that any one who attempts to take part finds himself swamped by the number of statements and opinions which he is forced to regard. We have, therefore, approached this research feeling it was no ordinary task, and that a path already so well traveled must be gone over with the utmost care if anything new or of value was to be discovered.

In March, 1892, Surgeon Lieut.-Col. Lawrie, whose interest in this subject is recognized by the medical profession the world over, wrote to one of us (Hare) asking that another chloroform research be instituted for which the Government of his Highness the Nizam of Hyderabad would pay. The express object of the research was the reconciliation of at least some of the contradictory conclusions reached by various experimenters during the past few years.

From the immense number of observations in regard to the action of chloroform in the laboratory and in the operating room it is evident that sufficient data are at hand to give us material to reach positive conclusions, and that the contradictory results hitherto obtained must have been reached by misinterpretation and error in experimental method, tinctured perhaps by opinions formed previous to

the completion of a line of study. There are certain facts in regard to chloroform which few will deny, the chief of which are that it has the advantage of rapid action without disagreeable preliminary or subsequent symptoms, its bulk is small and its odor agreeable but, more important than all, it is much more dangerous than ether.

Though the Hyderabad Commission in their preliminary conclusions (page 30, paragraph 43) assert that ether is as dangerous as chloroform if given sufficiently to produce true anesthesia, we believe that the safety of ether is so universally recognized that this conclusion of the Commission can only be excused by the remembrance that ether has probably been used as little by those who wrote this paragraph as chloroform is used in many parts of America. This possibility is made a probability when we read that "if surgeons choose to be content with a condition of semi-anesthesia, it can no doubt be produced with perfect safety, though with discomfort to the patient, by ether held rather closely over the mouth. Such a condition of imperfect anesthesia would never be accepted by any surgeon accustomed to operated under chloroform." That this statement shows, to put it mildly, that the writer knows not whereof he speaks is proved by the universal employment of ether by hundreds of the best surgeons the world over in preference to chloroform. Further than this, medical literature contains so many statistical papers showing the small percentage of deaths from ether, compared to chloroform, that this point need not be discussed.

There are certain other points in regard to the action of chloroform which may be put aside as settled and therefore not needing further study, being generally received as beyond criticism, and we have made no experiments looking to their reproduction but have devoted our efforts solely to the question over which discussion still proceeds. Thus, all investigators concur in the statement that chloroform even in ordinary therapeutic quantity, acts as a powerful and constant depressant to arterial pressure. This conclusion has been reached by Bowditch, Minot and Coats, H. C. Wood and H. A. Hare, Gaskell and Shore, the Hyderabad Commission Nos. 1 and 2 and by every experiment in the research now carried out which forms this report. There is no evidence to the contrary and practically it has never been denied. Gaskell and Shore state, however, that chloroform may cause anesthesia without lowering blood pressure and that chloroform causes primarily a rise of pressure. They also believe that the chief cause in the fall of arterial pressure is cardiac and not vasomotor depression. The rise we have never seen except from struggles, and we have never been able to produce anesthesia without lowering the blood pressure, even when the drug was used in the smallest quantity capable of causing anesthesia and given as slowly as possible. We agree with the statement of the Hyderabad Commission that a fall of blood pressure always occurs when chloroform anesthesia is produced.

We are also forced, as the result of our studies, to differ entirely with Gaskell and Shore in their statement that the fall of arterial pressure is due primarily to a weakening of the heart's action and not to paralysis (depression?) of the vasomotor nerves. We believe that both factors cause the fall, but that the dominant factor is vasomotor depression because

as will be seen in several of our tracings the pulse waves were quite strong though the blood pressure, through vasomotor relaxation, was absolutely to the abscissa line and we agree with Lawrie that no conclusions as to the action of chloroform when inhaled can be drawn from the injection experiments of Gaskell and Shore into the arteries. One of our reasons for this belief is the entire difference in result. Another reason is that any powerful drug injected into the brain will cause a rise of blood pressure. Even nitrite of amyl, the prince of vasomotor paralyzants, will do this. (See Gaskell and Shore 17, paragraph 68). We also believe that results obtained by such interesting methods of experimentation as those employed by Gaskell and Shore are not capable of giving positively reliable information, as the conditions are so utterly at variance with those in which chloroform is given to man and, further than this, their methods are such as to give room for fallacious results which can not be excluded by the greatest caution on the part of experimenters experienced as they are.

Secondly, it is not denied by any one, that we know of, that chloroform exerts a powerful, depressant, paralyzant action on the respiratory center. This is agreed to by clinicians and by every one who has experimentally studied the action of the drug on the lower animals.

Thirdly, it is universally conceded that chloroform is a lethal agent of great power when brought in direct contact with highly vitalized tissues. Aside from these facts there are a number of others in which we find ourselves strictly in accord with the conclusions of the first and second Hyderabad Commissions.

We find when the drug is pushed that insensibility comes first, then respiratory failure and finally cardiac arrest in every case in the lower animals. In other words, that cardiac arrest never ensues primarily from inhalation of chloroform.¹

SUMMARY.

From a careful study of the experiments so far reported, from studies made by one of us two years ago with H. C. Wood, and finally from this careful series of experiments we believe that the subject can be settled by the acceptance of both views in a modified form, or in other words that there is no real antagonism in the beliefs that chloroform kills by depression of the respiration or paralysis of the heart. We very positively assert that chloroform practically always kills by failure of respiration when administered by inhalation, provided, and this provision is most important, that the heart of the anesthetized is healthy and has not been rendered functionally incompetent by fright or violent struggles or, again, by marked asphyxia. By a healthy heart we mean one which has not undergone true fatty degeneration or has not so severe a valvular lesion as to make the slightest variation in the even tenor of the circulation fatal.

As positively as we assert that chloroform kills primarily by respiratory failure, so do we also assert that in excessive dose by inhalation it has a depressant effect on the circulation which is chiefly due to centric vasomotor depression with final depression

¹ Lack of space prevents the inclusion of three experimental records. They can be found in the issue of the *Therapeutic Gazette* for Oct. 15, 1893.

of the cardiac muscle itself. Depression of the cardiac muscle alone is never great enough to cause death when the chloroform is given by inhalation, but we believe that gradual asphyxia with the direct depression of the circulation may do much towards producing a fatal result, for vasomotor integrity is almost as necessary to life as an intact cardiac mechanism. This circulatory depression has been considered a safeguard because it was supposed to prevent chloroform going to the vital centers, but in reality it is no safeguard because profound circulatory depression is as great an evil as respiratory narcosis. That the circulatory depression may be dangerous is not only evident, but it is stated to be so by the Hyderabad Commission itself at the end of paragraph No. 8. This circulatory depression may be so profound that recovery is impossible, even with the most thorough artificial respiration, a fact stated by the second Hyderabad Commission in paragraph 25, which we quote in this paper. This emphasizes the fact that we can not afford to totally ignore the effect of chloroform on the circulation and we can not consider the patient in danger of circulatory failure *only* when the respiration ceases, but as soon as it becomes abnormal. On the other hand, we should remember that even if chloroform has been given properly, the arterial pressure may be so low as to give no pulse in the radial artery and yet the circulatory system be ready to respond at once when the drug is removed. If, therefore, the chloroform is properly administered is there danger of its circulatory effect in man? We think that it is just at this point that our research and every other research on animals fails and necessarily fails, to produce a positive reply. The variation in the action of a drug on a diseased individual from its effect on the normal one is notorious, and we have no right to dogmatically assert that there is absolutely no danger of a circulatory depression in man, even if we found no evidence of failure in dogs, because there may be many idiosyncrasies or variations through disease in the human being which may completely reverse the results of experiments on healthy animals.

In other words, supposing that the amount of depression from very full doses of chloroform equals 25 units this amounts to little in the normal heart, but if the heart be depressed 25 additional units by disease the depression of 50 units may be fatal, particularly if to this 50 is added 25 units more of depression through fright and cardiac engorgement through disordered respiration or struggling. That true depression of the heart muscle may take place under chloroform seems to us most undoubted and we think that the tracings in every research that we have seen support this view. There is always a decrease in cardiac power manifested by the decrease in the force of the individual pulse beat and this passes away only if chloroform is removed early enough. We also agree with McWilliams that from the very first inhalation of chloroform there is a constant tendency to cardiac dilatation.

We come finally to the all important questions:

1.—Is chloroform a safe anesthetic?

2.—Are we to watch the pulse or respiration during the use of the drug, and what are the signs in the respiratory function indicative of danger to the patient?

3.—What is the true cause of death from chloroform?

4.—Is death from chloroform possible when it is properly administered?

5.—Under what circumstances is the surgeon to use chloroform in preference to the less dangerous anesthetic, ether?

6.—What is the best way of administering chloroform?

To the first question the answer is yes, for the majority of cases, provided it is given by one who is skilled in its use and not only knows how to give it but to detect signs of danger. It is not as safe as ether at any time, other things being equal, and never safe in the hands of a tyro.

To the second question the answer is, watch the respiration, *because as soon as enough chloroform is used to endanger the circulation the respiration will show some signs of abnormality either in depth, shallowness or irregularity.* In other words, the very effect of the drug may be to cause such deep and rapid respirations that an excessive quantity of the drug is taken into the lungs and continues to be absorbed even after the inhaler is withdrawn.

As there is always a fall in pressure under chloroform it is difficult to feel the radial or temporal pulse and the respiratory center recognizes the degree of arterial depression, which its sister vasomotor center has permitted by finding that its blood supply is insufficient. As respiration fails first it should be watched first. Finally, it is only by watching the respiration that we can tell how much chloroform the patient is getting. We do not watch this function for danger alone but to tell us of the dose.

The answer to question three is that death is always due in healthy animals to respiratory failure accompanied by circulatory depression, which latter may be severe enough to cause death even if artificial respiration is used skillfully. Death only occurs in the healthy animal when chloroform is given in excessive quantities.

Question four is impossible to answer for man from the basis of experimentation, as we can not produce identical diseased states in animals with those developed under various conditions in man. The physician having a case of heart disease should always advise the patient of the danger of any anesthetic and he should remember, whether it is wise to tell the patient or not, that anesthesia always means a step toward death even in the healthiest of men. In the event of a death under chloroform the physician is not to blame if he has taken proper preliminary precautions and given the chloroform properly.

Every one is agreed that the patient taking chloroform should have plenty of fresh air and in India we understand that to all intents and purposes patients are operated on in the open air, at least as compared to the closed rooms necessary in America and Europe. This free supply of air is important, whether we believe death to be imminent from cardiac or respiratory failure, but this supply of air matters little to the patient if he does not breathe freely nor does the dose of chloroform amount to aught if it is not drawn into the chest. The dose of chloroform is not the amount in the inhaler but the amount taken into the chest and finally the amount absorbed by the blood vessels. The rapidity and depth of respiratory movements is therefore, as Lawrie asserts, the entire key to the situation. We watch a windmill over a well to see if it is pumping into a reservoir a given quantity of water. If the

windmill works irregularly so that we know its pumping action is deranged we separate it from the pump until the wind blows steadily, in order that it may pump regularly. Similarly we withdraw chloroform, as Lawrie says, whenever respiration becomes disturbed in rhythm or when struggling disturbs it, because it is the first indication that the drug's action is uncertain and because there is no telling the dose which is absorbed. While watching the respiration will not warn us of a sudden cardiac arrest in fatty heart plus chloroform depression, neither will the pulse give us such warning and we are confident that the statement of the Hyderabad Commission that the *respiration should be watched is correct*, for we believe from a long series of observations that gradual cardiac failure never occurs without producing respiratory changes from the very first. In other words, we do not believe that in a healthy heart chloroform can cause serious disorder without as a result of beginning disorder disturbing respiration and, second, that in a healthy heart a quantity of chloroform sufficient to disorder it will by its direct action disorder the respiration. If, as an extra precaution, one assistant watches the pulse while the other watches the respiration, very well; for though the respiration is the more important function the man watching the pulse might discover an irregularity which the anesthetizer may not see reproduced in the respiratory action, but as divided attention generally means a slighting of both objects in view Lawrie is right in insisting on the pulse being let alone.

In answer to question five we have several points to offer: 1. Hot climates where ether is inapplicable and where a free circulation of air increases the safety of the patient; 2. Chloroform may be used whenever a large number of persons are to be rapidly anesthetized so that the surgeon may pass on to others and save a majority of lives, even if the drug endangers a few, as on the battlefield where only a small bulk of anesthetics can be carried; 3. Its employment is indicated in cases of Bright's disease requiring the surgeon's attention, owing to the fact that anesthesia may be obtained with so little chloroform that the kidneys are not irritated, whereas, ether, because of the large quantity necessarily used, would irritate these organs. Quantity for quantity, ether is, of course, the less irritating of the two; 4. In cases of aneurism, or great atheroma of the blood vessels, where the shock of an operation without anesthesia would be a greater danger than the use of the anesthetic, chloroform is to be employed, since the greater struggles caused by ether and the stimulating effect which it has on the circulation and blood pressure might cause vascular rupture; 5. In children or adults who already have bronchitis, or who are known to bear ether badly or, in other words, have an idiosyncrasy to that drug, chloroform may be employed; 6. Persons who struggle violently and who are robust and strong are in greater danger from the use of chloroform than the sickly and weak, probably because the struggles strain the heart and tend to dilate its walls.

The safest method of administration is by Lawrie's or Esmarch's inhaler because these provide free circulation of air and do not distract the attention of the anesthetizer from the respiratory movement by complicated apparatus. Apparatus much like these in allowing a free amount of air, are the Hyderabad chloroform inhaler, or open-ended cone with

Krohne and Seseman's respiration indicator attachment. The Junker inhaler, even with its modifications, is too complicated and cumbersome and while less chloroform is wasted in administering the drug it must all be thrown out of the bottle afterwards. If used at all it should be used with the increased air supply and respiration indicator of Krohne and Seseman.

We agree so heartily with Lawrie's conclusions that we print them below:

1.—The chloroform should be given on absorbent cotton stitched in an open cone or cap. (A depression made through the opening in the inside flannel bag will answer as well.)

2.—To insure regular breathing, the patient lying down with everything loose about the neck, heart and abdomen, should be made to blow into the cone, held at a little distance from the face. The right distance throughout the inhalation is the nearest which does not cause struggling or choking or holding of the breath. Provided no choking or holding of the breath occurs, the cap should gradually be brought nearer to, and eventually may be held close over, the mouth and nose as insensibility deepens.

3.—The administrator's sole object while producing anesthesia is to keep the breathing regular. As long as the breathing is regular and the patient is not compelled to gasp in chloroform at an abnormal rate there is absolutely no danger whatever in pushing the anesthetic till full anesthesia is produced.

4.—Irregularity of the breathing is generally caused by insufficient air, which makes the patient struggle or choke or hold his breath. There is little or no tendency to either of those untoward events if sufficient air is given with the chloroform. If they do occur the cap must be removed, and the patient must be allowed to take a breath of fresh air before the administration is proceeded with.

5.—Full anesthesia is estimated by insensitiveness of the cornea. It is also indicated by stertorous breathing, or by complete relaxation of the muscles. Directly the cornea becomes insensitive, or the breathing becomes stertorous, the inhalation should be stopped. The breathing may become stertorous, while the cornea is still insensitive. The rule to stop the inhalation should, notwithstanding, be rigidly enforced and it will be found that the cornea always becomes insensitive within a few seconds afterwards. It is only necessary to add that the patient should be so dressed for an operation that his respiratory movements can be easily seen by the chloroformist. In the climate of India this is not difficult to manage, but it is rather more so in the climate of Europe; so that in this respect, and in this respect alone, the chloroformist in England and America is placed at a distinct disadvantage compared with the chloroformist in India.

NOTE.—Since writing this report two important papers upon this subject have appeared in the London *Lancet*. The one by Gaskell and Shore, in which they carried out a line of ingenious cross-circulation experiments, and from which they conclude that the fall in blood pressure seen under chloroform is due to cardiac rather than vasomotor depression; and another paper published by Lawrie, in the London *Lancet* for Feb. 11, 1893, in which he refutes the statements made by Gaskell and Shore, and details experiments which he believes combat those of the two investigators just named.

The concluding paragraph of Lawrie's latest contribution to the subject states the facts so clearly and is so in accord with what we have tried to set forth in our own report that we can not do better than quote the paragraph:

"The Hyderabad Commission's work proves that, while Syme's principles are right, there is no such thing as a safe method of chloroform administration. It is no longer a question of the superiority of the London method or of the Edinburgh method; absolute safety can be attained neither by watching the respiration nor the pulse for signs of danger—which are in either case proof of improper administration or of overdosing. Moreover, overdosing may take place whether the anesthetic is given on lint or on a towel, or on a cap such as we use, or with Junker's or Skinner's or any other form of apparatus. The all-important point is that the breathing shall never be interfered with in any way. Safety under chloroform can unquestionably be insured, but it can only be so by attending to regular natural breathing; and, whatever method is employed, no one can deny that it is the bounden duty of the chloroformist to maintain natural breathing throughout the whole period of administration. To maintain natural breathing requires careful training and considerable experience, but if those conditions be fulfilled it is impossible to produce anything with chloroform but anesthesia; and the Hyderabad Commission has shown that anesthesia alone is entirely free from risk," provided the drug is not pushed too far and that the patient is in ordinary health. We would prefer to make the last sentence read: "Anesthesia can be safely produced by chloroform."

SELECTIONS.

Summary of the Surgery of the Tendons.¹—WITH SIX ILLUSTRATIONS FROM LEJARS.—By JOHN B. HAMILTON, M.D., LL.D., Chicago.

The Surgery of the Tendons is the somewhat ambitious title laid down on the program as the title of my paper, but as time has not permitted me to review the whole subject in extenso, I will ask you to excuse my shortcomings, while I give you a condensed statement that will probably need no further condensation at the hands of your secretary. After all, I think most of the ideas that are new, or even presented in new form, can be stated in very few words.

I will pass over lightly the literature of the tendons, although that is very extensive, not only in medical literature, but in general literature. From the time Achilles' mother, Thetis, dipped him in the Styx, to make him immortal, by grasping tightly the tendons that have since borne his name, down to the middle ages when ham-stringing an enemy was a pleasant pastime of the bold Barons, the tendons have had considerable prominence.

In the surgery of modern war, where the wounds are made by bullets and rarely by bayonet or saber, the tendons are rarely wounded, for the very obvious reason that bullet wounds in battle are received on the body in exact proportion to the exposed area.

In civil life, the tendons are subject to dislocations more frequently than to wounds.

These accidents, though attended by few pronounced symptoms, need careful attention.

Contusions and punctured wounds need but scanty refer-

¹ Read at the Mitchell District Medical Society.

ence; owing to the lack of vascularity, contusion of a tendon is difficult to produce. Punctured wounds, when produced by any instrument causing laceration of the tendon fibers, are usually accompanied by retraction of the cut fibers and local necrosis of the connective tissue filaments; when made by a cutting instrument held parallel with the fibers, the wound closes with great rapidity, first by swelling of adjacent fibers from limited congestion; and second, by direct adhesion. The tendon sheath, however, made up as it is of loosely united connective tissue, is exceedingly liable to infection, and tenonitis, so called, almost invariably runs its course in the tendon sheath. Notwithstanding the fact that longitudinal wounds or separations of the tendons heal with rapidity, yet the resulting cicatrix impairs the efficiency of the tendon for a longer or shorter period, according to the area involved. Sometimes this impairment amounts to total suspension of the functions of the members; and the practical deduction from this lesson is, that in opening abscesses lying underneath a tendon, the incision should invariably be made at the side of the tendon, not through it. This practical advice, I believe, is not generally adhered to in the cases of finger abscess, but it should be. In abscesses of the fingers the original point of infection is usually the periosteum; the pus seeking exit in the line of least resistance usually projects from under the tendon on either side, and if the tension be not relieved at this time the adjacent bone participates in the inflammation, and it too becomes necrosed.

Fig. 1. Tendon Suture of LeFort.

Fig. 2. Tendon Suture Wöflar. After Lejars.

Fig. 3. Suture of LeDentu.

It has been mentioned that the cicatrix impaired the functions of the tendon. Not only does this impairment occur but, owing to the feeble circulation of the tendon, the absorption of the redundant cicatricial tissue is slow, and sometimes the impairment is permanent. The sheath and the adjacent connective tissue being richer in blood vessels, not only accomplish repair sooner, but they more speedily remove the cicatrix.

Theories of repair of tendons after injuries have undergone the same successive changes as that of repair of wounds elsewhere. The theory of Hunter, that the elements of the blood were alone concerned in the repair of tendons, held sway until Bouvier in 1857 asserted that the tendon sheath by proliferation filled up the interspace between the ruptured ends. Henle, Robin, Adams, Paget, Brodhurst, adhered to the theory of repair by exudation and direct union of the