

Naturally, the new malarial season once fairly started, the continuation of the epidemic is easily explained by the passage of the hæmosporidia from man to the anopheles and from the latter back again to man.

Finally, coming to the third subject of our researches—namely, the study of the localities in which the anopheles develop—it is necessary to recognise the fact that at Ostia, in spite of all the recent improvements, there exist all the conditions favourable to the production of the malarial mosquito. The district of Ostia is intersected by a dense system of canals which collect the water of the lowest levels and convey it to the elevating machines by which it is emptied into the sea. This system of canals consists of a main channel with secondary channels leading into it. Now in all the secondary canals, and in the part of the principal canal farthest from the machines, where there grows a rich vegetation of reeds and algæ, we found, from the beginning of June onwards, an immense number of larvæ of the anopheles. The larvæ were absent in the part of the principal canal nearest to the machines, where the vegetation was scanty or wanting, and where the action of the machines kept up a considerable current. Everywhere else, wherever the water moved very slowly or tended to stagnate—which in summer is almost the rule, because the machines on account of the low water-level only work in an intermittent way—the development of the larvæ was most vigorous. Another very favourite breeding-place of these larvæ is in the neighbouring small lake on the estate of Castel Fusano. We found larvæ very numerous everywhere from the beginning of June onwards till the middle of July, when they were fewer; and they were again numerous there and elsewhere in the Campagna, especially in the small lakes at Porto, in the months of September and October up to the time of the last rains. These facts explain in an entirely satisfactory way why the drainage works at Ostia, in spite of the removal of the marshes, have not, from a hygienic point of view, achieved their object.

And now, what practical conclusions can be drawn from these data with respect to measures of prophylaxis against the infection? These are abundantly evident. Indeed, everyone in Italy who has made a study of the subject in the light of these new researches has insisted from the first that the careful treatment of the malarial patient constitutes one of the principal tasks of social hygiene. We ourselves, in a short article published last April, were careful to note that the energetic treatment with quinine, from the commencement of the infection, of the æstival fevers not only reduced the dangers of relapses to a minimum, but had evidently a great importance for general prophylaxis, since the parasites by its means could be prevented from developing into those forms which continue their life in the anopheles. The treatment of the malarial patient ought, therefore, to be carried out systematically and energetically in the same manner as is practised when countries are invaded by other epidemic and contagious diseases. And yet we see here in Italy the peasants of our country districts suffering for years from the disease abandoned to themselves and continually disseminating the infection. This could be put to the test of experiment by treating all the patients in some circumscribed area systematically throughout the winter and spring from the date of their first febrile attack (thus reducing relapses to a minimum) in order to see subsequently what the behaviour of the infection might be in the new malarial season—that is to say, from the beginning of July onwards.

We urge that the treatment should be carried out energetically from the date of the first febrile attack because we know that quinine, although it can interfere with, and even prevent, the development of the young crescents, has no evident action on those already matured. Indeed, we have several times seen since July of this year that crescents from the blood of patients under the influence of quinine may complete their life-cycle in the anopheles in the normal manner.¹⁰ It is true that an ideal prophylaxis would be attained by removing the conditions which are necessary to the development of the anopheles in the malarial areas. Limiting ourselves to a consideration of the district of Ostia which we have studied we hold that such an object might, at least in part, be obtained by keeping the canals clear of the excess of

vegetation, by removing the obstacles which in some of them retard the flow of water, and by working the elevating machines more regularly so as to maintain a certain degree of velocity in the current. By these means the immense production of the larvæ of the anopheles might be at any rate diminished. But it is not our intention to occupy ourselves with the question as to the method best adapted for attaining the end in view; this is a task which does not concern us. We confine ourselves to indicating the fundamental points which ought to guide those who devote themselves to a practical solution of the problem, those being a knowledge of the localities in which the larvæ of the anopheles live and develop and of the reasons why they thrive in these places.

Note.—We have stated in the above paper that nearly all the quartan patients observed by us in the Hospital of Santo Spirito had contracted the fever in the summer and autumn—that is to say, under the same conditions in regard to temperature and surroundings as are required for the development of æstivo-autumnal and the greater number of tertian infections—and we have inferred from this that the anopheles must in general become infected with quartan parasites under the same conditions of temperature as those under which they become infected with crescents and the gametes of tertian fever. This is not opposed to the fact which has long been known that the majority of quartan cases are met with in our hospitals in the autumn and winter—that is, in the cold season. Indeed, if the history of these patients be closely inquired into it is found that in general they have been residing in the Campagna during the hot season and have taken the disease in a form at first not characteristic of the quartan type, but that subsequently a relapse occurred, occasioned by the cold weather, and that this relapse ran a typically quartan course.

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VOLKMANN'S ISCHÆMIC PARALYSIS; ITS TREATMENT BY TENDON- LENGTHENING.

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It is probable that few only are familiar with the title of this paper. Examples of the disease are happily rare, and the name is not to be found in our text-books. Yet the affection is one of serious gravity and hitherto has been found little amenable to treatment. I propose, therefore, to record a case in which great benefit has accrued from lengthening the contracted tendons, not only by enabling lost movements to be regained, but also by restoring the affected muscles to a seemingly more healthy state. In describing the case I hope to bring the salient aspects of the disease forward and to point out the causes which induce it. Volkmann's own account of it may then be considered.

A boy, aged four and a half years, was first brought to my notice and admitted under my care in St. Mary's Hospital on Nov. 5th, 1898. In August he had fallen on his left elbow and sustained a transverse fracture of the lower end of the humerus. Separation of the lower epiphysis was not, however, excluded from the diagnosis. For some inscrutable reason the limb was put up in the flexed position with anterior and posterior splints on the forearm. All seemed going on well the next day and the splints were not disturbed, but when the child was brought to the casualty room a few days later there was found to be total loss of power of the fingers and wrist. Immediately below the fold of the elbow the pressure of the end of the splint had caused a superficial slough, which became detached in the course of four weeks and left an open wound which healed by granulation. In the meantime the limb was secured on a single posterior splint, and when this was abandoned at the end of four weeks the parents were instructed to manipulate and rub the arm, for an increasing tendency to contraction of the fingers had been noticed and the wrist also was becoming more and more flexed. On one occasion under an anæsthetic a fruitless attempt was made to straighten the fingers and hand. Such was the story related to me.

When I first saw the child, three months after the accident, the arm was supported in a flexed position midway

greater pathogenic power of the æstival parasites, can be utilised for explaining, at any rate in part, the facts above mentioned.

¹⁰ The same facts have been noted independently by our colleagues Gualdi and Martirano.

between pronation and supination. The site and surroundings of the scar below the elbow were exquisitely tender, and there was tenderness of the whole anterior surface of the forearm also, so that the child could hardly bear to have his limb examined. There was extreme flexion at the wrist and the fingers also were flexed into the palm. So great indeed was the contraction of the flexor muscles that extension of both fingers and wrist was hardly possible even with much force, but the joints were clearly unaffected. Strong flexion of the wrist had but small influence over the extensibility of the fingers. Anæsthesia was partial only, involving the ulnar distribution in the hand, and there was some suspicion of wasting of the first dorsal interosseus. The flexor muscles felt peculiarly unyielding to pressure and decidedly firmer than natural. With the exception of the nervous affection which has been named I could detect no median, ulnar, or musculo-spiral paralysis, and I attributed the slight ulnar paresis to local injury to the ulnar nerve at the site of the fracture, and possibly to the pressure of callus which was to be felt at the lower end of the humerus. My own belief was that there had in reality been a separation of the lower epiphysis. An examination under an anæsthetic threw no further light on the condition, nor was the contraction of the flexors in any way lessened. That this deplorable state of things was in some way the result of splint pressure there could be no doubt, and in all its aspects the case was exactly like two other cases which I had had the opportunity of seeing.

This, then, being the state of the limb, and massage, electricity, and movement being each and all without remedial influence, it was thought that lengthening of the flexor tendons gave fair promise of benefit, and accordingly, on Dec. 20th, 1898, the tendons having been exposed by raising a rectangular flap immediately above the wrist were each in turn split longitudinally, severed to right and left at the opposite ends of each incision, extended, and sutured with the finest silk in the usual way. This part of the procedure was by no means easy. The diminutive size of the tendons in the small arm of a little child made it very difficult to fix the sutures, and I had slight hope that successful union would follow. I was not sure, moreover, that in every instance the divided tendon was joined to its own partner, and it is highly probable that a superficial flexor may have been attached to one of the deep tendons. This was quite unavoidable. In any case I thought that it would not materially affect the ultimate result. Exposure of the muscles enabled us to judge of their condition. They gave the impression of being stiff and unyielding, firmer than natural, and as if their structure had been changed—not at all as if they were affected by some spasmodic contraction of nervous origin. This, indeed, was their state as I have already endeavoured to describe it when estimated by manipulation through the skin, and it suggested that there really was some alteration in the physical constitution of the muscle fibres themselves and probably also of the connective tissue around and between them. The hand and arm were now secured in the flexed position, and the wound healed by first intention. After a fortnight we ventured to move the child's fingers and it then appeared that considerable union of tendons must have taken place. Tentatively, therefore, day by day, and each day with increasing boldness, both arm and fingers were extended, and at the same time massage and electricity were brought to bear upon the affected limb. Improvement was very slow and the patient left the hospital in February, 1899, with a feeling of disappointment on our part that it had not been more decided, but with the hope that much might yet ensue. The child was to attend as an out-patient for electrical treatment and from Dr. Wilfred Harris, under whose care he has since been, I learn that there was at first very decided reaction of degeneration, but that recently this condition is not so marked. Reference will be made again to the electrical phenomena when considering the causation and underlying structural changes of this remarkable form of paralysis.

I next saw the child at the end of July and was happy to note distinct signs of improvement. That, with the one exception of the forefinger, the tendons had united there could be no doubt; tenderness of the limb was much less and the wrist and arm could be extended without anything like as much associated flexion of the fingers as had been remarked when the patient left the hospital. The mother was urged to continue her attendance at the hospital with every hope of still further improvement. When seen again at the beginning of

October so great had been the progress that it was hard to believe that we had the same limb before us. All tenderness had gone. The forearm could be completely extended, the wrist and the hand could be voluntarily put in the extended position, flexion of the fingers was easy, and the little fellow could make a fist. The forefinger only was at fault, the flexor profundus alone acting on it. Furthermore, the muscles of the arm seemed to have lost that peculiar sense of resistance which had formerly characterised them. I had no longer any hesitation in holding out a much more favourable prognosis as regards the usefulness of the limb, believing that still greater improvement would surely be noticed as the limb in the natural process of growth increased in size. I feel justified, moreover, in placing the case on record now, sufficient time having elapsed to admit of a fair estimate of the result of operation.

Turning now to a consideration of the causes which induce this form of paralysis there is a consensus of opinion that in some way or other it is the prolonged pressure and fixation of the limb in splints which give rise to it. Thus Anderson¹ says: "It is a result of prolonged immobilisation of forearm fractures by any form of apparatus that intercepts the free circulation of blood through the muscles and nerves of the part." He seems in this extract to accept the view put forward by Volkmann as to the ischæmic origin of the affection and that it is not due primarily to a nervous lesion. On this point there is room for difference of opinion and the opinion of Volkmann himself may first, and very rightly, engage us. I find that the earliest reference made to it by him was in a footnote to a paper on inflammation of the muscles where he says²: "The severe contraction of the hand after too tight binding of the forearm after fracture belongs in part in all probability to such a process of inflammatory muscle-contraction and not to primary nerve-paralysis from pressure. The resultant *main en griffe*, as it is termed, has a very bad prognosis. The contraction of the fingers and hand remain so pronounced in spite of electrical treatment, passive motion, and forcible extension under an anæsthetic that amputation may in the end have to be resorted to. Complete recovery or even marked improvement I have never seen; and only in one instance, after unwearied trouble and long-continued treatment, have I noticed a diminution of the contracture and slightly better movement. The contracture comes on very rapidly and in a few weeks may attain to such a degree that the finger-nails bore into the flesh of the palm. In paralytic contracture no such acute contraction is ever seen." The next paper to which I will refer is by Kraske,³ a pupil of Volkmann, and since distinguished in other spheres of surgery, in 1879, on the changes found in transversely striated muscles after exposure to extreme cold. His observations are based on the microscopical examination of the muscles of two legs which had been amputated for gangrene the result of some hours' exposure to extreme cold. He described widespread necrobiosis of the contractile substance, complete loss of the nuclei in many of the primitive fasciculi, and the absence of striation and a granular appearance of the fibres. Reparative changes were also distinctly visible, and then he writes: "The appearance of the muscle tissue corresponds closely to the changes which are seen in the muscles of a limb which has been for a time rendered bloodless by circular constriction. The ischæmia, induced in the one case by lowered temperature and in the other by circular constriction, determines a partial necrobiosis of the contractile substance, to which is added some precedent inflammation in the perimysium internum and regenerative processes in the surviving muscle fibres."

It is unnecessary to dwell further on the microscopical changes which are to be found in muscles which have been deprived of blood. The clinical phenomena are here of more concern, and a précis may be given of the conclusions at which Volkmann had himself arrived and which are to be found in his paper on "Die Ischæmischen Muskellähmungen und Kontracturen."⁴ He there relates how the observation of a considerable number of cases in the course of several years had led him to believe that the paralysis and contracture, the result of too tight and too long fixation, were due to a direct effect upon the muscles themselves rather than to any nerve lesion. The following are his propositions: 1. The paralysis and contracture are of ischæmic origin and

¹ Fingers and Toes, p. 65.

² Krankheiten der Bewegungsorgane, 1875, p. 846.

³ Centralblatt für Chirurgie, 1879, vol. vi., p. 193.

⁴ Centralblatt für Chirurgie, 1881, No. ii.

dependent on the deprivation of arterial blood. The simultaneous venous congestion which is often seen appears only to hasten the onset of the paralysis. 2. The paralysis depends on the fact that the muscle fibres perish from prolonged want of oxygen. The contractile substance coagulates, breaks up, and finally disappears. Rigor mortis is in fact induced in the affected muscles. 3. It is especially characteristic that paralysis and contraction arise simultaneously, not as in paralysis of nervous origin where contraction is a later phenomenon and of gradual onset. 4. From the earliest moment and in quite recent cases the muscles assume the state of rigor mortis and there is the greatest obstacle to extension of the limb. 5. By contraction of the material thrown out in the attempts at repair the rigidity and contracture of the limb are made worse. 6. Ischæmic paralysis and contracture of the same nature are also seen to follow the application of Esmarch's bandage, injury to the greater vessels, and exposure to extreme degrees of cold. 7. The degree of paralysis and contraction depends upon the length of time that the blood-stream has been arrested. If the ischæmia has been extreme the most serious consequences may follow in half a day, or even less, while symptoms of a high grade of severity may result from slight constriction and pressure of several weeks' duration. This is most common after fractures of the radius. 8. The prognosis is bad, and in the worst cases involving the hand and fingers the condition is absolutely irremediable. It is better in the case of the lower limb, for here tenotomy may be practised. The slighter cases may be benefited by energetic treatment with massage, &c. 9. Mechanical treatment is practically useless. In recent cases one would endeavour to extend the muscles under an anæsthetic; but in those of long standing any attempt to do so would be found to break the bones or tear the tendons before the muscles would yield.

So far Volkmann, in unavoidable abbreviation, and with the single exception of Anderson's work already referred to I have failed, even with the invaluable help of Mr. Plarr, Librarian of the Royal College of Surgeons, to find aught else in medical literature upon this interesting subject. That this singular form of paralysis is due to the combined influences of pressure, fixation, and ischæmia there can hardly be a doubt, nor is there any doubt that my own case was an example of it. The rarity of the condition is probably one reason why so few cases have been placed upon record; but it is not unwarrantable to assume that some have not seen the light because, in the words of Anderson, the paralysis is "a reproach to surgery, since a careful observation of the hand and fingers during the use of splints will always give due warning of the danger."

It is not, however, by any means so certain which are the structures upon which continuous pressure exerts this injurious influence. In the observation of my own case I could never believe that it was simply a nerve lesion with which we had to do. The slight ulnar paresis was obviously the result of a trifling injury to the ulnar nerve at the site of injury to the bone, and there was no reason for thinking that the pressure of the splints could have been specially exerted upon this one nerve. If upon any, the median was surely more exposed to pressure, but there was no special evidence of injury to it, and as pointed out by Volkmann the simultaneous origin of the palsy and the contracture was altogether unlike the effect of gross lesion to a nerve trunk. But the electrical observations of Dr. Wilfred Harris show indisputably that nerve elements had not escaped injury. After describing the reaction of degeneration which he discovered, he says, "Any ischæmic condition would, I imagine, damage the nerve-end-plates as well as the muscle and one might expect, I should think, some reaction of degeneration." Mr. Anderson tells me that this view coincides exactly with his own as given in a previous quotation. Dr. Harris adds in a later communication: "The fact of the great reduction to faradism in the flexor muscles when I first saw the case suggested to me a nerve lesion, perhaps intra-muscular, and does still, since in acute myositis there are no electrical changes until the late stages when atrophy, if any, sets in. Erb, too, states positively in his book that primary muscular conditions do not cause reaction of degeneration, which is in his opinion proof of nerve lesion. But I know nothing of ischæmic myositis and probably Erb did not either and the reactions may very well be a part of the condition as you suggest. The obvious answer to the difficulty would I think be supplied by experiment." I have

ventured to repeat here what Dr. Harris has written to me, for he speaks with an authority on such matters to which I cannot pretend. But nothing that he tells me is, I believe, opposed to the view which I would provisionally hold as to the causation of this particular form of paralysis and contracture, that it is not primarily or even in the main due to a nerve lesion but that it is due to the combined effects of pressure, immobilisation, and greatly diminished blood-supply therefrom upon the muscular elements, the connective tissue elements, and the nervous elements which are met with in every muscle throughout the body. That pressure alone, or immobilisation alone, or deprivation of blood alone could bring about the results which are seen is not nearly so likely as the combination of all three, and there is no part of the body on which they can be so readily brought to bear as in the case of the forearm bound up and negligently left bound up after fracture. May the publication of this case be the means of making the evil consequences of such ill-treatment more widely known.

Lastly, as to the tendon-lengthening which was practised here. It seemed to me that it offered the best chances of amelioration of the distressing condition, and from personal experience of it in other instances and for other purposes I felt that the operation was one upon which we might rely. Originally performed and recommended, I believe, in this country by Anderson, and a year later in America by Keen, the operation has become a recognised procedure of much value in surgery, and has doubtless been used by many surgeons. Keen⁵ employed it in a case of post-hemiplegic contracture of the flexors of the fingers in a lady 25 years of age, and lengthened four tendons of the deep flexors, five of the superficial and those also of the two carpal flexors, of the flexor longus pollicis, and of the palmaris longus with some measure of success; and Anderson⁶ records a case of a girl, aged 17 years, with flexor contraction the result of some injury to the arm in childhood for whom he lengthened the tendons with much benefit. I am further indebted to my friend Mr. Anderson for the opportunity of reading the accounts of two other cases by Dr. Colgan and Dr. Wilson respectively, but they have no special bearing upon the subject of tendon-lengthening in such a condition as was seen in my own case. Akin to it, though owning a different cause, were the cases of Anderson and Keen; but there is, I believe, no case on record in which it has been practised in Volkmann's ischæmic paralysis. The result has far exceeded expectation, and I take it that the great improvement is due not alone to the mere fact that the tendons have been made longer, but that by their lengthening an increased range of movement has been made possible in the affected muscles. Muscles are meant for movement, and movement is an essential element in the treatment of any such affection as the peculiar contracture seen in this disease. Massage and electrical stimulation are also without doubt of inestimable value, but they are likely to fail of their purpose unless some movement is possible at the same time. At any rate, speculation apart, the result of the operation in this instance has been so satisfactory as to justify me in commending the adoption of the same method to the notice of surgeons.

HYPOTHERMIA.¹

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A MAN, aged 36 years, married, the father of three children, was admitted into University College Hospital under Dr. Charlton Bastian (whose clinical clerk I happened to be at the time, and for whose kind permission to make use of the notes of the case I am deeply indebted) on April 16th, 1887, complaining of drowsiness and occasional frontal headache of about two years' duration. He had had scarlet fever when he was a lad and soft chancre ten years before coming under observation, but he denied syphilis. There was no history of nervous disease in the family. He

⁵ Transactions of the College of Physicians of Philadelphia, 1891.

⁶ Loc. cit., p. 58.

¹ Being a contribution to a discussion on Temperature in Health and Disease, read at a meeting of the Windsor and District Medical Society on Dec. 20th, 1899.