cerebral artery—the chief branch of the internal carotid—is directed was much softened, readily breaking down into a pulp, and particularly so was the left corpus striatum. The other parts of the brain, and likewise the vessels at its base, were found quite healthy in appearance.

There was nothing worthy of note to be mentioned concerning the other organs of the body.

REMARKS. This case presents some points of interest both in a physiological and in a pathological sense. It appears clear that the arteria innominata was blocked up at the time of the admission of the patient into the Hospital, and for the reasons that the pulse was then imperceptible at the wrist, and that the clot itself was evidently not of recent formation. Consequently the functions of the brain were duly carried on, notwithstanding that its supply of blood through this artery was cut off, and that not more than one-fourth of the due amount of blood could have passed through the opening of the left common carotid. Also it is evident, that for the last sixty hours of life the only supply of blood which the brain received was that which passed through the left vertebral artery. The cause of the formation of the clots in the arteria innominata, and in the left cerebral artery, must be, to a certain extent, matter of conjecture. There were no local alterations perceptible in the vessels themselves which could give any account of clots. Both vessels, to all appearance, seemed perfectly healthy; the arterial branches at the base of the skull and around the obstructed artery were quite natural to the eye. Perhaps the existence of the aneurisms may give us a clue to the obstruction of the arteries. It is not unfair to presume that it was very possible for some broken down portions of fibrin to have been washed out of one or other of the aneurisms at the root of the aorta, and to have been carried along in the current of blood until arrested in their progress, acting then as a ligature of the artery, and causing behind it the formation of a firm fibrinous coagulum. This supposition is strengthened by the fact that the mouths of the aneurisms were large. There were no fibrinous beads around any of the heart's valves, so that the carrying away and arrest of one of these in the circulation could not have caused the clots, after the manner described by Dr. Kirkes. The softening of the brain was clearly secondary to the arrest of the circulation. In this case the supply of blood to the left middle lobe of the brain must have been wholly cut off; there could have been no compensatory supply to it through the right carotid artery.

To the practitioner, also, this case of paralysis, like so many others of its kind, offers a word of fruitful warning. What would a vigorous treatment have done here? What could any interfering treatment do?

Original Communications.

THE ATROPHY OF LIMITED GROUPS OF MUSCLES.

By F. BRITTAN, M.D., Physician to the Bristol Royal Infirmary, and Lecturer on the Practice of Medicine in the Bristol Medical School.

[Read before the Bath and Bristol Branch, February 19th, 1857.]

IN the *Dublin Quarterly Journal of Medical Science*, for November, 1856, Dr. Reade, of Belfast, published a paper, entitled, "Contributions to the Pathology of the Spinal Marrow." In it he relates three cases to illustrate "a diversity of morbid action arising out of disease or injury of the same portion of the spinal marrow." The three cases are: 1. "Symmetrical muscular atrophy." 2. "Paralysis of the body beneath the phrenic nerves, from concussion." 3. "Paralysis of the body beneath the phrenic nerves during dentition." The first of these cases is adduced in illustration of the views of M. Cruveilhier on muscular atrophy, announced to the Institute of France, December 1855, and published in the *Archives Générales*, January 1856. The conclusions at which M. Cruveilhier has arrived may be thus summed up:

That there is a species of muscular paralysis, sometimes partial, sometimes general, which gradually and successively invades, fasciculus by fasciculus, and fibre by fibre, the voluntary muscles, leaving all the functions of nutrition intact, excepting that of muscular nutrition.

That this gradual muscular paralysis is the consequence of

progressive atrophy of the anterior roots of the spinal nerves, and equally progressive atrophy of the corresponding muscles.

That this "*paralysie musculaire atrophique*" is analogous to that which results from section of muscular nerves.

That the facts appertaining to this form of paralysis confirm the theory of Sir Charles Bell.

And that these observations demonstrate a previously unknown influence of the anterior roots of the spinal nerves on muscular nutrition. These conclusions, M. Cruveilhier states, are founded on a great number of clinical facts; and they are preceded by a declaration so essentially French, that I cannot refrain from quoting it in the original. "Le hasard a voulu que j'aie été le premier à qui il ait été donné de faire l'anatomie pathologique de cette maladie; c'est là tout le secret de la part que j'ai prise à sa détermination, dont je ne crains pas de le dire hautement, parceque c'est la vérité; la priorité ne saurait m'être contestée."

I have lately had two cases analogous to the first in Dr. Reade's paper, under my care at the Infirmary; but as they offer certain important differences as to the history of the patients and early conditions of the disease, I think it may be worth while to publish them, with a few remarks; more especially as Dr. Reade seems to think them rare, or at all events to have been able to find but one recorded.

Dr. Reade's case is that of a young man, aged 19, nearly six feet high in his clothes, apparently a specimen of robust health and excellent constitution. Eighteen months previous to examination he had been distinguished amongst his companions in all athletic exercises, and well proportioned in his muscular development. "The first sign of the approaching disease was a degree of stiffness or difficulty of executing the motion of putting on and removing his hat" (in other words, of raising his hand to his head); never suffered pain, and has enjoyed uninterrupted health in all other respects. "When he stripped his body to the waist, he exhibited neck, chest, and arms to the elbow-joints reduced to the most abject degree of emaciation; this was symmetrical; the greater and lesser pectorals were little more dense than the strongest brown wrapping paper." The muscles of the neck, scapulae and humeri, were reduced to the mere elementary outline of muscles; whilst from the elbows, the forearms and hands displayed the full development of a robust and vigorous man, with all the concomitant power, sensibility, and aptitude for use. He was under treatment for two years. A seton in the neck, the use of dumb-bells, and electro-magnetism were tried, but without material improvement. Six years later he was again inspected, and was found decidedly improved; the muscles more developed. The muscles of the neck have been fully restored, and the others considerably augmented and perfectly obedient to the will. The forearm and hand have lost much of their former bulk; but his occupation for the six years has been sedentary. His health has been uninterruptedly good.

The analogous case is quoted from Rokitsansky. A labourer, aged 45, much exposed to wet and in the habit of allowing his clothes to dry on him, was attacked with pain in the left shoulder, which was most severe about fourteen days after it commenced. There was no swelling, redness, numbness, nor tension; but great pain when the arm was raised with the other hand: he could not lift it without. After the first week the shoulder was found wasted; as the pain subsided the wasting advanced, till the deltoid, supraspinatus, infraspinatus, and teres muscles seemed completely absorbed. The shoulder-joint was healthy, and there was no emaciation of the forearm or hand.

Dr. Reade regards this case as one of muscular atrophy from abeyance or destitution of the function of nutrition; and he states "these three cases illustrate a diversity of morbid action, arising out of disease or injury of the same portion of the spinal cord."

He also alludes to two other cases within his own cognisance; one of atrophy proceeding from the shoulders to the feet, accompanied by paralysis, and a second confirming Cruveilhier's statement of the pathology of this paralysis, the same morbid changes having been found in the roots of the anterior spinal nerves. We thus have muscular atrophy, partial or general, according to Cruveilhier, depending on atrophy of the anterior roots of the spinal nerves. Muscular atrophy partial, according to Dr. Reade, depending on imperfect nutrition, the result of disease of the spinal cord.

My two cases more accord with that quoted from Rokitsansky, and show, I think, that muscular atrophy, with of course necessarily a quarter or less degree of loss of power, proportionate to the amount of atrophy, may sometimes depend on

another cause, or at least appear to spring from a different origin, and be connected in some way with rheumatism and the rheumatic diathesis. They are as follows:—

CASE I. William Spencer, cotton-spinner, aged 24, was admitted under my care at the Bristol Royal Infirmary in November last. His history was as follows. Four years ago he was discharged from the 2nd regiment of foot, on account of an attack of rheumatism, for which he was in Hospital seven months. His joints were then much swollen. Three years since he felt aching pains in the left shoulder, with stiffness and inability to move it freely; these symptoms gradually increased, and, *pari passu*, the shoulder became more emaciated; he has, however, continued to work at the cotton factory until very lately.

He is now excessively emaciated all over the body as regards the muscular structures, the joints being very prominent, and the muscles feeling like mere cords. His ordinary position is with the elbows flexed to a right angle, the forearms supinated. He states that he can move his arms better at night, especially after exertion during the day; the attempt to straighten the arm gives pain. He can raise the arms without pain, but has not power to lift anything. His general health is good. The first sound of the heart is rather harsh and rough. He perspires excessively in his limbs, particularly on the thumbs, and most remarkably after the galvanism, which was tried without benefit. He left scarcely, if at all, improved.

CASE II. Charles Webber, aged 23, short, but healthy looking, and apparently well made, became an out-patient, October 1856. By trade he is a shoemaker, and always enjoyed good health. He was lately a corporal in the 17th foot, from which he was discharged on account of his present disablement. Six months since, whilst on duty at Limerick, he was exposed to very heavy rains, and continually wet; on rising one morning after exposure could not walk, on account of pain in his right hip, knee, and ankle joints, which then began to swell. He was confined to bed for fifteen days. After the first seven days the pain passed to his shoulder and side of the head, leaving his hips and legs. Soon after the pain reached his arm he found the flesh between his shoulder and elbow diminishing, on the right side first, then on the left. In two months the arms were no larger than his wrists, and he could not lift them to his head. This continued for six weeks, when they began slightly to increase again, and he felt more power in them.

Present condition. His arms, from the neck to the elbow, are less than half of the natural size, all the shoulder and humeral muscles being much diminished; the biceps only seeming of anything like the natural size. There is very little deltoid, and the triceps feels like a mere band. His appearance exactly coincided with that of Dr. Reade's patient, as shown in the drawing that accompanies his paper. He cannot raise the arm well, but can flex the forearm on the arm firmly. He had no particular treatment in his regiment; nothing but liquor potassæ. He had syphilis three years ago, and a few spots came out as the rheumatism appeared. He was under treatment for a month, principally with iodide of potassium, and much improved.

I should feel great diffidence in suggesting doubts as to the correctness of M. Cruveilhier's assertions, supported as they appear to be by the observation of the morbid changes, described in the *post mortem* examination of the corresponding case, alluded to by Dr. Reade, did I not conceive that the cases now reported bore me out in the supposition that the cause alleged by Cruveilhier is at least not the only cause of this curious disease; but there are many other objections to this theory to which these cases direct our attention.

It will be observed that in Dr. Reade's case, and in my own, especially the second, there are several common peculiarities.

1. The symmetry of the morbid action.
2. The localisation of it to particular groups of muscles; for example, those of the shoulder and humerus, and, to some extent, the biceps; whilst the muscles of the forearm remained unaffected.
3. The uniform commencement of the atrophy in the upper parts, and progress downwards.

4. The absence, in each case, of more paralysis than must necessarily be the result of such a degree of atrophy; in fact, it is not paralysis, but loss of power from want of muscle.

Each of these, independent of all other considerations, appears to me to afford strong objection to either of these alleged causes of the morbid actions. For,

- 1st. They require that we assume the anterior spinal roots to be exactly symmetrically affected on either side.

2nd. With a knowledge of the extraordinary complexity of the arrangement of the filaments forming the anterior roots of the cervical nerves, more especially the different distributions and variety of muscles supplied by branches from the fifth, sixth, seventh, and eighth roots, it is almost impossible to conceive atrophy of these anterior roots, affecting only limited groups of the muscles, to all of which these branches proceed; for instance, how can the scapular muscles be affected with paralysis and atrophy, caused by atrophy of the spinal roots, through which their nervous filaments pass, without involving also the phrenic nerve; or of the deltoid and humeral muscles without corresponding affection of those of the forearm and hand, whilst the subscapular, musculo-spiral, and circumflex nerves may be traced through the plexus to the 5th, 6th, 7th, and 8th roots, and the ulnar and median to the same.

3rd. If we allow Dr. Reade's view, that these cases depend on imperfect nutrition, the result of disease of the spinal cord, which involves the influence of the anterior roots—in other words, M. Cruveilhier's theory—we must suppose, to explain these cases, that this function of the cord is so located and circumscribed that disease of its structural instruments may produce its effect without inducing paralysis; or assume (as Dr. Reade states his case proves) that, as the result of organic lesion of the spinal marrow, "muscular atrophy may exist without paralysis, as paralysis of motion may exist without disease of sensation or the converse."

4th. The absence of complete paralysis in each case, and the proportion between the wasting and loss of power, seem to evidence that the loss of power is more fairly to be ascribed to want of nutrition and loss of substance, than to loss of nervous energy.

In fine, I cannot help thinking that the phenomena of these cases give no support to the views of M. Cruveilhier or Dr. Reade; and without considering the evidence of the two I have related, and that of Rokitsansky, as sufficient to prove any connection between this curious condition and rheumatism, they do, I think, afford reason to induce us to lean to the side of analogy, and look for some more general or diathetic reason to account for such peculiarities of mal-nutrition. I should add that there was no evidence to lead to the notion of lead affection, nor, as far as I could learn, had mercury ever been largely administered in either of the cases I have reported, though "spots," probably syphilitic, appeared with the rheumatism in the case of Webber.

THE "HYDATID FREMITUS" OF PIORRY.

By W. J. LITTLE, M.D., Senior Physician to the London Hospital.

I RESPOND, with much pleasure, to Dr. Markham's invitation to contribute my quatum of experience as to the value of the "son hydatique", or the "hydatid fremitus", of Piorry. After perusal of Piorry's treatise, entitled "*Exploration par la Percussion médiate*", I, like Dr. Markham, was inclined to consider that author's account of his *son hydatique* as a pedantic refinement of symptomatology. About 1840, however, I visited, late one evening, in consultation with Dr. Langmore, of Finsbury Square, a gentleman, aged 36, who presented, with other signs of hepatic derangement, an enormous abdominal tumour which had commenced in the right hypochondrium. A recent and sudden aggravation of symptoms occasioned the consultation. I discovered the presence of the "*son hydatique*" of Piorry, and realised the truth of Piorry's description, as quoted by Dr. Markham, "*La sensation que le doigt éprouve et que accompagne le bruit (humorique) peut être entièrement comparée à celle que donne une montre frappée sur la surface opposée au verre.*" Upon the strength of this sign, I suggested that the tumour was probably due to hydatids. I was subsequently informed by Dr. Langmore that the same night the patient discharged by vomiting a *païful* of them. Some months later I saw the individual, without signs of tumour, in tolerable health, although very spare in habit. I believe that he sank two years afterwards from the same disease.

With my present experience of hepatic tumours, I should, in such a case as that above related, rely much upon the great size of the more or less obscurely fluctuating mass in diagnosing its hydatid origin. I have never witnessed a perfectly similar case.

I once had under my observation an unmarried woman, aged 33, affected with a single hydatid or serous cyst of liver, causing abdominal enlargement equal in size to fully advanced