ON THE REFLEX FUNCTION OF THE BRAIN: A CORRECTION OF DATES.

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THE leading article in the JOURNAL of May 16th, "On the Comparative Study of Drunkenness", was instructive as well as personally interesting to me. It is a favourable omen when a general fact is made so practically available to diagnosis. As to the special points discussed, I would only add that injuries to the head, especially to the basilar region, may be mistaken for drunkenness, as they sometimes are, since they cause very similar symptoms. The facts of "reduction" have been long applied by me to mental pathology under the term "reversion".

I write, however, more especially to ask the favour of space in the JOURNAL for the correction of an error as to the date of my first introduction of the Reflex Function of the Brain to the profession. Dr. Carpenter, as quoted in your leader, has always fixed 1844 as the date, which is an error that I ought perhaps to have corrected sooner. It is true, that on September 28th, 1844, I read a paper "On the Reflex Function of the Brain" to the medical section of the British Association for the Advancement of Science, then meeting at York. But, on reference to that paper (which, with an important appendix, was printed in the January number of the *British and Foreign Medical Review* for 1845), the following introductory sentences will be found.

"Four years have elapsed since I published my opinion, supported by such arguments as I could then state, that the brain, although the organ of consciousness, is subject to the laws of reflex action, and that, in this respect, it does not differ from the other ganglia of the nervous system. I was led to this opinion by the general principle that the ganglia within the cranium, being a continuation of the spinal cord, must necessarily be regulated, as to their reaction on external agencies, by laws identical with those governing the spinal ganglia, and their analogues in the lower animals. And I was confirmed in this opinion by finding, after the investigation and collocation of known facts, that observations and arguments like those satisfactorily adduced in proof of the existence of the reflex function of the spinal ganglia, may be brought forward in proof that the cerebral ganglia have similar endowments. In the present paper, I propose to give these proofs connectedly." (*British and Foreign Medical Review*, vol. 19 (1845), p. 298.)

The "four years" that had elapsed since I called special attention to the subject, referred to the date of my treatise on the *Nervous Diseases of Women*, which was published in 1840, and which contains a chapter (p. 105) headed "The Instinctive Actions in Relation to Consciousness: the Brain subject to the Laws of Reflex Action".

But, in fact, this book was virtually a second or systematised edition of researches and conclusions regarding the various diseases of the nervous system peculiar to women, which were published during 1838-39 in successive numbers of the *Edinburgh Medical and Surgical Journal*. At that time, I was House-Surgeon to the York County Hospital, and began originally with a series of clinical observations and inquiries having in view to determine what were the special kinds of mental and cerebral disorders to which young women were liable. My primary object was to discover the true nature of the so-called mesmeric phenomena which the two sisters named O'Key were manifesting under the manipulations of the late Dr. Elliotson, and to this end it was necessary to ascertain how far they were cerebral and how far simulated. In this way, I accumulated a series of clinical observations of cases of hysteria in all their anomalies, and examined both the cerebral and the psychological phenomena of them in their relations to the anatomy and physiology of the nervous system. My inquiries into the diastaltic influence of the uterus and ovaries on cerebral and mental states led me to develop the laws of evolution in reference to both mental physiology and pathology; and I thus arrived at results which include much more than the theory of the reflex function of the brain, together with its applications. Some of these, as to evolution and reversion, were published in the Appendix to the paper read at York.

The then exceeding difficulty of these inquiries cannot be appreciated by the present generation of physicians and physiologists. Not only was it universally held that the mind or consciousness was the cause of all mental states, but, at that date, Dr. Carpenter, together with Alison, Müller, Reid, and others, strongly maintained that sensation was a cause of certain movements—*e. g.*, that pain caused the cries and writhings significant of pain. On the other hand, Marshall Hall had fixed the limit in the encephalon of his true spinal system to be at the tubercula quadrigemina; all above (he held) was the seat of the "soul", and beyond the range of physiological research.

The analytical essay in the *Edinburgh Medical Journal* for July 1839, but dated October 10th, 1838, contains many illustrations of my conclusions. I quote a passage in illustration of the views as to evolution of energy by which I worked.

"20. If we would obtain a large and definite knowledge of the action of force upon matter and intelligence, in exciting the phenomena of life and thought as displayed in man, we must examine the laws of its action as exhibited both in every living organism and in the molecular changes of inorganic matter. A thousand circumstances assure us that, between these last and the highest efforts of the human intellect, there is a continuous chain of phenomena, although we have been unable to follow it link by link" (p. 10). And I then give illustrations of evolutionary relations.

The subjoined will illustrate what is now termed *automatic activity*. "Since external stimuli produce such changes in the nervous centres that the involuntary motions termed reflex follow—Can they produce analogous sensorial phenomena?" I adduce certain considerations in proof, concluding thus: "It will be easy to show that the excitatory phenomena of Dr. Hall, confined by him to the spinal cord, have their analogues in the cerebral hemispheres. In short, that there is a large class of combined involuntary sensations or ideas which are excited secondarily from without, and may be termed excitatory, if Dr. Hall's nomenclature be followed." (p. 17).

Dr. Maudsley, in his valuable work on the *Physiology and Pathology of Mind*, claims for the late Dr. Griesinger priority in regard to the reflex function of the brain, who, it appears, published a paper in the *Archiv für Physiologischen Heilkunde* for 1843, entitled "Ueber psychische Reflexactionem mit einem Blick auf des Wesen des psychischen Krankheits". I have never seen this paper, but I can affirm that, when I was in Germany in 1839 (and I graduated at Göttingen in that year, my papers in the *Edinburgh Journal* being accepted as a thesis), I found German physicians familiar with my views. My treatise was also subsequently reviewed in German periodicals; so that Griesinger could, if he thought right, use my researches to elucidate his own. As applied to mental diseases, in his systematic work, they are, however, being metaphysical, unlike mine.

I venture to trespass further on your space as to another class of morbid phenomena, namely, those caused by the *will*, *attention*, the *imagination*, and *suggestion*, and which are so common in mesmeric and like experiments. In the article on Reichenbach's and Gregory's researches referred to in your leader, and of which I was the author, I develop my principles as to these points; but then they were equally, although not so clearly, set forth in the essays and the treatise I refer to above. As to the morbid influence of attention on bodily organs, I certainly anticipated my late valued friend Sir Henry Holland, who had not published his views when I wrote in October 1838.

In his excellent work on *Mental Physiology*, lately published, Dr. Carpenter develops certain theories of the influence of the will and of attention on the "cerebrum," as he names the "hemispherical ganglia" of Solly and others. I do not concur with these theories, having learnt their weak points; but I would observe here, that I discuss them both in the papers and in the treatise referred to. In the latter, there is a chapter (p. 109), headed, "The Action of the Will, and of Internal and External Stimuli on the Hemispherical Ganglia," which is, for the most part, a *verbatim* reproduction of the views I published in July 1839, in the *Edinburgh Medical and Surgical Journal*. A short extract will serve to indicate how definite my inquiries were.

"184. From the preceding statements [as to the reflex motor action of the brain], it is obvious that changes in the sensorial fibres of the encephalon may originate internally as well as externally, and excite movements. Tickling the fancy will excite laughter as much as tickling the feet. In fact, it may be hypothetically supposed that there is a surface on which sensorial fibres terminate, connected with ideas, and which is analogous to [that of] the sensitive fibres on the skin and mucous membranes. It becomes a matter of some importance to inquire how far the will can act on sensorial fibres in general, and whether external or internal stimuli, acting upon them, can excite sensations, emotions, and ideas, simple and compound, corresponding to the whole class of excitatory phenomena." (*Treatise*, p. 109).

I then adduce various instances of this action of the will. I show that new sensations can be thus excited, and that "the phenomena of mesmerism (so-called) are all illustrations of the power of the will over the brain." As to attention, I add, however: "An act of attention may be, and often is, involuntary and instinctive, when it is to be classed with the conservative, or rather with the excitatory phenomena. Point at a nervous female, and she will complain of a sensation as if cold or warm air were blowing upon her. [I repeatedly thus induced this "aura" in hospital patients.] The sensation probably depends upon changes in the central terminations of the sensitive nerve,

excited by the act of attention." Of a similar character are many sympathetic phenomena."

In making these corrections as to dates, I by no means wish to do more than set right what appeared to me to be oversight. I have no doubt Dr. Carpenter is sincerely anxious to do me justice, and I am sure no one has so clearly and fully developed my views as he has, and so has greatly facilitated the research. I hope, when I can secure leisure, to develop my views in relation to the will.

THE RELATION BETWEEN ARTERIAL TENSION AND ALBUMINURIA.

By GEORGE JOHNSON, M.D., F.R.S.,

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I LEARN from Mr. Mahomed's paper in the last number of the JOURNAL, that I had mistaken his explanation of the relation between arterial tension and albuminuria. I now understand his theory, but I cannot accept it as a satisfactory explanation of the facts. While I admit that there exists a physico-chemical, or, as some would say, a vital attraction, between the tissues and certain of the blood-constituents, I deny that Dr. Carpenter or any other physiologist has demonstrated the existence of a "capillary power", the mere diminution or suspension of which would so increase arterial tension as, when long continued, to cause hypertrophy of the muscular walls of the minute arteries. This hypothesis assumes the existence of a mysterious "capillary power", while it ignores the regulating influence of the muscular arterioles. And yet this regulating influence has been conclusively demonstrated by Bernard, Brown-Séquard, and others; and the doctrine has met with very general acceptance. We believe that, while the propulsion of the blood, and its regulated distribution to the various tissues and organs, are effected by the muscular contraction of the heart and of the arterioles under the influence of the vaso-motor nerves, the interchange of materials between the blood and the tissues during the processes of nutrition and secretion are the result of subtle chemico-physical or so-called vital attractions.

A true theory must be consistent with all ascertained facts. There are many facts which are not consistent with Mr. Mahomed's hypothesis. Let the following suffice for an illustration. In no cases of chronic Bright's disease is there greater arterial tension, in none are the minute renal arteries more constantly and conspicuously hypertrophied, than in cases of the small red granular kidney. This state of the arteries would, according to Mr. Mahomed, imply a great increase of capillary engorgement and pressure, and therefore a copious transudation of albumen; yet we find that in many of these cases the amount of albumen in the urine is comparatively scanty, and it may be entirely absent. These facts are intelligible if we admit that the contraction of the hypertrophied arterioles lessens the pressure on the capillaries in front; they are intelligible on no other hypothesis.

That a merely passive distension of the minute arteries, consequent on an obstruction in front, is not sufficient to cause hypertrophy of their muscular walls, is shown by the fact repeatedly observed by Dr. Kelly and myself, that in cases of chronic incompetence of the mitral valve, involving long continued passive engorgement of the lungs, the muscular walls of the pulmonary arterioles present no appearance of hypertrophy. More than twenty years ago, when I first discovered the hypertrophy of the renal arteries in the granular kidney (*Med.-Chir. Trans.*, vol. xxxiii, p. 107), I endeavoured to explain the phenomena by a hypothesis similar to that which Mr. Mahomed now adopts; but I soon found that my explanation was inconsistent with pathological facts and with the established doctrines of physiology.

The main difference between Mr. Mahomed and myself is this. He believes that the increased arterial tension in cases of Bright's disease is the result of an impediment in the capillaries—a loss or diminution of "capillary power". I, on the other hand, maintain that it is mainly due to the resistance offered to the passage of the blood by contraction of the muscular arterioles under the influence of the vaso-motor nerves.

With reference to Mr. Mahomed's concluding remark, I would suggest that there is the same "unity of action" in the propelling heart and the regulating arterioles as in the detrusor urinæ and the sphincter vesicæ; the same, too, as in the muscular coats of the large intestine and the sphincter ani. It is incredible that the muscular walls of the arterioles have no other function than that of resisting the pressure which results from a diminution of "capillary power" in front.

Mr. Mahomed's explanation of the pathology of acute Bright's disease appears to be this. Poisoned blood destroys the attraction between the blood and the tissues. The result is an increase of arterial tension; then follows congestion of the capillaries, and, "from the fact of in-

creased blood-supply, increased cell-production". Hence, according to this doctrine, an increased blood-supply to the tissues results from the suspension of the "capillary power", which is believed to be a motor force; and an increased tissue-formation is determined by a diminution of the vital attraction between the blood and the tissues. Surely these propositions are inconsistent with each other, and the supposed physiological mechanism would not work.

With regard to the proximate cause of albuminuria, it is unquestionable that blood-serum may transude through the walls of the Malpighian capillaries as a direct result of excessive pressure on the walls of the vessels, whether caused by an increased afflux of blood through the arteries, or an impeded efflux through the veins; but there is also good reason to believe that, while certain morbid materials are passing from the intertubular capillaries into the gland-cells, modifying their nutrition and causing a rapid desquamation, the same morbid condition of blood may result in an abundant passage of serous fluid through the Malpighian capillary walls, apart from mechanical pressure either on the arterial or the venous side of the capillaries. I have no doubt that the albuminuria and the intratubular structural changes which are characteristic of acute Bright's disease, are the result of something more than mere mechanical pressure on the walls of the blood-vessels.

CASE OF HYDROPHOBIA IN A CHILD.

By F. RHODES, M.D., Bradford.

CASES of hydrophobia have hitherto been so unfrequent, that few medical men have seen more than two or three cases, while the great majority have not even seen one. Notwithstanding this, it is apparent that the disease is now sufficiently common to make "Painful Death from Hydrophobia" a very constant heading in the newspapers, and to cause the intractability of the disease to be a most serious consideration.

At the onset, the diagnosis is no doubt difficult, from the rarity of the disease, and thus appropriate treatment is delayed until the pathognomonic symptom—dread of water—is fully developed. This, however, eventually comes on, the history of the bite is revealed, and the case draws quickly to a conclusion, giving scant opportunity to the physician to employ his "remedies".

The reports of cases of hydrophobia should especially state in what way the original wound was treated, and how long it was after the bite before such treatment was adopted. The following case, which occurred in my practice recently, may be of interest.

On the evening of January 27th last, I was called to see a little boy between sixteen and seventeen months old. I was informed that he cried, and had a "shivering fit", when moved. I examined him carefully, to detect tenderness about the spine and other parts, but did not succeed in finding any; yet, upon the slightest movement, he started and cried. The child would not drink, and, supposing that there might be disease of the cervical vertebrae, pharyngeal abscess, or some disease about the fauces, I endeavoured to obtain a good view of the back of the throat; but, as he struggled violently, the mother would not permit me to persevere, and I merely saw that there was phlegm at the back of the mouth. I also examined the teeth, but the child held its mouth so firmly closed that this was difficult. The pulse was 160, and the temperature in the axilla about 101 deg.

On December 26th, 1873, the child was found, after having been left a short time, lying crying upon the floor, and the dog beside him, according to the supposition at the time, licking a wound upon the child's cheek, inflicted by a fall which the little boy had apparently had. The parents, however, seem to have had some misgivings about the nature of the wound, and in a few days obtained the advice of a surgeon in Halifax, where they then resided. The wound was then covered with a scab, and nothing could be made out regarding it. Upon inquiry, the father of the patient told me that the dog was his own, and that it was not mad. The marks upon the child's face were now clearly those caused by a bite. Two days before I saw him, he began to show signs of restlessness, and, according to the father, appeared as though he had "caught cold". There had been slight cough, but not of a barking character, and throughout it did not assume that form. Upon the morning of the day before I saw the child (the 26th), it drank some tea all right, but did not eat, and it was noticed to start when asleep.

On the morning of the 28th, the dread of movement continued, and that of fluid also. The child had not slept during the night. The mother then, apparently more anxious for the safety of the child than desirous of vindicating the character of the dog, gave me the following account: The dog, a valuable smooth fox-terrier, had always been very savage, and was much disliked by the neighbours. It did not bite at