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.

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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