outwards, as above described. But, as said before, the syringes that I find here are of metal, large and unwieldy, with large calibre, throwing a large stream of water, and worked with difficulty.

[Here the writer showed several ear-syringes, and contrasted them with the small hard India-rubber syringe.]

The cut represents the American India-rubber ear-syringe, and the method of using it with one hand. It is two and a half inches long, the same in circumference, and holds about half an ounce. The orifice of the nozzle is one millimètre in diameter. A larger ear-syringe, one holding an ounce, is made by the American Rubber Companies; but I prefer the small one, as it throws a smaller stream of water.

The easy removal of foreign bodies from the ear depends upon two things that are indispensable.

1. A light small syringe, with a small nozzle, throwing a small jet of water suddenly and forcibly, must be used.

2. The auditory canal must be made straight, by traction outward of the lower lobe of the ear, at the moment of injecting water into it.

With these simple directions, failure is impossible.

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SOME REMARKS ON DR. JOHNSON'S PAPER ON THE MINUTE ANATOMY OF THE SMALL RED GRANULAR KIDNEY.

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It is only now that I find, in the columns of this JOURNAL, a short paper, written by Dr. George Johnson, under the abovenamed title (May 25th, 1878), in which he disputes my theories upon the same subject, published in Virchow's *Archiv* (Band 71, p. 453): "Ueber die Veränderungen der kleinen Gefässe bei Morbus Brightii und die darauf bezüglichen Theorien".

1. Dr. Johnson says that my (Ewald's) observations are incomplete and insufficient, because I have looked for hypertrophy of the arterioles in the pia mater alone, and not in the other parts of the body; and that, in the cases in which I found hypertrophy of the heart without hypertrophy of the arterioles of the pia mater, it is probable that the arterioles in other tissues may have been sufficiently hypertrophied to explain the cardiac hypertrophy; and, on the other hand, it is obvious that hypertrophy of the arterioles of the pia mater without arterial hypertrophy in other tissues would not cause cardiac hypertrophy.

2. He also says that the absence of the cardiac hypertrophy in amyloid and atheromatous thickening of the arterioles is *not* against the

vascular changes as causes of the heart-affection, because here is a mere degeneration of the walls of the arterioles with consequent loss of contractile power, while the arterial contraction and resistance, with resulting arterial hypertrophy, are the main causes of the hypertrophy of the left ventricle.

3. Dr. Johnson further says that the origin of the resistance which causes the arterial tension is not in the capillaries (as I consider), and that my view appears inconsistent with the facts and with the doctrines of vaso-motor physiology. Increased pressure of the Malpighian capillaries would, he says, cause not a copious secretion of urine, but an abundant transudation of albumen. The clinical fact is, that when, as a result of valvular disease of the heart, the return of venous blood from the kidney is impeded, the increased pressure of the Malpighian bodies is associated with a scanty secretion of highly concentrated urine, which, too, is often copiously albuminous.

May it be permitted, in reply to the remarks of this able and competent observer, to state that in general I quite agree with Dr. Johnson as regards the anatomical alterations of the vessels; but on the three foregoing points I have the following remarks to make, upon which I find Dr. Saundby has already partially insisted in his reply to Dr. Johnson in this JOURNAL (June 15th, 1878).

1. Dr. Johnson, as well as Gull and Sutton, always found the state of the vessels of the body identical with that of the pia mater. It was the same in the cases in which I examined, not only the vessels of the pia mater, but also those of the skin and the mesentery, which cases, in fact, were but few. It was permissible, therefore, to adopt the results found in the smaller field of the pia mater for the vessels of the rest of the body. On the other hand, the vascular changes are dependent upon a common cause, and this cause must equally concern all provinces of the body except those between the heart and kidneys. These are Dr. Johnson's own observations, which I followed, and which he now disputes.

2. I did not make use of the absence of cardiac hypertrophy in amyloid, or atheromatous degeneration, directly against Dr. Johnson's theory. They are examples to show that degeneration of the walls of the arteries is not sufficient to cause hypertrophy of the heart, as those would be inclined to think, who believe that degeneration of the walls would impede the flow of the blood, as has been found by Cohnheim in his experiments on inflammation; although, in these experiments, not even a visible degeneration of the walls of the vessels can be observed. But this point is of little interest if the following facts are correct.

3. It is true that the albumen is transuded generally from the Malpighian capillaries, but this only happens in cases where the wall of the Malpighian capillaries has undergone a pathological change. The simple pressure alone is never sufficient to cause transudation of albumen in the glomeruli, but the secretion of albumen goes on through the venous capillaries, when the return of venous blood from the kidney is impeded by valvular disease or otherwise. Nussbaum has shown, in an interesting communication (*Untersuchungen über die Secretion der Nieren*, Pflüger's *Archiv der Physiologie*, Band. xvi und xvii), that the function of the Malpighian capillaries is to balance the quantity of water contained in the blood, independent of the secretory power of the rest of the kidney, and that albumen and sugar pass through the Malpighian capillaries, if they be pathologically changed. The quantity of albumen secreted in the urine depends entirely upon the degree and quantity of the affection of the Malpighian capillaries, and has nothing to do with the pressure in front or behind them (see a very instructive case of Dr. Litten, *Berliner Klinische Wochenschrift*, 1878, No. 22).

I have shown that the view of Dr. Johnson concerning the regulating force of the small vessels is due to a wrong interpretation of a physiological experiment, correct in itself (*i.e.*, the increasing blood-pressure after suspension of the respiration). The action of the heart itself, if it acted in antagonism to the contracted vessels, would tend to counteract the intended result, *i.e.*, to arrest the flow of blood in the affected tissues. Further, I pointed out that, according to the experiments of Heidenhain (Ostroumoff), the vessels contract under a greater pressure, so as to retain their natural lumen quite independently of nerves entering from the outside, which are cut before. Now, as the resistance lies in the capillaries, and causes a higher pressure in the arterioles and arteries, the hypertrophy of the walls of the latter must be a purely mechanical effect caused by the over-tension in the system of blood-vessels, and independent of central influence. Besides, the thickening of the walls of the arterioles found *post mortem* does not prove that there was really a contracted channel during life.

I am not aware of any facts which disprove this theory. I hope that if Dr. Johnson will kindly read my original paper, where all these points are more explicitly related, he will agree with my views.