to put it more accurately, it depends on the difference between the blood-pressure in the glomeruli and the pressure in the renal tubules.

Non excretion of water then, if complete, means complete suppression of all renal excretion; and the effect upon the blood of a deficient excretion of water, apart from the retention of the urinary solids, supposing it not to be corrected either by a deficient ingestion or increased loss through some other channel, must be to increase the whole amount of the circulating fluid, while diminishing the percentage of its solids. This state may be called a "hydræmic plethora," (Cohnheim), or polyæmia serosa vel aquosa.

But it is a fact that even complete suppression in Bright's Disease, obstructive suppression, hysterical anuria, or experimental ligature of the ureters does not materially alter the volume of the blood or the blood-pressure (9); so that in some way or other the inactivity of the kidneys is compensated for. On the other hand, the injection into the circulation of considerable amounts of serum or salt-solution neither raises the blood-pressure nor produces general dropsy or uræmic symptoms, unless at the same time the ureters are ligatured. But Cohnheim and Lichtheim (10) have shown that by injecting a six per cent. solution of common salt until the death of the animal, which generally took place gradually, serous transudation occurred from the glands and into certain lymphatic channels and tissues; there was more or less ascitic fluid, the mucosa and submucosa of the stomach and intestine were œdematous, also the pancreas and kidneys, but there was no fluid in the pericardial or pleural sacs, nor was there œdema of the lungs, of the central nervous system, or of the subcutaneous connective tissues. The only way then in which deficient excretion of water can act as a cause of uræmia is by preventing the elimination of the solid matters of the urine.

There is, of course, an increase of blood-pressure in some forms of Bright's Disease, especially the chronic, but its cause must be sought elsewhere than in hydraemic plethora. The non-elimination of water, however, and the heightened blood-pressure, have given rise to the mechanical theories of uræmia. Owen Rees (11), attributing renal dropsy to hydræmia, supposed that when the great nerve centres became implicated in dropsy, that was quite enough to explain the uræmic symptons. Traube (12) and his followers advanced the same theory in a somewhat modified form. According to him, cardiac hypertrophy, heightened blood-pressure, and watery blood, caused cerebral œdema, followed as a consequence by cerebral anæmia, and this affecting one or other portion of the brain gave rise to the uræmic convulsions or coma. Munck (13) endeavored to support this theory by experimental evidence; and more recently Mahomed (14) has gone a step further in the same direction. He ascribes the drowsiness and coma to cerebral œdema, and the convulsions to the presence of numerous punctiform hemorrhages in the gray matter of the convolutions. It has been pretty conclusively proved, however, that every part of this ingenious theory is untenable. In many instances (15) the brain has been found *post mortem* to contain no more than the normal relative amount of water, and even where an œdematous or hemorrhagic condition has been observed, it is to be considered perhaps as the effect rather than the cause of the convulsions (16).

Of the solid constituents of the urine, *urea* is the most important. It represents the final stage in the metabolic changes through which nitrogenous compounds (proteids) pass in their transit through the body. There have been great differences of opinion among physiologists as to its antecedents and place of formation, and the only statements in regard to it that one can make with certainty so far are, "that it is the chief end product of the metabolism of the

(9). FAGGE. Loc. cit., p. 460.

(10). COHNHEIM J., LICHTHEIM, L. Ueber Hydrämie und Hydrämische Oedem. Virchow's Archiv, 1877, XCVI.

(11). REES, OWEN. On the Nature and Treatment of Diseases of the Kidney Connected with Albuminous Urine; London, 1850.

(12). TRAUBE. Gesammelte Beiträge zur Pathologie und Physiologie, 1871, Bd. 2, s. 551.

(13). MUNCK, PH. Ueber Urämie, Berliner Klin, Wochenschrift, 1864, s. 111.

(14). MAHOMED, F. A. On the Pathology of Uræmia and the so-called Uræmic Convulsions. British Medical Journal, 1877, II., 10, 42.

(15). CARTER. Loc. cit.

(16). BARTELS. General Symptoms of Renal Disorders. Ziemssen's Cyclopædia of the Practice of Medicine, Vol. XV., p. 138, 1877.