the anatomical facts, probably affords the true explanation of the phenomena.

In consequence of degeneration of the kidney, the blood is morbidly changed. It contains urinary excreta, and it is deficient in some of its own normal constituents. It is, therefore, more or less unsuited to nourish the tissues—more or less noxious to them. The minute arteries throughout the body resist the passage of this abnormal blood. The left ventricle, therefore, makes an increased effort to drive on the blood. The result of this antagonism of forces is, that the muscular walls of the arteries, and those of the left ventricle of the heart, become simultaneously and in an equal degree hypertrophied. The persistent overaction of the muscular tissue, both cardiac and arterial, is found registered after death in a conspicuous and unmistakable hypertrophy.

The hypertrophy of the minute arteries of the kidney is best explained by a reference to the analogous phenomena of apnoea. A ligature on the trachea of a dog destroys life in a few minutes. The immediate cause of death is the arrest of blood in the lungs. The left cavities of the heart are found after death nearly empty; the right cavities, the pulmonary artery with its branches, and the systemic veins, are much distended; the pulmonary capillaries are nearly bloodless; and the lungs, consequently, collapse to an extreme degree when the chest is opened.

The theory is, that, when the respiratory changes are suspended by the exclusion of air, the minute pulmonary arteries, under the influence of the vaso-motor nerves, by their contraction arrest the flow of blood. Similar phenomena occur during a fit of spasmodic asthma. Bronchial spasm lessens the supply of air; then the contraction of the minute pulmonary arteries, in a corresponding degree, checks the circulation. The skin becomes cold and blue, the pulse small and feeble; and the patient is apparently at death's door. When the bronchial spasm relaxes, the air gets ready access to the pulmonary cells, then the arterial resistance ceases, and respiration and circulation again become free. In like manner, when the secreting tissue of the kidney is partially disorganised, the working power of the gland is lessened, and it requires less blood. The minute renal arteries regulate the blood-supply in accordance with the diminished requirements of the gland. This continues and increases month after month, year after year; and the result of the persistent overaction of the minute renal arteries is, that their muscular walls become hypertrophied. This hypertrophy of the muscular walls of the minute renal arteries occurs in all forms of chronic Bright's disease; in the small, red, granular kidney; and in the large, white, smooth kidney. The hypertrophy of the muscular walls is quite distinct from the degeneration of the arterial tunics which occurs during the progress of that form of disease of the kidney to which the term "lardaceous" is commonly applied.

In conclusion, I may mention some of the phenomena of chronic Bright's disease which are explained by the facts to which I have referred.

1. The hypertrophy of the left ventricle is explained. That which Dr. Bright long ago surmised is now a demonstrated fact. There is an impediment to the passage of the altered blood "through the distant subdivisions of the vascular system"; hence arise greater action of the heart, and hypertrophy of its walls.

2. The full, hard, radial pulse, and the increased pressure on the arterial walls indicated by the sphygmograph, are explained by the co-existence of excessive cardiac force with an equal, or more than equal, excess of arterial resistance.

3. The excessive dryness of the skin, and the difficulty of exciting diaphoresis even by the hot-air bath, are accounted for by overaction of the hypertrophied subcutaneous arteries resisting the relaxing effect of external warmth, which has so powerful an influence upon the healthy skin.

4. One result of excessive resistance throughout the whole systemic arterial system must be to impede the passage of blood from the venous system through the lungs, and thus to favour the occurrence of dropsy by an influence not unlike that of a defective valve on the left side of the heart. The influence of arterial resistance in retarding the circulation will mainly depend upon the extent to which this is counteracted by hypertrophy of the left ventricle of the heart.

5. Lastly, the not infrequent occurrence of cerebral hæmorrhage is explained. While the minute cerebral arteries resist the passage of the blood, the strong left ventricle is forcibly driving it onwards. In the struggle between the two contending forces an artery gives way, and the result is cerebral hæmorrhage and apoplexy.

POSTSCRIPT.—During the discussion which followed the reading of this paper, I was asked for an explanation of the thick inner layer of longitudinal fibres, which forms so conspicuous a feature of the hypertrophied renal arteries. (See Fig. 2.) I replied, that I had in vain sought for a satisfactory explanation. It has since occurred to me that

the following may be the physiological explanation of the anatomical fact. The renal arteries are of large size, and have a very short course from the aorta into the gland. During the progress of chronic Bright's disease, the circulation through the kidney is much obstructed; so that, with a large afflux of blood to the diseased organ, there is a much impeded outflow. The degree of impediment to the circulation through the kidney may be estimated by the hypertrophy of the arterial walls. This usually appears to be greater in the renal arteries than in those of any other tissue. I have often seen the thickness of the arterial wall equal to the diameter of the canal of the artery. The tendency of the distending force of the hypertrophied left ventricle obviously is to elongate as well as to dilate the arteries. We have a good illustration of this in cases of incompetent aortic valves with a dilated and hypertrophied ventricle, when the subcutaneous arteries can be seen and felt to swell and lengthen, and so to move beneath the skin, at each systole of the ventricle. That the minute renal arteries, in cases of chronic Bright's disease, become elongated, is shown by their tortuous course, which is visible in some microscopic specimens. (See Fig. 2.) This elongation of the vessels would be checked by increasing the thickness and strength of the longitudinal fibres; and here probably we have the physiological interpretation of the hypertrophied longitudinal layer. This is more conspicuous in the renal arteries, because those vessels are subjected to greater strain and pressure than the arteries of other tissues, and because in them the general hypertrophy of the walls is excessive.

In confirmation of this view, I may refer to some specimens in my possession from a case of chronic Bright's disease, in which, with only moderate hypertrophy of the circular fibres of the minute renal arteries, the inner longitudinal layer is scarcely visible. These arteries have their canals here and there irregularly dilated, and some of them have a course almost as tortuous as that of the convoluted uriniferous tubes. I infer that in this instance the arteries had become unusually elongated, tortuous, and dilated, in consequence of the absence, or rather the slight degree, of that protective hypertrophy which is so generally present in cases of chronic Bright's disease.

FRACTURE OF BASE OF SKULL: LACERATION OF BRAIN.

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THE following case is recorded as a good instance of the length of time which may elapse from the receipt of the injury to the occurrence of the more serious and fatal symptoms. S. K., aged 65, a farmer of intemperate habits, on his return from market, was thrown out of his cart between 5 and 6 p.m., when about eight miles from home. He was momentarily puzzled by the fall, but quickly helped to replace his market purchases in the cart. A friend drove his horse for four miles, during which time he vomited, but said he was very well, only cold. He then drove on by himself, and on his way left some goods at a friend's house about a mile from home, when he complained of his head. Near his house, he had to alight to open a gate; and a person living close by remarked that he had come home sober to-night, as he managed to open the gate more quickly than usual. Indoors, he complained of his head and of the cold, and sat down, but soon got up again, and lay down before the fire, and appeared to sleep; all this was his usual custom. At bedtime, they tried to rouse him, but found it impossible; and he died shortly afterwards, just at midnight. I saw the body an hour afterwards, and found only a slight contusion, with superficial punctiform lacerations over a space two inches in diameter, a little posterior to the vertex, as if caused by gravel. There was no discharge from the ears. There were slight bruises on the left hand and right leg. No one in the house was aware of his having met with an accident; but I found the cart scratched and the harness broken.

Post Mortem Examination.—A small effusion of blood was found under the bruise of the scalp. Both anterior lobes of the cerebrum were covered with a layer of semi-coagulated blood from a quarter to half an inch in thickness; on the right side, this clot extends back to the posterior lobe, and likewise over a great part of the base of the brain. This had escaped from a rent in the anterior edge of the right hemisphere, which communicated with a cavity about the size of a large walnut, situate a little anterior and external to the anterior cornu of the lateral ventricle. The ventricle itself was uninjured. Blood was effused also under the arachnoid as far back as the junction of the medulla oblongata with the pons Varolii. There was a fracture of the occipital bone, commencing just to the right of the tuberosity, passing down and to the left side of the foramen magnum, into which it entered; a branch fissure passed forwards from near the foramen, and ended in the petrous portion of the temporal bone. Altogether it was about four inches in length, and freely admitted the point of a scalpel.