Anasarca is not uncommon without fluid in any of the serous cavities; but true dropsy of any of the serous cavities rarely occurs without more or less œdema of the subcutaneous connective tissue. The cavities in which dropsical fluid mainly collects are four; viz., the two pleuræ, the pericardium, and the peritoneum.

Dropsy of the pleuræ is called *Hydrothorax*. It is characterised by the physical signs of fluid in the pleural sacs, which I shall have to describe to you in a future lecture, occurring in conjunction with the signs of dropsy elsewhere, but without pain, pleuritic friction, or pyrexia. Moreover, it is a peculiarity of hydrothorax, that the signs of liquid effusion are not confined to one pleura, but that the upper level of the fluid rises to about the same height on both sides. In this respect it differs from the effusion resulting from inflammation. In exceptional cases, however, of hydrothorax, where adhesions over one lung prevent the accumulation of fluid in the pleura, or where the patient lies constantly on one side, the dropsy may be confined to, or preponderate in, one pleura. Fluid in the pleuræ compresses the lungs and impedes the entrance into them of air: as a rule, the respiratory functions are further embarrassed by the circumstance that hydrothorax is usually associated with more or less œdema of the pulmonary tissue. The combination of these two morbid conditions is a common cause of death by asphyxia in cases of dropsy. Hydrothorax, however, differs from inflammatory effusion in this respect, that as soon as the fluid is removed from the pleura there is no obstacle to the complete re-expansion of the lung, in the shape of bands of organised exudation.

Hydropericardium is the term applied to dropsy of the pericardium. Its presence is denoted by the physical signs of fluid in the pericardial sac, which we shall have to study when we come to discuss diseases of the heart, in conjunction with signs of dropsy elsewhere, but without pain, pericardial friction, or pyrexia. This, however, you must remember, that inflammation of the pericardium leading to effusion is an occasional complication of both renal and cardiac dropsy. Hydropericardium embarrasses the action of the heart and contributes to a fatal result by asthenia. In fact, the rapid effusion of dropsical fluid into the pericardium is far from being an uncommon cause of death in cases of dropsy.

Dropsy of the peritoneum is sometimes designated *Hydroperitoneum*, but more commonly *Ascites* (Ἀσκιτης, *from* ἀσκος, a leather bottle). I shall have much to say hereafter on the clinical characters of ascites; but here I may observe that it differs from hydrothorax and hydropericardium in this respect, that, although often associated with evidence of dropsy in other parts, it very often exists independently. A considerable quantity of fluid may accumulate in the peritoneum without causing the patient much inconvenience, except from its weight. But when the accumulation is so great that the abdominal walls are pressed out in every direction, ascites is the source of other evils than those arising from its mere bulk or weight. The pressure of the peritoneal fluid on the iliac veins causes venous repletion of the lower extremities, followed by œdema; its pressure upon the renal veins leads to a diminished flow of urine and albuminuria; and, what is the greatest danger of all, the pressure upwards against the diaphragm resists the due contractions of that muscle, and displaces the lungs and heart, so as seriously to embarrass the functions of these organs.

Dropsy of the cerebral ventricles or of the arachnoid space constitutes the disease known as *Chronic Hydrocephalus*. This, with rare exceptions, commences in intra-uterine life, or soon after birth. It is a purely local dropsy, unassociated with dropsy in any other part of the body. The quantity of fluid is sometimes enormous; it has been known to amount to ten or even twenty pints; and its specific gravity (about 1007) is unusually low, even for a dropsical fluid. The cause of the dropsy has been traced in some instances to pressure on the veins of Galen which return the blood from the ventricles, or on one of the cerebral sinuses, by a cancerous, cystic, or other tumour, or to obliteration of these vessels; the dropsy, in fact, being the result of venous repletion, in the same way as ascites results from any obstruction of the portal circulation. But in a large proportion of cases no such cause can be discovered; and it may be doubted if the disease have not been inflammatory in its origin, as Rokitansky thinks, rather than a true dropsy. West observes that each year leads him to estimate more highly the share exercised by inflammation of the lining membrane of the ventricles in the production of chronic hydrocephalus. In six out of forty-five cases collected by him, the enlargement of the head succeeded to an attack resembling acute hydrocephalus; and in one it seemed to be induced by an injury of the head (*Diseases of Children*, 5th ed., 1865, p. 121).

There is another form of accumulation of serous fluid within the cranium which calls for notice. In examining the bodies of patients who have died of general dropsy, it is not uncommon to find a few drachms, or even ounces, of serum in the lateral ventricles and in the arachnoid space. This is commonly regarded as merely part of the general dropsical effusion; but there are good grounds for thinking otherwise. Similar intracranial accumulations of serum are common when there is no dropsy elsewhere; as, for example, after death from protracted fevers, or from the contracted granular kidney, or in old age. In all of these instances, the brain first becomes atrophied, and, the firm unyielding skull-cap being unable to accommodate itself to the reduced size of the viscus within, a local venous repletion, followed by effusion of serum to fill up the vacant space, is the result. When death occurs in the early stage of acute general dropsy, even when preceded by cerebral symptoms, little or no fluid may be found in the cerebral ventricles. In most of the text-books you will find it stated that the fluid found in the cerebral ventricles in cases of dropsy may exercise a dangerous degree of pressure upon the brain-substance, so as to cause drowsiness, coma, and even death; and in practice these symptoms are constantly referred to serous effusion upon the brain, while efforts are made to relieve the symptoms by remedial measures supposed to have the power of removing the effusion. This opinion, although widely prevalent and endorsed by high authority, has long appeared to me to be a pathological error. It is quite true that patients, whether suffering from general dropsy or not, often die comatose; and that on *post mortem* examination no cerebral lesion can be found, except effusion of serum on the surface of the brain and into the ventricles. But a similar amount of cerebral effusion is not uncommon where death has not been preceded by coma; and in most instances the coma may be traced to a morbid state of the blood, resulting from renal diseases or other causes, and interfering with the proper nutrition of the brain. A large proportion, for example, of the cases of fatal coma which were at one time set down to what was called "serous apoplexy" are really instances of uræmic coma, consequent on destruction of the secreting tissue of the kidneys. In some cases, cerebral symptoms are no doubt to be accounted for by the atrophy of the brain consequent on its imperfect nutrition. When, however, the cranium has once become firmly ossified, if we exclude cases of an inflammatory nature, it is very doubtful if more serum is ever thrown out than suffices to fill the space vacated by the atrophied brain, or that such serum exercises an injurious pressure upon the remaining brain-substance.

[*To be continued.*]

CLINICAL MEMORANDA.

PARACENTESIS THORACIS.

JOHN HURST, aged 32, a boiler-smith, residing in Newcastle-upon Tyne, caught cold on 19th April, 1871, at Barrow-in-Furness. He returned home on the 24th. His journey homewards by rail brought on an attack of double pneumonia; all the symptoms of an aggravated form had set in on my arrival. I could not, however, then detect any inflammation of the pleura. The usual antiphlogistic remedies were applied by me until the virulence of the symptoms had subsided, and he had so far recovered as to be able to go to the Prudhoe Memorial Convalescent Home at Whitley. After a week's residence at the seaside, he became very ill with purging and profuse night-sweats. He returned to Newcastle, where he gradually became very weak and emaciated. He coughed up about a pint of thin purulent matter every day, until he had decreased in weight from 11 stone 6 lbs. to 9 stone.

Upon my examination of his chest, I found the whole of the right pleural cavity blocked up with fluid; a loud splashing was heard on succussion. The respiration in the left lung was 35; pulse 130; his feet were very œdematous. To ascertain the nature of the fluid, I passed an exploring needle. Dr. G. H. Philipson kindly visited the patient with me, and confirmed my opinion as to the necessity for paracentesis. In his presence, I therefore introduced a large-sized trocar into the fifth intercostal space, and six pints of pus flowed out. We gave him two grains of opium immediately, and prescribed a mixture of bark and dilute hydrochloric acid. A drainage-tube was retained in the cavity of the chest for nine days, during which time we allowed him to go out for a short walk every day. The discharge gradually diminished in quality and quantity, until it finally disappeared on February 24th. The lung quickly regained its healthy action; so much so, that we could scarcely distinguish any difference between the diseased and the healthy side of the chest. He improved every day, and has regained his original weight. He returned to his employment on March 4th. The wound closed in a few days after the tube had been withdrawn.

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