of the arterial bed is the chief factor to be considered. When the arteries are widely dilated the blood escapes readily into the capillaries and veins. If, however, vasoconstriction occurs, the driving power remaining the same, less blood will escape. The contraction of the arterial area causes an acceleration of the blood-current; but this acceleration, owing to the increased friction which it entails, is not sufficient to compensate for the diminution in volume of the blood flowing through the arteries. In short, the statement "rate of flow is in inverse proportion to area of bed" does not apply when the driving force remains the same unless the influence of friction is eliminated, which is only possible in theory; whence it follows that, in order to get the same arterial outflow as before, the *vis a tergo* must be increased. It will thus be seen that the amount of arterial resistance can be regulated with great nicety, and that the arterial outflow can be kept the same amid considerable modifications in this resistance by regulating the strength of the heart-beat. What is aimed at in the normal organism is the production of just so much resistance as shall suffice to cause the requisite degree of distension for the maintenance of a continuous capillary flow.

That the veins, in striking contrast with the arteries, are constructed with a view to minimising resistance seems indisputable, though, as far as I know, the anatomist has not studied them in this light. Two facts stand out prominently in this connexion: their great capacity and distensibility on the one hand, and on the other the numerous anastomoses of their trunks. The relatively great capacity of the veins is shown by comparing the venules and arterioles. The latter as they enter the capillary network are always smaller than the afferent venules, so that under normal conditions the capillaries can always readily discharge themselves into the veins. This same disproportion between the capacities of the arterial and venous systems is observed throughout: the veins can hold considerably more than all the blood in the body, and this not only on account of their great capacity but by reason of their distensibility; it therefore follows that the venous system as a whole can never be tightly stretched—viz., over-distended. In other words, the tension in the venous blood cannot be high—for high tension implies such stretching—and without high tension there cannot be great obstruction, since the two rise and fall together. The constant anastomosis of the veins has for its chief object the protection of the capillaries from unduly high pressure, for many veins are liable to obliteration from outward pressure, and their frequent anastomoses prevent the outlet from the capillaries being blocked. The capillaries are further protected from the injurious effects of such pressure on the veins by the valves which these latter contain.

To sum up, the very low pressure in the veins and the high pressure in the arteries are contingent upon the physiological requirements of the capillary blood-flow, but for which a heart weighing a few ounces only would probably suffice to carry on the circulation. Blood tension would then be low throughout the vascular system, and that deadly array of evils directly traceable to high tension would be practically unknown.

Devonshire-street, W.

DEATH FROM HÆMORRHAGE INTO THE PERICARDIUM.

By CHRISTOPHER ADDISON, M.D. LOND.

IN connexion with the cases illustrative of this condition recently reported in THE LANCET by Drs. Fraser¹ and Gordon,² I am enabled, by the kindness of Dr. Claye Shaw, to furnish details of another interesting example which occurred in the London County Asylum, Banstead. A widow aged sixty was admitted on May 25th, 1892, suffering from melancholia. She was pale and thin (weight 7 st. 11½ lb.) and had a vacant expression. She was very deaf. Although reported "dangerous to others," she never displayed herself in this way whilst in the asylum. She had hallucinations of hearing and many delusions of suspicion and unworthiness, on which she spoke freely when questioned; at other times she would talk quietly to herself. It was noted on her admission that there was a systolic murmur audible at the apex. As time went on she

became quieter and more apathetic, seeming to brood continually over her sorrows. No cause for the insanity could be ascertained. On Dec. 14th, 1892, she had an apoplectic seizure, in which consciousness was lost for a short time (duration not stated); there were paralysis of the left arm and impaired movements of the left leg. In the course of a few days motion returned completely in the leg and to a great extent in the arm. On June 9th, 1893, a similar attack occurred, again affecting the left side; it was of a milder character than the first, and paresis was transient. The patient was able to get up and walk about the ward. In July it was noted that she had become much more demented than formerly. On Oct. 30th a third seizure took place, affecting this time the right side. The patient was unconscious during one day and in a semi-conscious state during another. There was paralysis of the arm and lower part of the face on the right side. It was difficult to ascertain whether the speech was affected. The urine and fæces were passed into the bed. On Nov. 6th she was reported as having improved; her mental condition was clearer, and she was less wet and dirty. The paralysis continued. On Nov. 23rd, at 8 A.M., she was fed with a breakfast of sop. She had finished the meal and apparently swallowed well, when she sank back and, without struggling, ceased to breathe. Dr. Nixon was called and was in attendance at once. He found her to be quite dead. There was no sop in the mouth.

Post-mortem examination.—This took place on Nov. 24th. The state of the body, the skull cap, and the dura mater presented nothing noteworthy. Much fluid remained in the posterior fossæ after removal of the brain. The sub-arachnoid space of the left hemisphere contained many lakelets of fluid. The arachnoid and pia mater stripped off the convolutions with slight laceration and were not thickened, save around the softened patches. The arteries at the base were not atheromatous. Over the middle of the left ascending parietal convolution there was a yellow patch of softening to the superficial extent of half an inch. On the same side, behind the external parieto-occipital fissure on the superior occipital convolution, there was a depression with a floor of yellow softened material extending in the middle line, superficially, for one inch and a half; passing into the longitudinal fissure, it increased to two inches and a half antero-posteriorly and involved the cuneate lobule, and, in front of the internal parieto-occipital fissure, the posterior part of the marginal convolution. On the right side, of somewhat less extent than on the left, there was a similarly situated patch of softening. At the anterior part of the longitudinal fissure there was some adhesion between the hemispheres enclosing a certain amount of fluid; at the posterior part of the upper surface of the cerebellum, on either side of the middle line, there was a cup-shaped depression (able to lodge the end of the index-finger), the floor of which presented similar yellow softening. On section the substance of the brain was found to be generally soft. The superficial grey matter presented nothing to note. There were some dilated perivascular spaces. In both occipital regions, corresponding to the depression noted, the substance of the brain was yellow and soft to the depth of an inch, the right side being less affected than the left. The softened area presented a sharp line of distinction from the rest of the brain substance. The change in the left ascending parietal convolution extended to a depth of a quarter of an inch. The lateral ventricles were a little dilated. The fluid was normal in appearance. The endyma in the region of the choroid plexuses was somewhat thickened. There was marked depression of the anterior ends of both caudate nuclei, and section at this spot revealed no softening. The change noted in the cerebellum extended to a depth of a quarter of an inch. Examination of the internal capsule, crura cerebri, pons, medulla, and other parts of the brain revealed nothing abnormal to the naked eye. On opening the chest the pericardium appeared to be distended; after making an incision into it a large blood clot was seen in its interior. The whole contents of the sac were turned and consisted of six ounces of clot and five ounces of bloody fluid; the heart was removed, and its weight was found to be twelve ounces and a half. On inspection the left ventricle presented about the middle of its left border a small radiate opening. On section this was found to lead into an irregularly shaped cavity situated in the wall of the heart; roughly, it was circular, with a diameter of about two inches. The muscle substance was separated into two layers, the outer of which, in the neighbourhood of the rupture, was extremely thin—not more than one-sixteenth of an inch in thickness; further away it became thicker. The interior of the cavity

¹ THE LANCET, Jan. 6th, 1894. ² THE LANCET, Dec. 2nd, 1893.

was rough and torn at the circumferential parts, but smoot over the thinner central region. The inner wall, generally thicker than the outer, was bulged towards the cavity and also presented at its central part a rupture, torn rather more widely than the external one; the wall around the rent was one-eighth of an inch in thickness. The valves of the heart did not present any vegetations and did not appear to be incompetent. The left coronary artery when dissected out was found to be very atheromatous and showed many thickened patches and local bulgings; one large branch was distinctly seen to terminate in the right upper part of the cavity; but it was not possible to inject fluid through the vessel to this place, apparently because of the extensive thickening on account of the blood clot contained in its canal. The wall of the left ventricle, apart from the site of the lesion, was thicker than usual. The right heart was not dilated. Microscopical osmic acid preparations of a portion of the wall of the left ventricle showed fatty degeneration in parts of the sections. The lining of the aorta presented a few small, white, atheromatous patches. There were some old scars containing portions of calcareous matter at the apex of the right lung. There was no emphysema or congestion. The larynx and trachea were normal. The left kidney weighed three and a half ounces and was granular and shrunken. The right kidney weighed four and a half ounces; its capsule tore off portions of its substance on being stripped. The liver and other organs presented nothing noteworthy.

Remarks.—As to the immediate cause of death, the appearances were those of an aneurysm of a branch of the left coronary artery, which had dissected up the substance of the heart into two layers, the outer one, as would have been expected, being the thinner of the two. In contrast to the condition of the aorta and cerebral arteries, the amount of atheroma of the coronary vessel was very marked indeed, the whole of the vessel from its origin to the aneurysm being affected. Both walls of the sac were found to be ruptured. Perhaps the explanation is that, the outer having ruptured first towards the pericardial sac, the attenuated inner one was not able to withstand the force of the next contraction of the ventricle and gave way too. The fact that the rupture in it was larger than that in the outer wall accords with this supposition. It does not seem to be so likely that the inner one should rupture first and the outer follow; and, moreover, in such a case one should have expected the rent in the outer wall to be the larger of the two. The changes in the kidneys, the existence of atheroma, and the hypertrophy of the left ventricle, apart from the aneurysm, speak for themselves. Certain measurements of the skull and notes of other matters, made chiefly in connexion with the brain, are not given, nor are comments made on the apoplectiform attacks. The case is brought forward at this time in connexion with the mode of death.

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A CASE, DIAGNOSED AS TUBERCULAR MENINGITIS, TREATED BY TREPHINING AND DRAINAGE OF THE SUBARACHNOID SPACE; RECOVERY.¹

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A GIRL five years of age was admitted into the Victoria Hospital for Sick Children, Chelsea, on Oct. 23rd, 1893. Up till five weeks previously she had been perfectly well, and had never had any fits or otorrhoea; but about the middle of September she began to suffer from acute pain in the head. She had occasional vomiting, she was very fretful, her appetite was bad, and her bowels were constipated. There was no history of tubercle in her family. When first seen the child presented a dull, heavy appearance; she was very

restless and uttered from time to time a shrill scream. When asked if she felt pain she put her hand to her forehead; she lay with her head bent forward and her legs drawn up. The temperature was 101.6° F., the pulse was 104, regular and weak in volume, and the respiration was 20, regular. There was no lesion discoverable in the thoracic or abdominal viscera. Tache cérébrale was well marked; the knee-jerks were normal. There was commencing optic neuritis in both eyes, being less marked in the right. During the first night she was very restless, constantly screaming. There was no vomiting, but she took food badly. The optic neuritis became more marked. On the night of Oct. 25th she was evidently suffering from great increase of pain. She screamed almost without intermission from the evening of that day until 6 A.M. on Oct. 26th. There was no retraction of the head nor vomiting. The pulse at 2 P.M. was 108, regular. She referred the pain to the frontal region. When the patient was seen again by me at 3 P.M. she was evidently worse. I then asked my colleague, Mr. Waterhouse, to examine her with me. We found the pulse to be wavering in a striking manner. At one time it was 120 per minute and regular, on a second occasion it was 70 per minute, with several intermissions, and two or three minutes later it was 80, with no intermission. The respiration was regular, but inspiration was long-drawn. The child was lethargic and apathetic, except for the typical hydrocephalic cry she uttered at times, and she lay on the left side with the lower limbs slightly flexed on the abdomen. She had the appearance of one rapidly passing into a state of coma. The abdomen was retracted, the face was pale, and the tache cérébrale was well marked. The temperature was 101°. On examining the eyes we found that the pupils had been dilated with homatropine, and therefore no account can be given of their size and mobility, but the notes stated that they had been contracted on admission and had become more dilated before the homatropine was instilled. There was no ptosis or strabismus as far as could be ascertained. Upon ophthalmoscopic examination optic neuritis was found, being marked on the left and less marked on the right side. The ears were examined and the membrana tympani was seen to be healthy on both sides. We agreed that the child was suffering from tubercular meningitis, that there were distinct signs, as evidenced by the great pain and optic neuritis, of increased intra-cranial pressure, that the child was rapidly approaching the stage of coma, and that if such pressure should be unrelieved, there was every probability of death taking place in a few hours from compression. On these considerations we agreed that an operation for the relief of the pressure was both justifiable and advisable. It was decided to open the subarachnoid space, and to drain this space for a few days. There was some doubt in our minds whether to open the subarachnoid space of the brain or of the spinal cord, but eventually it was decided that the opening should be made into the cranial cavity, and we feel convinced that this was the right decision. Accordingly at 5 P.M. the child was placed under chloroform, and, after the scalp had been shaved and rendered aseptic, a curved incision about two and a half inches in length was made over the left cerebellar fossa of the occipital bone, commencing below and behind the mastoid process, passing with its convexity upwards, and ending externally to the external occipital crest. The occipital artery bled furiously and was tied, and the pericranium was raised in a flap corresponding to the scalp flap. There was some bleeding from the torn mastoid vein. A trephine, three-quarters of an inch in diameter, was then applied midway between the external occipital crest and the mastoid process. The disc of bone removed was placed in warm boric solution. The dura mater bulged tensely into the trephine opening, and no pulsation was observable. The dura mater and arachnoid were then incised, and some thirty drops of a slightly greenish serous fluid escaped. The cerebellum then bulged into the foramen in the skull, fitting it tightly, like a cork in a bottle. A silver probe, the terminal half-inch of which was bent to a right angle with the rest of the instrument, was then inserted between the cerebellum and the arachnoid inwards towards the falx cerebelli. As soon as the latter was felt the probe was rotated, so that the end projected forward into the large subarachnoid space between the cerebellum and the medulla. Some drachms of the serous fluid at once escaped. A drainage-tube was then passed along the probe and left in position; it was found that fluid passed slowly along it. The dura mater was then sutured, and the disc of bone having been cut up into small fragments with bone forceps, these were packed carefully in a mosaic, after the manner of Professor

¹ A paper read by Dr. W. W. Ord before the Medical Society of London, March 5th, 1894.