

10 centimetres beyond origin, 1·2 centimetres; circumference of the right carotid at point of origin, 1·7 centimetres; circumference of the right carotid at point six inches beyond origin, 1·6 centimetres; circumference of the left carotid at origin, 1·8 centimetres; circumference of the left carotid at 10 centimetres beyond origin, 1·8 centimetres; circumference of the descending thoracic aorta, 3·5 centimetres; circumference of the abdominal aorta, three centimetres; and circumference of the abdominal aorta immediately above the division into the common iliacs, 2·8 centimetres.

It will be seen that the difference in the size of the lumen of the vessels supplying the upper limbs on the two sides presented quite an insignificant difference. The interior of the vessels was quite healthy, presenting no signs of atheroma. The bronchial glands were large and deeply pigmented. Both pleural cavities were practically obliterated except a small space over the apex of the left lung. What adhesions existed over the upper part of the left lung were easily broken down, but over the lower part of the same lung, especially at its lower posterior part, there were dense adhesions of almost cartilaginous consistency, firmly binding the lungs to the ribs. The cavity of the right pleura was obliterated except over the apex of the lung by firm fibrous adhesions. In consequence of these dense adhesions it took a long time to remove the thoracic viscera from the chest. The thickened pleura over the lower lobes of both lungs posteriorly was half an inch in thickness and at the lowermost part fully an inch. There was no sign of tubercles in connexion with the pleuræ or lungs. The lungs were compressed by the thickened pleura, but appeared to be more or less crepitant throughout. The heart, as before said, was adherent, not only to the external layer of the pericardium, but the external layer of the pericardium was adherent to the chest wall and to the lungs and mediastinal tissue. The heart was somewhat enlarged, but as the whole preparation was required for museum purposes it was considered advisable not to dissect and remove the organ from its connexions. An antero-posterior incision through its substance, passing through the right into the left ventricle, was, however, made. The muscular walls of the ventricles except for being slightly increased in thickness appeared to be healthy.

With regard to the abdomen there were general peritonitic adhesions uniting the coils of the intestines together and these to the liver and spleen. The peritoneum over the liver was greatly thickened and on the right lobe it presented a white, porcelain-like appearance. Over the anterior abdominal wall the parietal peritoneum was also thickened and of a white, porcelain-like appearance. There was no evidence of tuberculosis in the peritoneum or in the mesentery except a calcareous mass more or less spherical about half an inch in diameter situated in front of the right kidney. The mucous membrane of the intestines was generally pale and anæmic with patches of congestion in some places, but there was no ulceration. The liver was closely surrounded by adhesions with a markedly thickened capsule, as before described. The whole organ was much compressed by the adhesions and the thickened capsule; its transverse diameter measured 21 centimetres; it was not apparently increased beyond its normal dimensions. On transverse section the organ seemed to be in a condition of passive venous congestion; the whole organ was of dense consistency and there was possibly a certain degree of cirrhosis present. Subsequent microscopical examination showed that although there were strands of fibrous tissue extending into the liver substance from the thickened capsule there was little or no increase of the hepatic interstitial tissue beyond a short distance from the surface of the organ. The liver cells showed various signs of degeneration. As it was desired to keep the whole preparation for a museum specimen the various organs could not be detached for their weight to be taken. Both kidneys were enlarged, the vertical measurement of each being 10 centimetres; both were markedly congested. The spleen was enlarged; it measured 13·5 centimetres by nine centimetres, was firmly adherent to surrounding parts, and was of a dark-red colour and firm consistency. The stomach and pancreas except for surrounding adhesions presented nothing unusual. The uterus and ovaries were of infantile type, surrounded and matted together by fibrous adhesions. The brain was not allowed to be examined.

The post-mortem examination confirmed the diagnosis of indurative mediastino-pericarditis with its common complication of chronic peritonitis, but we cannot say that it has

completely solved the problem as to why the pulsus paradoxus was present in so marked a form in the arteries of the left arm only. Certainly we could not satisfy ourselves that there was any particular adhesion which during an inspiratory act would drag upon and kink the left subclavian artery. As far as we could see, the adhesions were equally numerous and of a similar nature and arrangement around all the vessels springing from the arch of the aorta. All the vessels springing from that part of the main arterial trunk were embedded in a mass of dense fibrous tissue. Not only around the origin of the main arteries to the neck and to the upper extremities were the adhesions apparently similar on the right and on the left of the median line, but all down the mediastinum the adhesions were similar on the two sides of the body. The only difference in the arrangement of the newly-formed fibrous tissue in the thorax on the one side as compared with that on the other was that the right pleura was thicker over the lower part of the right lung than was the corresponding part of the left pleura, and also that the pleuritic adhesions were more general and firmer over the upper part of the right lung than over the upper part of the left organ.

Although we could not actually see any difference in the arrangement of the adhesions around the main vessels proceeding to the upper limbs on the two sides of the median line it appears improbable that the arrangement of the adhesions was such that they took no part in the production of the pulsus paradoxus of the left side. In other words, it is probable that, although we could not demonstrate to our satisfaction that the adhesions round those main vessels were different on the two sides, the adhesions were the important elements in the production of this peculiar paradoxical pulse. It was suggested to me that most probably the explanation of the unilateral pulsus paradoxus in this case lay in the greater length and the narrow lumen of the left subclavian artery as compared with the length and lumen of the innominate artery—that adhesions in the mediastinum would be likely to have a greater effect on the narrow subclavian and more likely to cause a kinking of that vessel than would adhesions connected with the innominate artery. This explanation appears a not improbable one, but whether it is the true one we cannot say. It appears, however, to be the most feasible explanation of the occurrence in this case of a pulsus paradoxus which was much more marked in the arteries of the left than in those of the right arm.

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CERTAIN POINTS IN THE PATHOLOGY OF THE PERICARDIUM.¹

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WHEN a large quantity of venous blood is driven into the right heart the pericardium supports the heart so as to prevent over-distension. It prevents dilatation beyond a certain point at which the walls of the heart are thick enough and strong enough to empty themselves at each recurring systole. The pericardium bears to the heart muscle the same mechanical relation as the leather case does to the inside case of a football or the outer case to the inner case of a Dunlop tyre. As I believe this observation on the function of the pericardium to be original you will perhaps allow me to demonstrate it. (The full account of the experiments will be found in the Proceedings of the Physiological Society, March 12th, 1898.)

The pericardium is a very strong and inextensible membrane. Strips of it rolled into cords will bear a very great strain without stretching or snapping. If an entire pericardium is inflated by a bicycle pump it ruptures at a pressure of from one and a quarter atmospheres to two atmospheres which is many times greater than arterial blood-pressure. It stretches very little before it ruptures. The pericardium limits the capacity of the heart by about one half. The excised heart of a cat in its pericardium was squeezed empty and then distended with saline solution at about a venous pressure. The amount required to fill it was

¹ A paper read before the Medical Society of London on March 27th, 1899.

noted and the pericardium was then freely opened. The heart then took as much saline again before it was distended. The extra quantity of saline was mostly received by the right auricle and ventricle. Experiments upon frogs and cats and dogs—the latter animals under anæsthesia—showed that in these the pericardium supported the pulsating heart, especially the right heart and during diastole. The heart was supported not only when a large quantity of blood was forced into it but also when it was passively dilated by pushing the chloroform.

We must now consider the circumstances which lead to such engorgement of the right heart that it is necessary that it should be supported by the pericardium. In the first place every contraction of the muscles of the limbs will flatten the deep veins and suddenly inject into the superior or inferior vena cava, and so into the right heart, a very considerable quantity of blood. The venous blood is returned from the legs against the action of gravity by this pumping action of the skeletal muscles of the legs. The most frequent and powerful cause of sudden engorgement of the right heart with blood, however, is the active working of what has been called by Dr. Leonard Hill and myself the respiratory pump. The object of this pump is to raise the blood from the capacious sinuses of the abdomen and when the vascular flow is rapid, as it is during active exercise, each stroke of this respiratory pump will drive a considerable wave of blood through the heart.²

We must pause for a moment to consider this respiratory pump and the important action of the skeletal muscles in carrying on the circulation. The researches which have been carried out by Dr. Leonard Hill and by myself would seem to show that in man the heart cannot carry on the circulation alone for any length of time in the erect position—that the action of the skeletal muscles is necessary to secure the return of venous blood from the legs and the abdomen. This is illustrated by the death of people who have been crucified. After a day or so œdema of the feet appeared; this spread until the heart was so depleted of fluid that it could not supply the brain. When hutch-bred rabbits are held up by their ears for five or 10 minutes the blood accumulates in their abdomen to such an extent that their brains become anæmic, their corneæ become insensitive and their pupils dilated, and they soon die.³ In man the heart is a pump which is situated above its reservoir or well, the abdomen (see figure, R.H.). At each relaxation it must fill itself from the great veins placed vertically below it. In all such pumping arrangements there are two means of raising the required fluid—either by sucking it up from above or by driving it up from below. Both these methods are employed to keep the heart supplied with venous blood. Every inspiration produces a negative pressure in the thorax (see figure, 2) and therefore in the right heart, and venous blood is sucked from the abdomen into the heart and is driven at each systole into the lungs. This suction pump is all-sufficient during rest and quiet exercise, but during violent exercise and exertion, when the full flood of the circulation is pouring through the dilated arterioles of the muscles and the stroke of the heart pump is both deeper and quicker, the blood must be lifted more rapidly and supplied to the heart in fuller volume. It is then that the abdominal force-pump comes into action.

When we consider the anatomy of the abdomen we find that the viscera are contained in a muscular sphere (see figure). Above is the dome of the diaphragm, and below the basin of the levator ani, closing the outlet of the pelvis. Its walls are formed in front by the recti and behind by the quadrati lumborum muscles, and at the sides by the oblique and transverse muscles. Its veins are large and flaccid and capable of containing all the blood of the body were they not supported by this muscular sphere. During vigorous exertion this sphere of skeletal muscle contracts with each inspiration and drives the blood from the great venous sinuses through the aperture for the inferior vena cava in the diaphragm, directly into the right heart. As this occurs at the same time as suction is exerted from the thorax the maximum flow of venous blood is effected. When we consider that the limbs and the abdomen frequently contract simultaneously and violently in such exertions as running or lifting weights it will at once be perceived that a large quantity of blood, probably amounting to one or two pints, may be forced into the heart in one huge gulp. It is under such circumstances that the pericardium prevents over-distension.

The pulmonary circulation during vigorous exercise is in a very large degree carried on by the respiratory movements. The pericardium supports the heart and prevents it dilating, whilst the abdominal muscles empty the venous sinuses of the abdomen through the right heart into the lungs. This is assisted by the coincident inspiration of the lungs which enlarges their capacity for both air and blood. During the ensuing expiration the lungs are squeezed empty like a sponge into the left ventricle, for the way to the right heart is barred by the pulmonary semilunar valves. The flow of blood through the liver is also brought about by respiratory movements. The descent of the diaphragm in inspiration squeezes the portal vein and empties the blood through the liver into the thorax. It seems probable that the great veins of the abdomen store a reserve of blood during rest and sleep, the muscular sphere containing these veins relaxing to a corresponding degree. Directly the person awakes or resumes active exercise the tonicity of the abdominal muscles is increased and this reserve of blood is driven into the general circulation and distends the heart and the arteries and veins supplying the active muscles.

We will now turn to certain points in the pathology of the pericardium and of the mechanism of venous circulation of which it is a part. When the pericardium becomes inflamed it becomes softened and we may infer that any sudden exertion or effort, especially a cough, will distend the heart with blood and cause it to press against the pericardium and so dilate it. I believe that a pericardium so dilated cannot recontract—that the heart within it will not in future receive support when it becomes engorged with blood, and that every effort will throw the heart out of gear, dilating its cavities and rendering its valves relatively incompetent and so producing cyanosis and breathlessness. I would therefore suggest that when the pericardium is inflamed the most absolute rest should be maintained not only until the inflammation has subsided, but until the inflammatory materials have organised into firm fibrous tissue. It appears to be of the greatest importance that pericarditis should be recognised early, for if the child be allowed to run about the pericardium may be irretrievably dilated. To return to the comparison of the pericardium and heart to the outer and inner case of a Dunlop tyre, if a hot day softens the outer case so that it bulges under the pressure of the air within we may patch it for a while, but it is irreparably damaged and dilated and goes progressively from bad to worse.

We will now consider one or two points about pericarditis with effusion. At first sight it appeared a most difficult problem how the effusion could exert sufficient pressure to dilate so tough a membrane as the pericardium without collapsing the thin auricles and preventing the ingress of venous blood. It was, of course, certain that venous blood must be introduced into the auricles at a higher pressure than that of the pericardial effusion or it could never enter at all. It was equally certain that this pressure must be high or it could not dilate so inextensible a membrane, but how this high venous pressure was obtained did not appear clear. Then the terrible dyspnœa of pericarditis with effusion occurred to me and I perceived that this was the violent working of the respiratory force-pump, driving a gulp of blood from the abdomen through the right heart into the lungs at each inspiration and forcing it once more through the left heart into the aorta at each painful expiration. Allowing no sleep by night or day it was one long struggle of the skeletal muscles to maintain the circulation through the heart in spite of the strangling pressure of the pericardial effusion. It seemed, moreover, probable that this terrible dyspnœa worked in a pernicious cycle and like the spreading œdema of the cerebral hemispheres tended to become progressively worse and worse. Each fluid wedge of blood driven through the compressed heart by the spasm of the abdominal muscles and the aspiration of the thorax acted across the incompressible effusion upon the softened pericardium, stretching it still more and making further room for fresh effusion. It then seemed clear that this type of dyspnœa was an imperative call for operative interference and the release of the effusion which was strangling the heart, for a pericardium so progressively dilated would probably not again contract so as to support the heart even were the effusion absorbed.

This matter seemed to be of such importance that I determined to investigate it experimentally, the research being carried out at the Brown Institution. A small rubber tube was ligatured into the pericardium of an animal under anæsthesia on Cohnheim's method but in such a way that the

² Journal of Physiology, 1897.

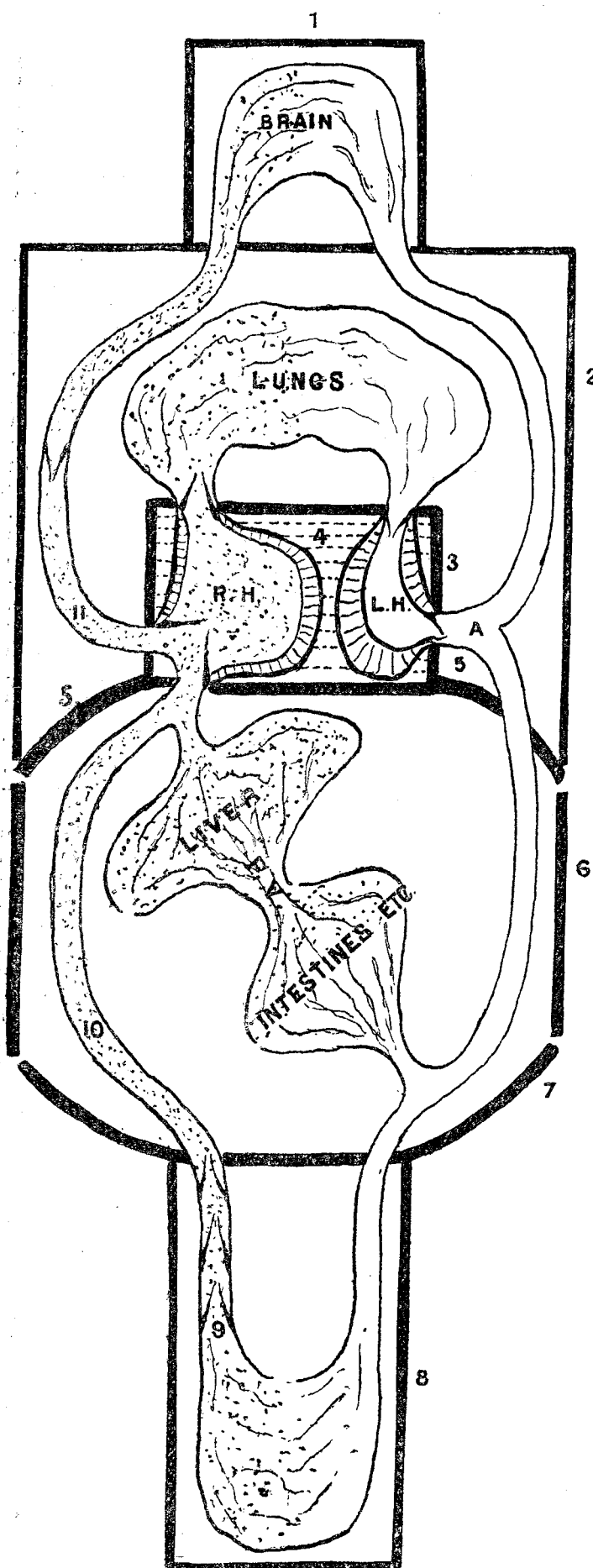
³ Dr. Leonard Hill: Proceedings of the Physiological Society, 1898.

thorax could be closed again and the animal resume normal respiration. Then warm oil was introduced into the pericardium, whilst a tracing of carotid and vena cava pressure was taken. The warm oil produced no effect whilst it filled the space in the pericardium which was not occupied by the heart, but directly it began to press upon the auricles the vena cava pressure rose and the arterial pressure fell and violent dyspnoea then followed, of the type which I had so hopelessly watched in several cases of pericarditis with effusion.

Each gasping inspiration of the thorax was accompanied by violent contraction of the abdominal muscles and deep descent of the diaphragm, whilst expiration was accompanied by a sound which indicated obstruction of the larynx to the escape of air, thus raising the thoracic pressure and driving blood through the compressed left auricle and so into the aorta. That each respiration was successful in forcing a gulp of blood from the veins to the lungs and from these to the aorta was shown by the wide oscillations of the carotid blood-pressure with each respiration. Directly the oil was allowed to escape from the pericardium the dyspnoea ceased, the carotid pressure rose, and the vena cava pressure fell to its usual low ebb, showing how beneficial the release of the fluid would be to patients suffering from pericarditis with effusion. I would therefore urge that dyspnoea of the type which I have described is the indication in pericarditis with effusion that the auricles are being pressed upon and that the effusion must be released. Of course this should be done by the open method and not by blind stabbing with a trocar and cannula, for there appears to be the greatest difficulty in distinguishing pericarditis with effusion from a widely dilated heart in a dilated pericardium. I know of a case where a young girl dropped back dead whilst the aspirating needle was being passed without an anæsthetic. A large pericardial effusion had been diagnosed, but on a post-mortem examination 15 ounces of pure blood were found in the pericardium and a wound in the heart. My experiments would seem, however, to indicate a diagnostic sign between these two conditions, although this has not been confirmed clinically. If tricuspid regurgitation is indicated by marked systolic pulsation in the veins of the neck the condition is probably one of a dilated heart in a dilated pericardium and not pericarditis with effusion. If the effusion exerts sufficient pressure to cause dyspnoea of the type which I have referred to the heart will be compressed and the tricuspid valve will scarcely show relative incompetency. If the heart is so dilated as to simulate pericarditis with effusion the tricuspid valve must be widely patent during systole and the systolic pulse will be communicated to the veins. This brings me to another point. In animals relative incompetency of the tricuspid valve can only occur in a slight degree whilst the pericardium is intact. When the pericardium is removed and the heart is engorged with blood by pushing the chloroform or squeezing the abdomen the valves at once become incompetent and the systoles of the right ventricle appear as oscillations in the vena cava pressure. It would therefore appear to be a mistake to attribute relative incompetency of valves to stretching of their fibrous ring. It would seem to be due to dilatation of the pericardium.

We will now turn to some of the results which follow from the failure of the skeletal muscles to efficiently raise the blood from the legs and abdomen to the heart. When the muscles of the legs fail to act vigorously the venous blood stagnates and produces œdema and sweating of the feet and in some cases varicose veins. It seems probable that loss of tone in the muscles of the calf is the cause of the association of varicose veins and sweating of the feet with flat foot. It is even more probable that varicose veins are produced by long-continued standing because the muscles do not periodically relax, allowing the deep veins to fill with blood and then pump it into the abdomen by a contraction. Pott's fracture, however, provides us with the best example of failure of the muscular venous pump in the leg. This fracture always involves the ankle-joint and is frequently followed by fibrous ankylosis. The muscles of the calf only serve to move the ankle-joint, so that when that joint is immovably fixed they cease to contract at regular intervals. As a result venous stagnation follows and the most obstinate œdema of the foot and leg, frequently going on to chronic ulcers. Ankylosis of the knee does not produce the same result because the muscles of the thigh act also upon the hip-joint and continue to contract at intervals to move that joint. A similar œdema follows ankylosis of the wrist.

The moral to be drawn from the powerful vascular action of the skeletal muscles would appear to be that regular exercise to maintain the tone and power of the muscles is essential to the well-being of everyone but more especially to people with a low blood-pressure. The development of the muscles of the abdominal wall is of the greatest importance, for a flaccid abdominal wall produces a languid venous return and this again reacts upon the circulation of the brain and produces lassitude and depression. Especially will such general exercises as walking and cycling cause a rapid vascular flow all over the body and eliminate waste products



Schema of the circulation of the blood in man in the erect position. 1, Skull. 2, Thoracic wall. 3, Fibrous pericardium. 4, Pericardial effusion. 5, Diaphragm. 6, Oblique and transverse muscles. 7, Levator ani. 8, Muscles of the legs. 9, Femoral vein and valves. 10, Inferior vena cava. 11, Superior vena cava. R. H., Right heart. L. H., Left heart. A., Aorta. P. V., Portal vein.

from the tissues. The production of syncope by the relaxation of the arterioles of the abdominal organs is a well-known phenomenon. The blood leaks through into the capacious portal vein and the ill-filled heart cannot supply the brain with blood. In fact, the man is bled into his abdominal veins. This could scarcely occur with a tonic and powerful abdominal wall, for a few deep breaths would rapidly return the venous blood to the heart. It is apparently in anticipation of such emotional relaxation that Marryat makes his privateersman and Kipling his soldier take a reef in their belts or tightly tie a handkerchief round their waists before going into action.

But there is the commoner instance of what may be called partial syncope seen in nervous or over-worked youths presenting themselves for examination. The diarrhoea and sickness point to abdominal congestion, and the mental confusion and loss of memory point to cerebral anæmia, whilst the thumping pulse and breathlessness indicate an ill-filled heart. Sometimes this condition is associated with albuminuria and forms one of the less recognised causes of the albuminuria of adolescence. The treatment of such people would appear to be obvious. There should be regular exercises, especially of the abdominal wall, and where such an ordeal as an examination has to be undergone an abdominal belt should be worn sufficiently tight to constrict the abdomen. I am convinced that neurotic and emotional people, who have as a rule a low blood-pressure would escape from a large part of their depression and lassitude were they daily to perform calisthenic exercises and develop the tone of their abdominal wall. In this connexion it is interesting to note the results obtained by Dr. Craig at Bethlem Hospital in cases of mania and of melancholy. He observed their blood-pressures, using for the purpose the sphygmometer invented by Dr. Leonard Hill and myself. In cases of mania associated with excessive restlessness he found the blood-pressure to be low, whilst in cases of melancholy with the sufferer sitting motionless in a stupor of grief he found the blood-pressure to be high. Further, he showed that in cases of mania the uniform pressure of a bath raised the blood-pressure and diminished the restlessness.

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NOTES ON A CASE OF UNILATERAL OPTIC NEURITIS.

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THE patient, a female, aged 21 years, consulted me on May 13th, 1897. A week previously she had severe and continuous neuralgia on the right side of the head. The sight of the right eye became dim and was lost in two days; she was menstruating at the time. Her previous history had been generally good, but menstruation was very irregular, two or three months often being missed. She was in the habit of taking a large amount of outdoor exercise. Three months ago she had a blow on the head severe enough to leave a black eye; six weeks ago she had measles badly. Her memory was extremely bad, but the patient said that it had always been the same. With regard to her family history her mother died three years ago from cancer of the larynx, otherwise there was no history of tumour, tubercle, syphilis, or other hereditary disease. The patient was above the average height, slight, and fairly muscular; her face was pale and looked pinched as if she had been suffering a good deal. She complained of pains in the head which shifted about but were mostly in the right temporal region where there was tenderness on pressure. The sight of the right eye was doubtful perception of light; of the left $\frac{1}{2}$ J. 1 at one foot. The pupil reflex of the right eye was not quite abolished. Ophthalmoscopic examination showed in the right eye intense congestion of the retinal veins. The optic disc was red and swollen and the outline was blurred. There were no hæmorrhages and no opaque exudation. The left eye showed no change whatever. I found three very bad teeth, two molars and one bicuspid, in the right upper jaw. Two of the teeth were extracted on the following day, the third two days later. Mr. Longhurst reported that there were abscesses at the roots of the first two, the third was carious but there was no

abscess at its root. 15 grains of bromide of potassium were ordered to be taken at night. On May 14th I saw the patient with Sir William Broadbent who found no evidence of disease in the chest or abdomen and agreed with me as to the probability of the teeth being the cause of the trouble. On the 15th she suffered much during the night from headache. Oleate of mercury and extract of belladonna were ordered to be rubbed into the head wherever painful. On the 16th the right eye had lost all perception of light; there was no pupil reflex and the swelling of the disc had increased. 10 grains of iodide of potassium and solution of perchloride of mercury were ordered. On the 17th I saw the patient in consultation with Mr. Victor Horsley who thought there might possibly be some taint of influenza in the case and recommended salicine. The iodide and perchloride made the patient sick and were discontinued. Mercurial inunction was commenced. The knee-jerks and other reflexes were normal. On the 20th strabismus was noticed. I did not see it myself, but was told that it was inward and affected both eyes. On the 22nd retinal hæmorrhages had appeared. On the 24th she was seen again with Mr. Horsley. We discussed the question of trephining but agreed to let it stand over for the time being; we ordered the patient to bed—up to this time she had been going about when she felt inclined. Some swelling of the eyelids was noticed. The urine was examined and found to be normal. The temperature was 100.4° F. On the 25th the swelling of the eyelids had increased. There was much pain in the eye, side of the head, and face, and there was tenderness on pressure on the eyeball and over the points of exit of the fifth nerve. An abscess forming in the orbit or antrum (?). Three leeches were applied to the temple. On the 29th the swelling of the lids had subsided, there was very little pain, and no tenderness on pressure. On June 2nd the menstrual period was due. The swelling of the disc was greater and the retinal hæmorrhages were much more numerous and larger; there were large clots scattered all about. On the 8th the patient had continued in much the same condition, having headache at times; the pain shifted about and did not remain fixed in any particular part of the head. Ophthalmoscopic signs were unchanged; she said she thought that her left eye was going wrong; up to this time vision was $\frac{1}{2}$ J. 1, field normal. On the 10th there was decided narrowing of the outer part of the field of the left eye. Ergotin was ordered. On the 11th Mr. Nettleship and Mr. Horsley saw the patient with me. In the left eye the outer half of the field for form was entirely gone, but light perception was still good; movement of the eye upwards was much impaired and outwards to a less extent. The ophthalmoscopic signs in the right eye were the same; the swelling of the disc amounted to 7 D. There was nothing abnormal in the left eye. The knee-jerks were not affected but there was slight ankle clonus. She was ordered iodide of potassium in 10-grain doses and to continue the mercurial inunction. Mr. Nettleship thought very badly of the case and was of opinion that the symptoms were due to a tumour probably growing backwards from near the right optic foramen. On this date the temperature which, with the exception of having been 100.4° on May 24th and 97° on the mornings of May 25th and 26th, had kept fairly level—the extremes being 97.4° on May 31st and 99.8° on June 7th—began to jump up and down in an unusual fashion between 101.6° on June 26th and 98° on July 11th. On the 15th movements of the eyeball were much improved; the condition of the retina and hæmianopia were unchanged. The patient was depressed and felt weak and languid. She was ordered pilula aloes c. myrrha. On the 24th movement of the eyeball had improved and the swelling of the optic disc was less. On the 28th the patient was flushed and excited. The left eye did not see so well but she could make out J. 1 with difficulty; she could not see the light of a match in the outer half of the field. On the 29th the mercurial inunction was finally stopped. On the 30th menstruation was due. The patient was much worse; she had a good deal of headache shifting about to all parts of the head. She said that everything looked dark; she could make out J. 16 badly; the pupil reflex was abolished. On July 1st I met Mr. Nettleship and Mr. Horsley in consultation. Sir John Williams also examined the patient and found nothing wrong. The patient was drowsy and evidently very ill. Iodide of potassium and iron were ordered. The urine examined was found to be normal. On the 3rd the left eye had lost perception of light but the ophthalmoscope showed no change. The blood examined was found to be normal. On the 5th Dr. Goodhart saw the patient and was of opinion