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Cardiac Hypertrophy with constriction and incompetency of the Mitral Valve following Rheumatic Fever ; sudden apoplectic seizure ; paralysis of whole of left side ; death in eight days. Obstruction of right middle cerebral artery by a firm mass of fibrin and softening of brain.

Edwin Davis ætat 28, of middle height, good muscular development, fair skin, light hair, blue eyes, admitted into the North Staffordshire Infirmary on the 25th. of June, 1858.

Previous History.—Has been by occupation a Miner, and has generally enjoyed good health, with the exception, of an attack of Rheumatic fever, from which he suffered 5 years previously, but he does not recollect that this was attended with pain in the chest or palpitation.

Present Illness.—Five weeks before admission, he first became conscious of pain in the precordial region, attended with some palpitation. Both were slight in amount, until a week preceeding his admission, during which period, however they had considerably increased in severity. He has also suffered during the past few days from cough and dyspnœa, which were greatly increased by exertion.

Present State, June 25.—Patient lying on back. Face jaundiced looking, especially about conjunctivæ. Skin covered with a profuse perspiration, but temperature to hand of observer does not appear abnormally high. No pain in joints, nor œdema of limbs.

Examination of Thorax.—Both lungs give good resonance on percussion, anteriorly and posteriorly. Loose mucous râles are heard at both bases posteriorly, vocal resonance nowhere exaggerated. Coughs a good deal, expectoration loose, glairy, transparent mucus, mingled with some black masses diffused through the sputum.

Heart's impulse diffused.—Point of maximum impulse is in 6th. interspace, about half an inch outside a line drawn vertically from the nipple. It is felt also very strongly at the ensiform

cartilage ; action very irregular both in force and rhythm. Both superficial and deep percussion dullness extends abnormally to the right and left of sternum. There is considerable pulsation in the external jugular veins, and that on the left side refills slowly from below. At left apex a slight thrill apparently diastolic may be felt.

On auscultation the first sound at the left apex, is almost entirely masked by a soft blowing murmur which commences before the systole, and is continued into it. It is conducted towards the ensiform cartilage, at which spot it attains its maximum intensity. It is heard with diminished clearness as the stethoscope, is carried up the sternum, and is lost at the midsternum, at the level of the 3rd. cartilage, at which point the first sound is heard free from murmur, but somewhat muffled in tone.

The second sound is frequently entirely inaudible at the apex, during a period corresponding with several impulses. When present it is normal in tone and is unattended by any murmur. It is more accentuated at the 2nd. left, than at the 2nd. right cartilage.

To keep his bed. To have middle diet, and to take the following—*R. Vin: Antim: Tart: m××, Mist: Salin: 1 oz. ter die; R. Tr. Camph: Co: 1 oz. Vin. ipecac m ×, Mist: Camph: 1 oz. ter die.*

June 26th.—Patient feels much better, cough is less troublesome. Action of heart more regular, less intermission of 2nd. sound. To take Pill Hydrarg g. v. nocte maneque.

June 30th.—Gums are affected by the Mercury. *To take the pill once a day.*

July 4th.—Gums are considerably affected. *To discontinue pill.*

July 6th.—Chest is entirely free from r  le. Murmurs of heart are unchanged.

July 11th.—Was found this afternoon by a fellow patient in the washing room, leaning on a table, in a state of insensibility. When seen by Mr. Alcock, the pulse was almost imperceptible, face pale ; extremities cold ; and the left side of the body was found to be paralysed. Some sp. Ammon. Arom. was administered.

July 12th.—Patient lying on back. Face pale and covered with perspiration. Breathing laboured, and at times slightly stertorous. He is apparently unconscious, but can be roused by loud speaking. He moans at intervals, and complains of pain in the head and back. Moves right hand uneasily to head. Mouth slightly drawn to right side. When told to protrude tongue, he makes an effort to do so, but cannot bring it beyond level of teeth. No convulsive movements observed to day, (Mr,

Alcock reports that when first seen, there was a slight twitching in the muscles of the right arm.) The left arm when raised by observer, falls again as an inert mass. Fingers of this hand are slightly flexed, but do not close upon an object placed upon their palmar surface. There is a slight spontaneous flexion of the biceps of the left arm, but its resistance can be overcome with great ease. No reflex movements can be excited in the left arm or hand. Left leg lies extended and entirely motionless. Slight reflex movements can be excited in it by tickling sole of foot. These are chiefly observed in the extensor tendons of foot, but exist also, though to a less degree in the extensor cruris. Motion in right arm and leg perfectly preserved.

Sensibility entirely wanting in left arm. When left side of face is pinched, the patient gives signs of uneasiness. Sensibility apparently perfect on right side. Has passed urine voluntarily two or three times since attack. Has occasionally recognised attendants, and spoken to them by name, but lapses into unconsciousness after a few minutes. Eyelids habitually closed, right more perfectly so than left. Strabismus to right on both sides. He rolls eyeballs upwards, and to the right, but does not seem able to bring eyeball beyond middle line towards the left. Pupils of moderate size, contract equally under light, but dilate again, and finally contract strongly. Some uneasiness manifested under light. Heart's impulse excited, action very irregular, both in force and rhythm. Pulse 100, equal on both sides in radials, carotids, femorals, and arteriæ dorsales pedum. Murmur with first sound unchanged since admission. Bowels not open since yesterday, has vomited once yesterday, and once to-day, swallows easily. 4 *Leeches* to each temple, *Enema Terebinthin, c: ol: Ricini Statim.* *Strong beef tea to be given.*

July 13th.—Great rigidity noticed to-day in muscles of neck on left side. Any attempt to move head causes great pain. Contraction of biceps of left arm slightly increased but is easily overcome. No rigidity in left leg. Enema has not been returned. Passes urine voluntarily. It is small in quantity and loaded with lithates.

July 14th.—Contraction of muscles on left side of neck increased. Some difficulty of deglutition observed last night, is increased to-day. Fluids given by mouth return through nose. A second enema of simple barley water was given this morning, and returned unattended by fecal matters. *To have ol. Croton. Tigl: gtt: 2 statim.*

July 15th.—Coma much deeper than before. Face more flushed than at previous reports. No perspiration on face. Rigidity

of muscles of neck increased. Pupils contract less under light. Pulse 100, small and very irregular. Has had one copious loose stool.

July 17th.—Much more consciousness exhibited by patient to-day. Speaks articulately. Recognises attendants and addresses them by name. Rigidity of muscles of neck greatly increased, that of biceps is slightly increased. Left leg still perfectly flaccid. Reflex movements are less easily excited in it. Pulse 84, very irregular and weaker than at previous reports. July 19th.—Patient died at 1 a.m. this morning. From report of House Surgeon it appears that he gradually sank into a deep and fatal coma. No convulsions were observed.

Post Mortem, July 20th.—36 hours after death. Weather warm and moist. Right arm flaccid. Rigor mortis persistent in left arm and leg. Very strongly marked in muscles of left side of neck.

Skull opened.—Bones of normal thickness. Dura Mater very strongly adherent to calvarium. Superior longitudinal sinus contains a fresh clot of blood. On left side membranes separate easily, but they cannot be removed on right side without laceration of the substance of the brain. Glandulæ Pacchioni much enlarged and strongly adherent to dura mater. Considerable sub-arachnoid effusion on base at right side. Veins on surface of right hemisphere are much congested. On slicing brain from above downwards on left side, the tissue is found perfectly normal, but on right side there is found at the depth of about $\frac{1}{2}$ an inch from the surface, a reddish grey spot, situated principally in the white substance, but extending also into the grey matter of those convolutions which pass into the fissure of Sylvius. This is found increased in extent as sections are carried deeper into the substance, and on opening the right lateral ventricle the discoloured portion is found to be in continuity with the right Corpus Striatum. This body is greatly softened, breaking down under a slight stream of water; it is of a dirty reddish grey colour, and the softening extends through its entire substance. The discoloured portion of the hemisphere, is also much softened and breaks down easily under a stream of water. The softening extends to the grey matter of the convolutions on the lateral surface of the hemisphere. Parts in lateral Ventricle on right side normal. No accumulation of fluid in the ventricles. Rest of brain perfectly healthy. On examination of the arteries at the base of the brain, the middle cerebral artery of the right side, is found obstructed at the point of its second bifurcation, by a firm coagulum of fibrin. The obstruction extends through both the branches leading from this point, into the fossa of Sylvius, and

into the smaller branches entering the substance of the brain at the locus perforatus anticus, as far as they can be traced. The Coagulum with which they are filled is firm and hard, and adheres strongly to the walls of the vessels. The basilar arteries, and other portions of the circle of Willis are quite free. Microscopic examination reveals extensive fatty degeneration of the ganglion-cells of the right corpus striatum, while in many parts of this body, nothing but fatty debris can be found. The capillaries and smaller arteries of this part have also undergone a highly marked fatty degeneration of their walls.

Thorax Opened.—Right Lung.—Bronchi considerably injected, even into the finest divisions, and contain much reddened mucus. In the posterior part of the middle lobe there is a spot of pulmonary apoplexy of about three inches in diameter, of a dark purple colour, hard and resistant to finger, and evidently recent. In the centre of this spot there is found a branch of the pulmonary artery occluded by a fibrinous coagulum which is firmly adherent to its walls, but its further ramifications cannot be satisfactorily followed. Pulmonary tissue in other parts healthy.

Left Lung.—Characters of Bronchi same as on right side, Pulmonary tissue perfectly healthy throughout.

Heart.—Amount uncovered by lung abnormally large. Pericardium opened, contains about 2 oz. of a clear yellow serum. Heart is very much enlarged. This is especially the case in the right auricle, which measures $1\frac{3}{4}$ inches in length, by $2\frac{1}{2}$ inches in breadth; and when distended with blood $1\frac{1}{2}$ inches in depth. Breadth of heart at base, 5 inches; length of ventricle from Conus Arteriosus to apex $5\frac{3}{4}$ inches.

Right Auricle.—Greatly distended with a dark clot of firmly coagulated blood. Walls much thickened. Musculi pectinati strongly developed. Considerable thickening of walls of venæ-cavæ superior and inferior, both at orifice of auricles, and extending for some distance into veins. In auricular appendage there is a firm fibrinous coagulum, of a dark reddish grey colour, very firmly adherent to walls and to musculi pectinati, and presenting the appearance of having existed for some time previously to death. It nearly fills the auricular appendage, and extends also into the auricle, where it is firmly interlaced among the musculi pectinati. Its edges are rough and irregular.

Tricuspid Valve, admits easily the thumb, and the tips of four fingers of observer. Right ventricle is greatly distended with a dark clot of firm well coagulated blood. Walls of ventricle in central part are $\frac{3}{8}$ of an inch in thickness, not quite $\frac{1}{4}$ inch at apex. Considerable thickening has taken place at the edges of the flaps of the valves. Chordæ tendineæ attached to the an-

terior flaps are greatly thickened and shortened. Aortic valves close perfectly, and retain a column of water in the aorta.

Left Auricle, not nearly so much hypertrophied as the right, but still enlarged greatly beyond normal size. The auricle is almost filled with a firm fibrinous mass, strongly adherent to walls and presenting characteristics similar to those found in analogous situations on the right side. The free edge which is very rough and uneven, projects for some distance into the auricle.

Mitral Orifice, very much contracted, barely admitting tip of little finger of observer. The contraction of the orifice is due to the adhesion to one another of the upper two thirds of the flaps of the valve at their margin, and of about two lines in depth of the opposing surface on each side. The remaining portions of the free edges of the flaps are greatly thickened and almost cartilaginous to the feel. No warty vegetations on valve. Aortic and Pulmonary valves are quiet healthy. At posterior surface of left ventricle near the apex, there is a patch of recent lymph, firmly adherent to visceral pericardium, and of about $\frac{1}{4}$ inch in diameter. No other appearance of recent pericarditis is present. Weight of Heart, 12 oz.

Stomach.—Mucous Membrane thickened, and presents numerous spots of hæmorrhagic erosion, which are especially frequent in the neighbourhood, of the Pylorus. Duodenum strongly injected.

Liver, presents the "Nutmeg" appearance to a highly marked degree, depending on congestion of the hepatic veins. Tissue not fatty, but loaded with bile pigment.

Gall Bladder, greatly distended by a dark coloured bile, which can scarcely be forced into the duodenum, by the strongest pressure on bladder: but when a drop is finally forced through the opening of the Duct. Com. Choled, no plug of mucus is observed to be expressed. A highly marked contraction is found at the upper extremity of the cystic duct, which is thickened, but no other obstruction can be found.

Other Viscera.—Healthy.

Commentary.—The Clinical history of the case here narrated, presents several points of peculiar interest, on which it may be worth while to bestow a little attention. A young, well nourished and to all appearance, a strong and healthy man, after suffering for a short time from pain in the Precordial region, and palpitation, is suddenly attacked by paralytic symptoms, and dies within a few days of the seizure. The circumstance would be in itself sufficiently remarkable to attract attention, had not cases of a similar nature been fully recorded, and elaborately elucidated

by other observers, more especially by Professor Virchow, of Berlin, and more lately by Kirkes, in this country, and by Bouchut in Paris. Apoplexy as a concomitant of cardiac disease, has indeed been long recognized as a not unfrequent cause of death, but those whose attention it had especially attracted as Rostan and Bouillaud, have dwelt more particularly upon hæmorrhagic effusion into the substance of the brain or its membranes, while of the numerous observations made upon softening of the substance, as recorded by Abercrombie and Andral, and in the elaborate work of Durand Fardel on this subject, not one has been traced to the condition here existing, and which, there can be little doubt, stands to the softening in the relation of its immediate cause—I mean the occlusion of the Cerebral Artery by the plug of hardened Fibrin, which is found in this case, obstructing the supply of nutritive fluid to the very part in which the alteration of nutrition has occurred. Carswell indeed pointed out that in some cases of the so called white softening there was more or less obstruction to the circulation in the cerebral arteries, and Abercrombie drew a parallel between this condition and gangrene of the extremities; but Carswell's generalisation was shaken by the results of other enquiries, which tended to show that this state of the arteries was not an invariable concomitant of the disease, while the question of the inflammatory or non-inflammatory nature of white softening of the brain, has remained to the present day a subject of dispute among the leading pathologists both at home and abroad.

Into this question it would be foreign to my purpose at present to enter, but it may not be unprofitable to pass in review, some of the principal points of Clinical interest, which this case affords.

From the physical signs which I have above detailed, I had no doubt that the patient was suffering from Cardiac hypertrophy and Mitral regurgitant disease. There were however two points concerning which some doubt was felt in the diagnosis, and which were of considerable importance, as affecting the plan of treatment to be pursued, which would be materially modified by the judgment to which we might arrive, as to whether it was recent or of old standing.

(1)—The pain and Cardiac symptoms had only existed—according to the account the patient gave of himself—for a period of five weeks.

(2)—The Systolic murmur heard was soft in its characters. Both these facts were in favor of the recent origin of the affection; against them were to be urged,

(1)—That the patient had had an attack of acute Rheumatism five years previously, and that as a general rule, when heart disease exists after such an attack, it is usually a result of the previous affection.

(2)—The evident hypertrophy was also in favor of the opinion that the disease was of long standing.

(3)—The murmur heard at the apex was presystolic in time, pointing to the diagnosis of a constriction of the mitral valve, an affection which requires a considerable time for its evolution; and the state of the veins of the neck also indicated that there was some regurgitation through the orifice of the tricuspid.

The balance of this conflicting evidence, was a point of serious consideration; but in the absence of positive certainty, though inclining strongly to the opinion that the disease was of old standing, I thought it desirable on the possibility that there might be at any rate some recent recrudescence, to give the patient the chance of the benefit which a mild course of Mercury might afford, in removing the effects of any inflammatory action which might be established; and I did so on the ground, that in the face of the experience of such men as Latham and Hope, who have spoken so strongly of the advantages they have seen to accrue from its use, we are not justified in abandoning this remedy until a further and conclusive series of cases has established its inefficiency, which, much as the action of this drug has been doubted of late, has not as yet been done to the satisfaction of the profession.

Under the influence of rest in bed, low diet, and expectorants, both the Cardiac and Pulmonary symptoms, from which the patient suffered on admission, became in a few days greatly alleviated, but *no* change took place in the character of the cardiac murmurs; and when after 10 days administration of the Mercury, the patient's gums became considerably affected, I finally discontinued the use of the drug, employing only expectorants, and allowing the patient to take a more nourishing diet, he continued gradually to improve for 5 days, during which time no change was observed in the sounds of the heart, and the patient was rapidly approaching an apparent convalescence, when he was seized with the fatal attack, the particulars of which, I have detailed. The collapsed state into which he at first passed, prevented the employment of any of the active measures usually resorted to in apoplecticform seizures. On my visit on the following day, the probability of the nature of the affection being such as the post mortem appearances revealed, at once presented itself to my mind, and led me to refrain from any very active measures of treatment,

There are some points of contrast to be observed in the mode of seizure in this case, to the symptoms usually observed in apoplecticform attacks resulting from acute inflammatory softening, on which it may not be uninteresting to dwell.

The absence of prodromata is remarkable ; for so commonly is "acute softening"—as it has been termed—preceded by various anomalous sensations in the head or organs of sense, or in the motor or sensory nerves of the body, that Durand Fardel, in his elaborate essay on this subject, relies much upon their absence as a diagnostic sign in favour of hæmorrhage. On referring to the cases of this nature recorded by Virchow, Kirkes, Simpson—who also quotes one from Dr. Burrow's work—and to the case recorded by Dr. Todd, of a paralytic seizure, resulting from occlusion of the carotid by a plug of fibrin escaping from an aneurism of the subclaran artery, I find the same absence of any symptom referable to the nervous system preceding the apoplectic attack, in all cases recorded to which I have had access. We have therefore an additional element of difficulty in diagnosis, quoad the symptoms, introduced into that already sufficiently obscure subject, the diagnosis between hæmorrhage and softening; though, from the light which has been already thrown on the class to which this case belongs, it may not prove so perplexing at the bedside, as might at first sight appear; for there still remains a sufficient number of points on which to form an opinion; and indeed for the future, the absence of prodromata may be taken in conjunction with a previously existing heart disease, in cases when apoplexy occurs suddenly in a young and healthy subject, to favour the opinion, that a lesion of the kind now under consideration, has in all probability occurred, since, though Andral was of the opinion, that cardiac disease was a frequent determining cause of cerebral hæmorrhage, other observations have not borne out the correctness of this supposition, and indeed Walshe's analysis of the recorded cases, would if anything tend to show that cardiac disease, was a more frequent attendant on cerebral softening, than on cerebral hæmorrhage, though in neither is the connection so constant, or so direct as many have supposed; *i. e.* only $7\frac{1}{4}$ per cent. of persons dying of disease of the heart had cerebral hæmorrhage, while 39.9 per cent. have it in cases of softening—or from another calculation, 45.5 per cent. of persons dying of apoplectic seizure have some disease of the heart, but 25.5 per cent. of all old persons, taken indiscriminately, have some organic cardiac disorder. Suddenness of attack seems an essential feature in all the classes of affections resulting from the cause of which we have an instance before us; whether the seat of the obstructed artery be in the brain or in the extremities. Several very interesting and instructive examples are recorded in

Dr. Simpson's paper, where the crural, femoral, and iliac arteries were plugged by masses of fibrin formed in veins and carried into those positions by entering the current of the circulation; in all of these pain in the limb, coldness and paralysis were the essential symptoms. Other cases where the obstruction took place in the pulmonary artery, are recorded by him and Mr. Paget, who first drew attention to this affection of the lung, though at first wrongly estimating its meaning, and also by Professor Virchow; and it is interesting in cases when the arteries of the extremities were affected, to note the difference between the local paralysis which ensued, and that when the supply of blood was cut off from the central organ of innervation, the one class belonging in all probability to the category of muscular paralysis, the latter to a paralysis of innervation. It is interesting also to note, that when the attack was caused by interruption of the cerebral circulation, collapse has been the almost invariable symptom at the *outset*, differing thus in a remarkable manner from either hæmorrhage, or acute inflammatory softening, where the previous or concomitant determination of blood to the brain, produces the pressure or congestive symptoms, which have been ordinarily recognised as the distinguishing features of the apoplectic seizure.

This abruptness of invasion may also be noticed as distinguishing clinically, this class of softenings of the brain, from those in which there is an obstruction to the circulation from disease of the arterial coats, in which case the cause being a gradually progressing diminution of the calibre of the vessel, the consequent impaired nutrition of the cerebral substance, and the symptoms to which it gives rise, are also gradual and insidious in their invasion.

The attack once established, the symptoms which were subsequently observed were tolerably distinctive of softening, and it is remarkable that they partook in some degree of the characters which are supposed to be somewhat distinctive of softening of the inflammatory kind. Among these may be noted the deep seated pain in the muscles of the upper extremity on the paralyzed side, and the gradually increasing rigidity which attained at length a very marked degree in those of the neck, and existed also, but to a less extent in those of the arm. They were noticed though to a slight extent, the day after the seizure; and had it not been for this early appearance, its existence could hardly have been thought characteristic of softening, since hæmorrhage into the central parts of the brain, is not unfrequently attended by this phenomenon as a *secondary* symptom, in consequence of softening set up around the clot. As a primary symptom according to Durand Fardel, it is not seen in cerebral hæmorrhage, unless the extravasation takes place to a very great extent, entering by the

ventricle and breaking down the septum lucidum, in which cases the paralysis attendant instead of being unilateral is general.

The deep seated pain is said to be very characteristic and valuable as a diagnostic symptom, though I must confess that in some other cases of softening, which have fallen under my notice, this has been almost entirely wanting. The anæsthesia on the paralysed side of the face was not at all well marked, and in this particular, the case in question forms no exception to what is usually observed. It was much more complete in the limbs, according in this respect to the law which Virchow has pointed out, in contrasting embolic paralysis of a single extremity, with that produced by cerebral lesion, arising from the same cause, viz :—that in the former class of cases, motion suffers more than sensation, while in the latter the reverse condition obtains,

The intellectual state was also characteristic of softening; the patient lay in a state of apparent coma, but could be roused with great facility, even recognising attendants, and calling them by name, but again speedily relapsing into a comatose condition. The recovery of consciousness before death is a very striking, though by no means an unusual phenomenon, difficult of explanation as the cause may be. It is an important fallacy to guard against in forming a prognosis, as in the case in point, where 13 hours before the fatal termination, there was apparently a striking amelioration in most of the patient's symptoms.

There is one other point deserving of notice, as its cause is by no means clear, and that is the perspiration which appeared so freely, during the first days of attack. Virchow has observed a similar phenomenon in three cases, and attributes it to some peculiar derangement of the nervous system. I would suggest the explanation, that as in all these cases, and especially in the one now under consideration, it has been always noticed most abundantly about the head and face; whether its appearance may not be due to the implication of the sympathetic system of nerves, which ramify so abundantly around the cerebral arteries, and whose influence on the vessels of the skin has been so clearly shown by the experiments of Bernard, and by Brown Sequard.

There are two features in the pathological appearances, which are interesting in their relation to the affection of the brain. The most important of these is the spot of palmonary apoplexy found in the right lung, and associated with an occluded branch of the pulmonary artery. This is now generally believed to depend on the same cause as the cerebral affection. The precise spot of the primary occlusion could not be satisfactorily traced, but the state of the coagulum in the artery left me no doubt that it had been formed during life. How such occlusion should produce hæmorrhage

hage into the pulmonary tissue, has not as yet been fully elucidated, but Professor Virchow gives as a very probable explanation, that it is due to undue pressure on the arterial circulation, giving rise to rupture of the smaller vessels. The source must be sought for in the right side of the heart or in the veins. The veins were free, but we had in the right auricular appendix, a condition precisely similar to that found in the left, viz ;—a mass of old fibrinous coagulum, and it was probably to portions of these becoming loosened, and entering the circulation, that the whole of the pathological phenomena which we have here passed under review, are to be ascribed. Dr. Kirkes has noticed in some of these cases, spots of hæmorrhage in the mucous membrane of the stomach, he attributes this to the same cause as the hæmorrhages observed in the lungs, and supposes it to be due to occlusion of the small arteries of the mucous membrane.

These hæmorrhagic erosions of the stomach are very common in cases of cardiac disease. I have examined them very carefully in numerous instances, but have never found an indication of the existence of the cause to which Dr. Kirkes ascribes them. They are most frequent in cases where the tripuspid valve is deficient in its action,—as was the case here,—but are also very common in cases of cirrhosis of the liver, and are I am inclined to believe simply the result of obstructions to the return of blood from the organ, which cause rupture of the smaller vessels.

In illustration of the case, a preparation of the diseased brain of the patient was exhibited.



