with sulphate of copper, and the eye bathed twice daily with a solution of alum or of tannic acid, which latter, in the proportion of four or six grains to the ounce, is very useful. The alum and tannic acid have been prescribed in combination; but a curdy precipitate of tannate of alumina is thrown down, and a large portion of tannin is rendered inert. By the treatment mentioned, the cicatrix soon acquires firmness, and the eye becomes free from mucous discharge.

The nature of the change by which such opacities, or such heaping up of abnormal tissue, as that described in case II, is produced, has been well explained by Mr. Bowman: "The nature of the nutritive process in the lamellated tissue is such, that this tissue recovers itself in a great measure, by timely treatment, from almost any amount of inflammation, and consequent effusion, which falls short of actual destruction of its elements; but when these elements are at all displaced or consumed under the morbid process, then permanent opacity is very likely, and, indeed, almost certain, to follow; for so artificial is the mechanical arrangement of the elementary lamellæ, on which the transparency of the cornea depends, that when their substance is once removed, its place cannot be supplied with a tissue of an equally elaborate organisation. The new material, though its bulk and strength may be equivalent to those of the old, is fibrous instead of being lamellated, and opaque instead of being translucent. It contains a considerable quantity of yellow tissue, intimately mixed with the white, and both most irregularly interwoven and ill-developed—ready to become the nidus of small granules of earthy or fatty matter, such as readily settle in parts of deteriorated structure."

The presence of such earthy or ossific deposit in the eye is far from uncommon; osseous cataract being perhaps the most frequent example. Of this, a very excellent specimen was exhibited at the Pathological Society by Mr. Canton, in May 1851; the capsule of the lens was completely converted into calcareous material, and osseous patches had been formed in several places between the retina and choroid It was taken from a woman aged 73, who, thirty years before her death, had received a blow on the eye, followed by wasting of that organ.

It does not necessarily follow, that ossific deposits in the eye should be attended with neuralgic pain; but they are so in a large number of cases. In the first volume of the *London Journal of Medicine*, four cases were related by me, which illustrate this point. The case above described affords another example; and one very similar is mentioned by Mr. Bowman. A girl, aged 14, was brought to that gentleman, suffering from severe inflammation of the eye, which had been partially sunk several years before, and in which no perception of light remained. The irritation was very great, especially on every movement of the lid: on examination, a hard, angular piece of bone was observed, imbedded in the front of a dense opacity of the cornea, and projecting beyond it, quite bare. The fragment, which was of the size of a large pin's head, was easily removed with a pair of forceps, and the patient speedily got well.

I am an advocate for the use of the knife, in preference to escharotics, for removing leucomatous growths or tumours from the eye; it is more sure, more speedy, more manageable, and less painful. In a few cases which have fallen under my notice, where inflammation has followed excision, it could generally be traced to long continued and abortive attempts to pare off the growth, accompanied with violent pressure on the eye to steady it. If chloroform be used, the operation can be performed at once with neatness and a light hand, and the relief is immediate.

19, Berkeley Square, July 1853.

## CASE OF RUPTURE OF AN ANEURISM OF THE TRANSVERSE PORTION OF THE ARCH OF THE AORTA INTO THE TRUNK OF THE PULMONARY ARTERY.

By W. BIRD HERAPATH, M.D.

On a Sunday evening, at half past nine, P.M., I was summoned to attend Mr. Charles M—— R——, aged 53, who was then residing near me. I found him in bed; he was complaining of pain in the epigastrium, and of disposition to nausea, which had been increased to vomiting by the ingestion of warm water. He had an uneasy sensation in the bowels, with slight relaxation also. The substances ejected from the stomach were highly charged with acid. He also suffered from thirst; the tongue was, however, clean; the pulse was quicker than it ought to be, 100, soft and regular. The skin of the extremities, where exposed, was cold and moist, but under the bedclothes, warmer. He had no headache. The respiration was somewhat quicker than usual; he also had a slight cough, with frothy mucous expectoration; he compared the pain in the epigastrium to a constant burning sensation. He had often suffered from it before; and had hitherto always obtained relief from a draught of cold water. This had failed, as well as the emetic treatment on the present occasion.

The history of the case was the following, as given me by the wife. He was a brewer and distiller, and had held a situation in a neighbouring brewery during the last six months. He was rather fond of Scotch whiskey, and generally had several glasses of "toddy" during the day at intervals; but he was never intoxicated. The day previously, he attended to his duties as usual, and came home in the evening in his ordinary state of health. He had supped on beef, which was not fresh or good. The child had also eaten of the same dish, and not liking it, declined taking any more. Mrs. R. could not eat it, as it tasted strange and "fusty". He had tried to go to church on the Sunday, but came home again faint, and had taken no food. Taking all these circumstances into consideration, I thought that he laboured under gastralgia, from acidity; that the faintness and depression were produced by the emetic and exertion, together with abstinence from food and stimuli during the day.

I prescribed a pill containing four grains of calomel and one grain of opium, to be taken directly, and an antacid saline aperient, with some compound spirit of ammonia, every two hours; a mustard cataplasm to the epigastrium, and bottles of warm water to the feet; a little arrowroot and brandy as diet. At this time he only sent for me in consequence of the advice of a friend: the patient himself did not think it necessary to have a medical man.

About a quarter before eleven the same evening, I was again summoned to this patient, who was stated to be much worse. Thinking it to be over anxiety and timidity, I at first advised perseverance with the remedies; but a second messenger, following on the heels of the first, said that he was certainly dying, and begged my instant attention. In crossing the road, I met a third on the way to me. This had prepared me for a great and sudden change; but I certainly was not a little surprised to find my patient moribund. He was pulseless; cold; clammy; the face was pallid; the eyes glazed and insensible; no reflex excitomotory phenomena of eyelids; and the jaw fallen. He made two or three convulsive gasps, and all was over. I attempted to restore animation by placing the head low, and applying burnt and smouldering rag under the nostrils, whilst I at the same time induced and kept up artificial respiration, by alternately compressing the thorax, and relaxing the pressure on the ribs. The heart was auscultated, but it had ceased to beat: and all attempts to restore animation were unavailing.

Upon more calmly questioning the spectators of the last moments of this sudden calamity, the following facts were elicited. He had complained of being worse and in more agonising pain, and of having more difficulty in breathing.

All at once he was seized with a "few spasms, *not* convulsions"; he became blue in his face; made some rattling noises in the throat; a cold clammy perspiration stood on his face in large drops; he groaned repeatedly as if in great pain; this groaning gradually became a dull moaning sound; then the countenance and skin assumed a dull slate colour; the eyes rolled about spasmodically; the patient appeared to fall into a comatose, stupid, or unconscious condition and rapidly assumed that appearance in which I found him. In the meantime he had been raised from the recumbent to the sitting posture, and the features were bloodless and pallid when I came in.

The whole appearances to me were those of a combination of syncope with asphyxia. As, however, considerable doubt remained as to the cause of these symptoms, and even some suspicion subsequently arose as to whether he had not committed suicide in consequence of some existing mental disquietude, it became my duty to acquaint the coroner with the whole circumstances of the case and await his decision, before I proceeded to request a *post mortem* examination.

In my letter to him, I hazarded the conjecture that one of four causes produced death: suicide or other poisonous agency; rupture of the stomach or duodenum from perforating ulcer; laceration of an aneurismal sac and internal hæmorrhage; or an epileptic attack merging into apoplexy. In the absence of further evidence, however, a decided opinion could not be arrived at.

Examination of the Body, by order of the Coroner; eighteen hours after Decease. The surface of the abdomen was thickly covered with fat, and the whole body was plump and well nourished. There were no appearances of violence; no ascites or anasarca; on opening the peritoneal and thoracic cavities, there were no traces of peritonitis, or any evidence of hernia, intussusception, or other intestinal displacement. There was no hæmorrhage; no rupture or perforation of the stomach or viscera. The pleural and pericardial cavities were free from fluid.

The coats of the stomach and smaller intestines were uniformly engorged with blood; all the branches of the portal system were in a highly congested state.

The lungs were equally gorged with blood. The vesicular structure, however, was everywhere crepitating; and there was no appearance of blood having been extravasated into the pulmonary structure, or of recent pneumonia.

The stomach and œsophagus were carefully dissected out, as it at first appeared necessary to undertake a chemical examination of their contents. There were no traces of any corrosive irritant, however, upon the œsophageal mucous membrane. The stomach contained about a wineglassful of dark coloured fluid, somewhat bloody. The gastric mucous membrane was everywhere gorged with blood; many ecchymosed patches of small extent being evident. The whole surface was dotted over with these, and some long narrow streaks of ecchymoses marked the rugæ, and were very apparent; and three small ulcerations existed at the lesser curvature. These appearances were so similar to those produced by an irritant corrosive poison, that considerable care was taken of the whole viscera and their contents. The intestinal mucous membrane presented the same pathological appearances, more especially that of the duodenum and the jejunum. The large intestine was empty, the coats were covered only by a whitish creamy mucus; they were destitute of fæces and fœtor. There was a small portion of fæces in the cæcum.

The heart was now examined; and then arose a change in the whole aspect of the case. The pericardium was empty, and not adherent; the heart was flabby and large; its muscular coat was pale. The aorta was dilated very considerably, and was strongly adherent to the pulmonary artery. The lining membrane was diseased, being rugous, roughened, and atheromatous throughout. At one portion of this vessel a lacerated opening was found. This was situated on the anterior and inferior wall, at the junction of the transverse with the descending portion of the arch of the aorta; it was three-quarters of an inch in length and about one-third of an inch in width. The edges were everted. A probe used very gently passed through it into the cavity of the pulmonary artery; around the borders of the opening in the aorta were red patches of fibrin, in flakes, adherent to the serous coat; some few were between this and the middle coat. No distinct aneurismal pouch was apparent; but a thinning and semiprotrusion forwards of the aorta was somewhat perceptible in this position. Upon slitting up the pulmonary artery—carefully avoiding the probe—the serous membrane was found lacerated to the extent of an inch in width, horizontally or transversely just above the free border of the anterior semilunar valve. Upon lifting this free and lacerated edge of the loosened serous membrane, a cavity was found underneath it, capable of holding a pigeon's egg. This cavity was not an ordinary aneurismal sac, but was merely produced by the dissection of the serous from the fibrous coat of the vessel. The superior border of the cavity was convex upwards, and extended to within half an inch of the pulmonary insertion of the ductus arteriosus, which was distinct from and not implicated in the disease. No clots were found in this cavity, nor any fibrinous layers; but, at the superior border, a thin waving line of clot marked the boundary of the dissecting process. The whole loosened serous membrane now appeared more like a loose valvular fold superposed on the aperture of communication between the two large vessels. Previously, however, to the *laceration* of the serous membrane of the pulmonary artery, it must have formed the wall of an aneurismal sac protruding into the cavity of this vessel, and greatly interfering with its duties. The calibre of the vessel must have been more than half obstructed; and the whole of the right side of the heart and veins, both systemic and portal, gorged with blood, even to the cutaneous and mucous capillaries; hence the decided cyanosis preceding dissolution. Soon after this followed the rupture of the serous layer; and then took place the engorgement of the pulmonary capillary system, the spasm, with attendant syncopal asphyxia, and pallor of countenance, so marked upon my entrance; the systemic capillaries being relieved at the expense of the capillaries of the pulmonary circulation.

The valvular apparatus of the aorta, and pulmonary artery, and right ventricle, were perfectly healthy and efficient. The mitral nerve was thickened and indurated; some patches of calcareous matter were deposited at its base, and in the anterior plate: it was perfectly capable of performing its functions. The sinuses of Valsalva were large and aneurismal. The left ventricle was hypertrophied to some extent; the right ventricle was dilated slightly, but this arose just before death, from distention, in consequence of the accident interfering with the course of the systemic venous circulating current. The pulmonary artery was not dilated; its external circumference being precisely the same as that of a healthy adult, namely, three inches. The calibre of the aorta was dilated.

The pleuræ were extensively adherent on the right side, and slightly on the left. The lungs contained some minute hardened bodies in the intralobular spaces, and disseminated between the pleura and vesicular structures. These were numerous, and not unlike minute tubercles of the miliary variety. The trachea and bronchial tubes every where contained frothy saliva. The vascular system of the lungs was universally in a state of distension, but the blood had not a florid hue. It is probable that the length of time elapsing before death was sufficient to blend the whole circulating current, and render it homogeneous: and secondly, the period intervening since the fatal result would be sufficient to remove any arterial tint. The blood was perfectly fluid, and such as we invariably find it in cases of asphyxia. The quantity of circulating medium was above the average, and was certainly at variance with the appearances of syncopal prostration, as observed before death. The coronary arteries had commencing fibrinous deposits within their coats, disseminated at various distances; but no calcareous matter was yet apparent. The brain was perfectly healthy; and the vessels did not present any unusual turgescence, or any morbid appearance.

In order to obtain more exact data of the extent to which the aortic dilatations had proceeded, the following measurements were taken, and compared with those of a normal aorta:—

| | *Case.* | *Normal.* |
|---|---|---|
| Internal circumference of the aorta at the superior border of the aortic valves | 2.85 | 3.00 |
| Internal circumference at the level of the lacerated opening, situated about 1.4 inch above the superior border of aortic valves | 3.50 | 3.00 |
| Internal circumference at the origin of the brachio-cephalic artery | 4.30 | 2.80 |
| Internal circumference after the origin of the left subclavian, and at the remains of ductus arteriosus | 3.15 | 2.40 |
| Length of arch of aorta, from the attachment of the anterior and left semilunal valves to inferior edge of the brachio-cephalic origin | 3.00 | 3.00 |
| Internal circumference of pulmonary artery | 3.00 | 3.00 |

The following CONCLUSIONS may be drawn from an examination of this case:—

I. That aortitis had existed for some time previously to death.

II. That dilatation or true aneurism of the ascending and transverse portions of the arch of the aorta also existed during some considerable period antecedent to the fatal accident.

III. That this dilatation was not sufficiently extensive to produce the *general* symptoms of aneurism of the aorta, the pressure on surrounding parts not having been sufficient.

IV. That it is probable that the stethoscope would have discovered the disease by the physical signs, viz., "a rough, harsh sound, loudest above the clavicles, and beneath the first bone of the sternum"; whilst there would have been little or none over the region of the semilunar valves, or at the apex.

V. That a purring tremor might have been discovered in the same situation, more especially at the root of the neck.

VI. That the rupture of this dilated aorta took place immediately preceding death.

1. From the absence of the peculiar pulse at the first visit, at 9½ P.M.

2. From the total absence of dropsy, and any appearance of cyanosis, or other marked derangement of the circulating apparatus at this period.

3. From the sudden and well marked supervention of these important symptoms.

4. From the ragged and lacerated appearance of the opening, and the absence of coagula and blood stains in the cavity of the false aneurism, and from the non-dilatation of the pulmonary artery, it is evidently one of sudden formation.

I consider this case as one of importance in a physiological as well as in a pathological point of view; and that it proves the following general propositions:—

I. That, in rupture of the aorta into the trunk of the pulmonary artery, death may take place suddenly from syncopal asphyxia; the embarrassment of the circulation and respiration being sufficient to occasion death.

II. That, under these circumstances, no blood escapes from the hydraulic apparatus; the vascular system being as full as ever.

III. That dyspnœa would exist as a prominent symptom, arising from the excessive and irremediable engorgement of the pulmonary capillaries.

IV. That dropsy does not occur under these circumstances, the death being too instantaneous to admit of its occurrence; but, should life be prolonged, the congestion of the systemic and portal capillaries would occasion it.

V. That the size of the orifice of communication would probably determine the occurrence of sudden or protracted death; a small opening, gradually enlarging, being likely to permit of the patient rallying from the first shock, and gradually accommodating himself to the accident.

VI. That, although a communication was established between the aorta and pulmonary artery, yet there existed a great difference between the circulation and that of the fœtus or the new born infant before respiration.

1. The valvular nature of the opening demonstrates that the current must have been from the aorta to the pulmonary artery. In the new born infant and fœtus, it is from the pulmonary artery to the aorta.

2. The cause of the cyanosis in these cases cannot be the same. It probably arises from the fact that the current of venous blood, propelled by the weak right ventricle, is hindered in its course, in consequence of the stronger action of the left ventricle producing a more powerful current of arterial blood, and from the aortic elasticity being more constant in its action, by which means the semilunar valves of the pulmonary artery are *kept in a state of tension;* and the course of the venous current being thus retarded, a state of general congestion and cyanosis results.

REMARKS. The importance of this case of rupture of the aneurismal aorta into the pulmonary artery will appear from the scanty literature of the subject.

Rokitansky and Andral do not give any case of the kind. The Museum of the College of Surgeons of England contains, in its pathological department, two specimens of aneurism of the *ascending* (intrapericardial) portion of the arch of the aorta opening into the pulmonary artery (Nos. 1650 and 1651). In the latter case, it is mentioned that the patient died of jaundice and dropsy; the aneurismal disease not being distinctly indicated during life. Dr. Hope never saw a case; but he gives the details of one which Dr. David Monro of Edinburgh observed, and kindly communicated to him (p. 469). Dr. Copland, in his *Dictionary of Practical Medicine*, vol. i, p. 74, states, that three cases are on record; one related by Dr. Wells, in the *Transactions of Society for the Improvement of Medical and Chirurgical Knowledge*, vol. iii, p. 85; a second by M. Sue (*Journal de Médecine Continentale*, tome xxiv, p. 124); and a third by MM. Payen and Zeink (*Bulletin de Fac. de Méd.*, No. 3, 1819.)

Mr. Thurnam's paper on Aneurisms of the Heart, in the *Medico-Chirurgical Transactions*, vol. xxi, does not contain an instance, although he gives the analysis of seventy-four cases. The *Lancet* contains a case, in which an aneurismal tumour of the aorta threatened to burst into the pulmonary artery. This is the only one contained in it at all similar (vol. ii, 1850, p. 605).

As Dr. Monro's case differs very materially from the one which has fallen under my notice, I will transcribe it in full. It is contained in Dr. Hope's work, 3rd edition, p. 469.

James Evans, aged 24, a porter, admitted into the Edinburgh Infirmary, Oct. 30th, 1833. Accustomed from his profession to lift heavy weights. Had a severe attack of acute rheumatism about ten years ago. About ten months ago, had an attack of pneumonia, which yielded to copious depletion. To this he ascribed his symptoms, viz., palpitation, dyspnœa, followed, three months before admission, by swelling of the abdomen and lower extremities, which has gradually increased.

On admission, the following were the symptoms: great dyspnœa, amounting to orthopnœa; abdomen much distended, and fluctuation; lower extremities swollen and tense; countenance tumid, and somewhat *livid;* great general uneasiness; action of the heart tumultuous, diffused over a large space, not *strong;* cough, with expectoration; pulse large, harsh, and thrilling, 112.

*Physical Signs.* Much dulness on percussion in the præcordial region: first sound accompanied by a loud soufflet, audible over the whole fore part of chest, and on the back on both sides of spine, but most distinct at middle of the sternum: second sound short, and much obscured by the first; hence it appears that a *continuous* murmur extended from the first over the second sound. The treatment employed, viz., digitalis, calomel, and squills, had the effect of reducing the pulse, and increasing the quantity of

urine, but produced no impression on the symptoms. His general uneasiness continued, though temporarily relieved by a small bleeding. The pulse became intermittent some days before his death a fortnight after admission.

*Autopsy.* Much anasarca. The chest contained several pounds of serum in both pleuræ. The heart, enveloped in the pericardium, occupied a great part of the left side, displacing the corresponding lung. It was found to be more than twice the natural size, pale, flabby, and blunt towards the apex. All the cavities were much dilated, together with the corresponding orifices. The walls of both ventricles retained their natural thickness. All the valves were healthy, excepting the semilunar at the mouth of the aorta, which were thickened. The aorta itself, from its origin to the arch, was dilated into a large irregular sac, which adhered firmly to the pulmonary artery, and communicated with it by two openings, situated an inch and a half from the valves; the largest capable of receiving the point of the little finger; the smaller, of transmitting a crow-quill. The edges of both were regular, round, and cartilaginous. Nearer the arch, a third smaller opening was discovered, with thin rugged edges. The internal membrane of the dilated portion of the aorta was reddened and rugous, from numerous cartilaginous patches, which had advanced in some parts to ossification.

In comparing this case with one of rupture into the right ventricle, Dr. Hope makes the following remarks:—1. A lift was the immediate cause of the symptoms, although disease of the aorta had preceded. 2. Pulse pre-eminently jerking; for such was evidently the large, harsh, and thrilling pulse of Dr. Monro's case. 3. A loud superficial murmur, with both sounds incessant in one case, and apparently so in the other, judging from Dr. Monro's description. 4. A livid venous tint of complexion. 5. A great, rapid, and universal dropsy.

And he gives the following, as signs of aneurism of the aorta opening into the pulmonary artery:—

*Physical Signs.* 1. A very loud, superficial, sawing murmur, prolonged continuously over the first and second sounds, probably weakened during the interval of repose, and loudest along the tract of the pulmonary artery. 2. A purring tremor in the pulmonary artery, in the interspace between the second and third ribs. 3. The second sound weakened at the clavicles.

*General Signs.* 1. The jerking pulse. 2. Great, rapid, and universal dropsy. 3. Livid venous tint. 4. The circumstance of the symptoms having followed an effort would afford corroborative evidence.

I am indebted to Mr. J. S. Soden, of Bath, for this report of Dr. Wells's case.

CASE. Mr. A. B., a merchant, of a fair complexion, thin make, and temperate habits of life, in 1789, being then thirty-five years old, became affected with symptoms which were thought to denote the approach of pulmonary consumption. These, however, after some time, entirely disappeared. In 1798, he was attacked with a slight hemiplegia, from which his recovery was soon so nearly complete, that the only inconvenience left by it was an inconsiderable sense of coldness in the foot of the side which had been affected with palsy. In March 1804, he complained to me of being frequently troubled with a noise in his ears, flatulence in his bowels, and pains in his hands and feet, sometimes attended with slight swellings in the same parts. From one or more of these symptoms he was never, I believe, long entirely free during the remainder of his life. He never spoke to me, however, concerning any unusual feelings in the chest; and I have not learned upon inquiry that he ever mentioned his having such feelings to any of his friends or relations, after his recovery in 1789, though his attention was always much directed to the state of his health, and he was not indisposed to make it a subject of conversation.

On the 11th of August, 1807, he fatigued himself considerably with walking. Upon his return from London, on that day, to his house in the country, he ate rather a full dinner, and, having fallen asleep afterwards, he felt so much refreshed in the evening, as to play with his children in the garden. While thus amusing himself, he was suddenly seized, between eight and nine o'clock, with a sense of great oppression in his chest. He soon after became sick at the stomach, and vomited; in the matter thrown up, some streaks of blood were observed. He now went to bed; but, though the weather was warm, and he was covered with bedclothes, his skin felt cold to those who were attending upon him. Mr. Bliss, a medical practitioner at Hampstead, saw him soon after midnight. He then laboured under a constant desire to cough, and was continually expectorating mucus tinged with blood. His body was moistened with a cold sweat; and his pulse was extremely feeble, sometimes it was scarcely perceptible. Mr. Bliss having declared him to be in great danger, a messenger was sent to desire me to see him; but, as I happened to be from home, the messenger applied to Dr. Baillie, who in consequence visited him about five o'clock in the morning. His pulse was then very feeble and irregular; his breathing difficult; his skin pale, cold, and covered with a clammy sweat. Frequently he tossed and writhed his body, as if he was suffering great pain or uneasiness. The faculties of his mind, however, seemed unimpaired. Dr. Baillie concluded, from what he observed himself, and from what he was told by Mr. Bliss and the patient's attendants, either that a large quantity of serous fluid had been suddenly effused into the cellular texture surrounding the air-cells of the lungs, or that some considerable blood vessel had been ruptured in the chest. Mr. B., about a quarter of an hour after Dr. Baillie had seen him, became suddenly worse, and in a few minutes expired. Almost immediately before his death, he complained much of heat in his chest, and threw off the bedclothes.

Two days after death, the body was opened by Mr. Bliss and his partner, Mr. Haynes, when the following appearances were observed.

The blood-vessels of the lungs were very much distended, and there was also a considerable quantity of blood in the air cells. The right lung adhered slightly to the ribs and pericardium; but this seemed to have been the consequence of some disease which had existed long before death. Each cavity of the chest contained about ten ounces of a fluid highly tinged with blood. The pericardium contained about two ounces of a fluid similarly tinged. The ascending aorta was distended to about the size of a large orange. The tumour adhered to the pulmonary artery, just before its division into the right and left branches. Within the circumference of this adhesion, *there was a narrow hole*, by means of which a communication was formed between the two arteries. The cavities of the heart and the great blood vessels were very much distended with blood. The diseased portions of the aorta and pulmonary artery were afterwards seen by Dr. Baillie and myself. Dr. Wells says: "The occurrence of such a disease as I have described might be readily imagined; but I have found no instance of it in the books which I have read; and no previous example of it, I believe, had been seen by any of the surgeons or anatomists of London." Also: "It will be admitted, I suppose, that the communication between the aorta and pulmonary artery took place on the evening before the patient's death, when he first felt an oppression in his chest. In consequence of the superior strength of the left side of the heart, a part of the blood, which was thrown by it into the aorta, must have been forced into the pulmonary artery; from which circumstance an explanation may readily be derived of all the symptoms the patient laboured under, except the sickness and vomiting. These would probably have occurred if any other great disease had attacked him shortly after a full meal, without proving immediately fatal."

I have also been kindly furnished with the following report of M. Sue's case, by Dr. Peacock. It is evidently the same as that of MM. Payen and Zeink. It is entitled, "*Observation d'une Aneurisme de l'Aorta s'ouvrant dans l'Artère pulmonaire, et l'obstruant.* Par M. Sue: receuillie et presentée à la Société par MM. Payen et Zeink."

CASE. Lefrure, a soldier of the Imperial Guard, aged thirty-eight years, was admitted into the military hospital in July 1808. He had been ill eight months, suffering

from palpitation and sense of suffocation, caused by walking quickly, or by ascending stairs. He was compelled to sit constantly upright in bed; face swollen, lips livid, respiration difficult, pulse almost imperceptible; he became anasarcous, and gradually got worse: four days before his death, he became jaundiced, and died exhausted in February 1809.

*Autopsy.* The pericardium contained some fluid. The heart was of its natural size, with traces of inflammation on its surface. The pulmonary artery was greatly dilated, and, on being opened, was found to contain a tumour of the size and shape of a small egg, *and this being cut open*, the finger passed into the aorta. The aorta was dilated at its curvature, and the aneurism proved to have been formed in the aorta, and to have become adherent to the pulmonary artery; and had destroyed its parietes, and closed the orifice. The liver was fatty, but the viscera otherwise healthy.

The case, adds Dr. Peacock, is not very clearly described; and it does not appear that the aneurism had formed any opening into the pulmonary artery.

It follows, from carefully reconsidering these reports, that there are only *three* cases of rupture of an aneurism of the aorta into the pulmonary artery on record: Dr. Wells's case, Dr. Monro's case, and my own. The one related by M. Sue, and quoted by MM. Payen and Zeink, was merely that of an aneurismal tumour pressing on the pulmonary artery, and partially obstructing it, eventually causing dilatation of the vessel. It is so far similar to the one detailed in the *Lancet*.

Dr. Wells's case and my own agree in many respects, more especially in the suddenness of the fatal attack, and the insidious nature of the progress of the disease, merely being marked by slight palpitation, dyspnœa, cough, followed by vomiting, burning heat in the chest or epigastrium, and pain; then by sudden dyspnœa of a greatly aggravated character, prostration, and collapse: cyanosis in my case, pallor in his; asphyxia predominating in mine, syncope in his; coma then following in mine, but the sense remaining unimpaired in Dr. Wells's; death taking place about nine hours after the fatal calamity in his case, but occurring almost instantaneously in mine.

Dr. Monro's case is remarkable for there having been found three perforations, the symptoms having lasted over as many months as in the other cases hours and minutes: the extent to which the circulatory system was embarrassed was, therefore, considerably greater; but, in consequence of the gradual manner in which the chain of events came on, the body became accustomed to the change, and probably some pathological balancing power accomplished. Do the anasarca and ascites thus act? It is certainly very extraordinary that such a serious derangement of the apparatus of circulation should have existed, without death having come on at an earlier period. It is probable that Dr. Monro's case permitted a free circulation of mixed arterial and venous blood, and that the force of the left ventricle was not therefore thrown on the pulmonary capillaries; for the rounded, cartilaginous openings, in Dr. Monro's case, are decidedly an evidence of the communications having been made in a more gradually progressive manner, and from a different pathological cause. Were they the result of ulceration in his case? Mine was decidedly a lacerated opening, and so was that in Dr. Wells's case. The number and size of the communications would allow a perfectly equalized circulation between the two arteries; and probably the livid cyanosis in Dr. Monro's case arose from very nearly the same physiological cause as exists in the fœtus through the ductus arteriosus.

In Dr. Wells's case, the size of the opening and its character are not stated; it is merely said to be a *narrow one;* and probably, from the detail of the case, occurred nine hours before death, and commenced in sudden laceration. Dr. Wells does not state whether any hypertrophy of the left ventricle existed; but *all* the cavities were distended with blood: therefore, I should imagine that it was dilated, and weakened in its power.

Bristol, July 1853.

# PERISCOPIC REVIEW.

## MICROSCOPICAL DISCOVERY.

### THE STRUCTURE OF TISSUES.

*Virchow's Archiv*, v, 270, contains an interesting paper by Dr. F. Hoppe, on certain chemical researches into the anatomical structure of various tissues. These are based upon the fact that animal structures which can be exposed to the action of water boiling under a pressure of three atmospheres for several hours without being dissolved, are not of gelatinous character. In this way a mechanical analysis of the tissue can be made, the gelatinous part is dissolved away, and the cells are demonstrated, by their resistance to the action of the boiling water, to be perfectly distinct from the intercellular matter, to have non-gelatinous walls of their own, and thus to be in all cases more than mere cavities in the surrounding tissue.

Small portions of cartilage, enclosed in glass bulbs, were boiled under high pressure in a Papin's digester. The intercellular substance was dissolved, and the cells could be detected by the microscope, floating free and uninjured in the fluid. Elastic fibro-cartilage, treated in the same way, gave a like result: all was dissolved except the fibres and the cells, which were found free in the solution of chondrin; disproving Mulder's dictum, that "the cells of fibro-cartilage yield chondrin on being boiled."

Bone was next examined; the earthy matter having been removed by a weak acid, small fragments of the animal matter were boiled, as above described. All was dissolved except the cells, which were found, as in the other cases, in the fluid, with portions of the canaliculi still connected with them. Our author concludes that the corpuscles and canaliculi of bone are no mere hollows or canals in the tissues, but have independent walls of their own, containing no gelatinous matter, and distinguished from the surrounding bone substance, exactly as the cells of cartilage are from the seemingly structureless matter in which they are imbedded. Our readers cannot fail to be interested in this experiment, taken in connexion with the facts respecting the structure of bone which we recorded in a former number. Dentine, submitted to the same ordeal, underwent changes precisely analogous to those observed in bone; and Hoppe concludes, respecting tooth substance also, that the tubes and fibrillæ which it contains, possess walls of their own,—of a smooth, thin, membrane, containing no chondrin. Other tissues consist of cells alone, without intervening matter; and, in general, the tissues of the vertebrata may be arranged in two classes: 1. Those which consist of cells and intercellular matter, as areolar tissue, cartilage, fibrocartilage, bone, and tooth; 2. Those which consist of cells alone, as epithelium, muscle, nervous and elastic tissues. This division holds chemically, anatomically, and also physiologically; since the first class compose the supports of organs, the latter the organs themselves.

### NERVES OF THE HEART.

A. Cloetta (*loc. cit.* v. 274) gives the result of his microscopical examination of the nerves of a hypertrophied heart. He has, indeed, done little more than confirm the observations of Dr. R. Lee, on the increased development which these nerves undergo in such cases; a development precisely analogous to that which the same author so successfully demonstrated to take place in the uterus, hypertrophied from natural pregnancy. The matter is well worthy of our notice, chiefly for the practical purpose of observing how closely mere nervous (so-called "functional") disturbance of the heart's action may be connected with the subsequent induction of organic disease.

Cloetta found that the nervous filaments, where they happened to cross arterial twigs, presented small swellings; but he cautiously adds, that the examination he made did not enable him positively to say what proportion of the said enlargement depended upon real increase in the nervous tubules, and what upon hypertrophy of the fibrous neurilemma. Our readers, who are acquainted with the structure of the spleen, will readily recall to mind the error which might be committed in respect to the large nerves of that organ in certain animals, by supposing them to owe their unusual size to nervous matter, while they are in truth indebted for it to the dense neurilemma alone.

### THE RELATION OF THE APPEARANCES IN THE URINE TO DISEASE OF THE KIDNEY.

A paper upon this subject by C. E. L. Mayer (*loc. cit.* v, 199) is well worthy of perusal. He attempts, by careful examination