

T H E S I S

for the Degree of

M. D.

"OBSERVATIONS ON SOME OF THE RARER CARDIAC AFFECTIONS."

by

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Hydatid Disease of the Heart is, I think, of sufficiently rare occurrence to justify me in including it among the rarer forms of Heart Disease. Indeed, from the point of view of Hydatid Disease itself, it is extremely rare to find the heart the seat of the primary lesion. I shall give statistics later as to the relative frequency.

The following case is of considerable interest. It was that of a young girl whom I saw while resident in hospital some years ago. She was 22 years of age, single, a domestic servant.

Previous History:-

She first came under observation in June, and the history was that she had had haematemesis, gastric ulcer being diagnosed. For the immediately preceding six months she had suffered from cough, shortness of breath on exertion, and from frequent febrile attacks, during which the shortness of breath was more marked. On February 23rd. she coughed up a number of 'small white balls/'

balls', which were recognised to be hydatid cysts: from that time onwards up till June she frequently coughed up cysts, some broken, some unbroken.

Family History:-

There was nothing of note in the family history.

State on admission:-

The patient to all outward appearance looked perfectly healthy: if anything, she was slightly anaemic, but was well-nourished. There was no cyanosis, dyspnoea, nor jaundice. The temperature was normal, and she had no cough.

Heart:- The heart's apex-beat was in the 5th. interspace, $\frac{1}{4}$ in. internal to the nipple-line: the area of cardiac dulness on percussion was natural. On auscultation a soft systolic murmur was audible over the apex, but best heard internal to the apex over the **right** ventricle. The pulmonary second sound was louder than the aortic second sound.

Lungs:- There was no deformity of the chest on inspection: expansion was somewhat feeble but equal on/
on/

on the two sides. At the left apex, close to the sternum especially, there was some impairment in the percussion-note, and some small moist crepitations were audible on auscultation. On the right side posteriorly, below the angle of the scapula, and extending into the lower axillary region, there was slight impairment on percussion, and a few small moist sounds were audible on auscultation.

Abdomen:- The liver was apparently normal, and the only abnormality to be made out was a floating right kidney.

Further progress of the case:-

The patient remained well for the first week, and showed no untoward symptoms, but at the end of this time she began coughing up typical-looking hydatid cysts, some broken, some unbroken, varying in size from a pea to a grape. At times there was slight streaking of the sputum accompanying the expectoration of these cysts, but not always: there was always some muco-purulent sputum at the same time, but no foetor could/

could be distinguished. No difference could be observed in the physical signs after expectoration of these cysts.

June 16. The systolic murmur had become more evident, and was heard best down the left edge of the sternum. The physical signs at the right posterior base were also more marked.

June 21. The temperature, which had been normal, rose to 101.4°. The pulse numbered 120, and the respirations 26. Since the previous note there had been more staining of the sputum than usual with expectoration of the cysts.

June 28. The temperature became normal once more, and the pulse and respirations quieter. The symptoms abated, but the physical signs remained the same. The weight had not diminished since her admission. She still coughed up cysts from day to day however, and there was occasional dyspnoea during expectoration of these cysts.

July/

July 13. The patient suddenly experienced sharp pain low down in the mid-axillary region on the left side: the pain was worse on deep inspiration and on coughing. Pleural friction could be heard over this area. Temperature 101.2°. Pulse 128. Respirations 40. The patient looked flushed and ill and there was rather more hoemoptysis. An erythematous rash appeared over the chest, but disappeared the following day. The breathing was rapid and the alae nasi moved with each respiration. The physical signs at the right posterior base were still more marked, bronchial breathing being heard in places, while the physical signs at the left apex were in statu quo.

July 16. Temperature 99.4°. Pulse 100. Respirations 32. The patient had had occasional sickness, but felt better, the pain in the side being considerably less. In addition to the systolic murmur a diastolic murmur was now audible/

audible close to the edge of the sternum as before.

July 20. Temperature again 101°.

Since July 13 the patient had rapidly got worse, and, after consultation with a surgeon, it was deemed advisable to explore the right base in the hope of reaching some large focus, and thus relieving the patient by its removal, if possible. It was recognised to be an unfavourable case for operation on account of the fact that there were presumably separate foci in three different portions of Lung.

Operation:-

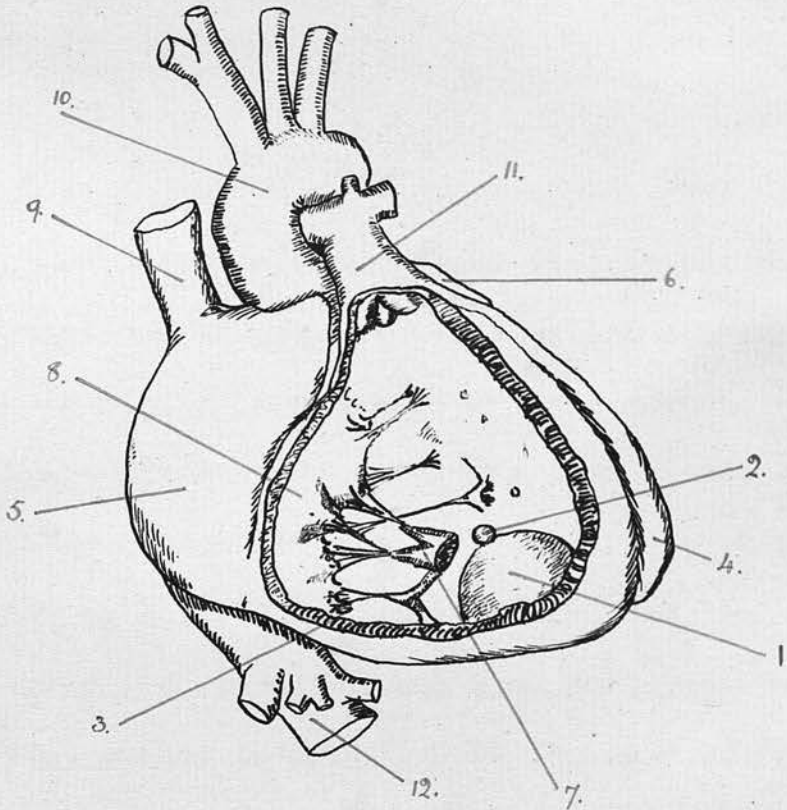
On July 22 the patient was anaesthetised with Chloroform. A portion of the ninth rib on the right side over the dull area was removed: the Pleural cavity was opened: finding nothing in the Pleura, the surgeon explored the Lung, and found a hard resisting mass in the centre of the Lung tissue. He therefore stitched the surface of Lung surrounding this to the Pleura/

Pleura, and deferred further operation until the Pleural cavity should be shut off by adhesions. The patient, however, unfortunately died the following day from heart failure: she had not taken the anaesthetic very well, and never really recovered from the effects of the operation.

Post-Mortem:-

Heart:- There was a cyst, a little smaller than a golf-ball, situated near the apex of the Right Ventricle, and more posteriorly than anteriorly: one-third of it was covered by the heart-muscle and embedded in the heart-wall, the other two-thirds bulging into the cavity of the Right Ventricle. The cyst was ruptured at its most prominent point, and a small secondary cyst was just being shed from this opening into the blood-stream: another tiny cyst was lying loose beneath the posterior flap of the Tricuspid valve. This tiny cyst was about one-third of an inch long, and no thicker than thin string: it had assumed this elongated form no doubt by passing through the small rupture. The valves of the heart were all normal/

Diagram to illustrate Case of Hydatid Disease of Heart



The Right Ventricle has been laid open.

1. Hydatid Cyst - in cavity of Right Ventricle.
2. Daughter-Cyst - escaping from mother-sac.
3. Daughter-Cyst - already escaped.
4. Left Ventricle.
5. Right Auricle.
6. Left Auricle.
7. Musculus Papillaris - cut across.
8. Tricuspid Valve.
9. Superior Vena Cava.
10. Aorta.
11. Pulmonary Artery.
12. Inferior Vena Cava.

normal.

Lungs:- Both Lungs contained innumerable hydatid cysts scattered throughout their entire substance and varying in size from a pea to a large walnut. The largest was situated in the upper lobe of the left lung, corresponding to the position of the physical signs during life. The hardness felt at the right base during the operation was due, partly to Broncho-pneumonia, partly to cysts. At the base of the left lung, corresponding to the position of the pain and friction during life, was found a wedge-shaped recent infarct. On following up the bronchial tubes from the main bronchi, numbers of hydatid cysts were found in them, some of these being quite loose, others more adherent. It is probable that only a small number of the cysts had an arterial origin, but each of these became the source of many others by growing and presently discharging into the bronchial system.

No hydatids could be found in any other part of the body/

body.

Hydatid of the Heart is rarely seen. Dr. Davies Thomas has collected 1900 cases from all countries, and finds the heart affected in 18% of these. Davaine and Cobbold¹ in their combined statistics of 700 cases calculated that 25 affected the heart and pulmonary vessels, i.e. about 3½%. From Schleisner's Table it would appear that hydatid disease is commoner in women than in men. In Graham's book on Hydatid Disease² he notes that Neisser and Finsen drew attention to the preponderance of female subjects affected with the parasite: the former gave his proportion as 210 to 148, the latter as 255 to 181. In Iceland and Germany, where the disease is said to affect females most, their avocations must bring them into closer contact with hydatid ova: especially is this so in Iceland, where the women are occupied in cleaning places where dogs are quartered. In Australia men are most commonly affected, because their habits and pursuits/

¹ Parasites. A Treatise on the Entozoa of Man and Animals. T. Spencer Cobbold. p. 122.

² Hydatid Disease. Graham. p. 72.

pursuits are such as to render them more liable to the disease.

The case above described is interesting in many ways. It seems extraordinary that, with such extensive affection not only of the heart but also of the lungs, the symptoms should have been, comparatively speaking, so slight, and the physical signs too so scanty; yet, as Verco and Sterling say, - "In conformity with the comparatively slow growth of the parasite, the surrounding parts so accommodate themselves to its presence, that, even when of great size, it may occasion surprisingly little inconvenience." The patient looked so well, and the physical signs were altogether so indefinite that, had it not been for the expectoration of the cysts, one could have had no idea that she was suffering from such a serious condition as Hydatid Disease. With regard to the expectoration of these cysts in her case, there was sometimes slight hoemoptysis both prior and subsequent to the expectoration. When rupture of a pulmonary/

¹. Allbutt's System of Medicine. Vol. II.
p. 1124.

pulmonary hydatid cyst occurs into a bronchus, there is usually violent suffocative cough with hoemoptysis and dyspnoea; she sometimes experienced this.

In many cases of hydatid disease, especially of the lungs, there is apparently no deterioration in the general health, and there are many cases on record where 'skins' have been expectorated abundantly, and yet the general condition has remained unaffected. In these cases too, the absence or paucity of physical signs is by no means an unusual thing, notwithstanding the most careful examination of the chest. A small cavity which may be left in the lung after expectoration of a cyst, may be deep-seated, and so contract on its gradually-diminishing contents as to give rise to none of the classic signs of vomica. As a rule, however, the physical signs are said to be more marked after a profuse expectoration.

Another point of interest in this case was the presence of the Erythematous Rash. This so-called Hydatid Rash is sometimes observed to follow the tapping/

tapping of a hydatid cyst, especially those situated in the abdominal cavity. Its association with Pleurisy in this case is interesting, as Finsen and others have described rashes, especially urticarial, as occurring when a hydatid cyst ruptures into a serous cavity. It is apparently not common, and is said to be seen only in a small proportion of cases by those having a large experience in observing and treating hydatid disease. The rash may last any time from a few hours to two or three days. It has occurred prior to, or independent of, any operative interference, even although the patient may never have experienced anything of the kind before. Generally accompanying it are high temperature and considerable abdominal pains. In Graham's book a case is mentioned of Dr. M'Gillivray's, (Sandhurst, Victoria), in which rupture of a cyst occurred into the abdominal cavity: intense urticaria followed, which lasted for three days. It has been ascribed to absorption into the blood of a poisonous ptomaine present in normal hydatid/

¹ Hydatid Disease. Graham. p. 78.

hydatid fluid.

Hydatid of the heart may be found in any of the cavities of the heart, but the usual place is the apex of the right ventricle, either beneath the endocardium, or it may develop within the muscle itself.

The course that the cyst may take varies in different cases. Thus it may remain quiescent for an indefinite time: it may grow to such a size, or be so placed, as to obstruct the circulation: it may rupture into the blood-stream or pericardium: it may die or become calcareous.

Hydatid cysts generally may die at any stage of their existence. Dead hydatids are found most commonly in the liver, both because the liver is the most frequently affected viscus, and because spontaneous evacuation per vias naturales is less easily effected than in viscera such as the lung. Much more ought this latter statement to apply to hydatid of the heart, and a case has been described by Goodhart¹ of a stableman/

¹Path. Soc. Trans. Vol. xxvii. p. 72.

stableman, aet. 20, in whom was found a dead unruptured cyst in the anterior wall of the left ventricle: death in this case was sudden.

Course of Hydatid cyst of the Heart as affecting the patient:-

1. The cyst may give rise to no symptoms whatever, being found accidentally after death, as in Martin Durr's case.¹ Walshe² mentions a case of Carswell's (among the latter's unpublished drawings) in which the heart contained in the posterior wall of the left ventricle a good-sized hydatid cyst protruding on the pericardial surface. The patient, a woman, was cut off by phthisis and the sac accidentally found: as she died in hospital and the heart had not attracted attention, the probability is that there were no cardiac symptoms.

2. It may cause shortness of breath and palpitation after an interval of a year or more, as in a/

¹ Bull. Soc. Anat. de Paris. 1889. lxiv. 131.

² Diseases of the Heart. Dr. Walshe. p. 360.

a case of Peacock's. The patient, a man of 38 years, had lived several years in Australia. He complained of dyspeptic symptoms a week before his death, which however disappeared under treatment. He was suddenly seized with sickness and shivering, and died collapsed the same day. On enquiry post-mortem it was found that he had been short of breath for twelve months. A tumour the size of an orange was found embedded in the posterior wall of the heart, so as to project on each side of the septum into the cavities of both right and left ventricles: on the right side the walls of the cyst were so thin that pus could be made to exude on the slightest pressure, and on cutting into the portion of cyst protruding into the left ventricle, also very thin, numerous hydatid cysts, varying in size from peas to plover's eggs, escaped mixed with pus. The heart generally was enlarged, but there was no valvular disease. All the other organs were healthy. Here the cyst caused no serious symptoms until it became so large, and its coat so thin, as to lead/

lead to inflammation of the serous covering of the heart and effusion of lymph, thus attaching the two pericardial surfaces to each other. In this case rupture must have occurred into one or other ventricle very shortly had life been prolonged.

3. It may cause sudden death without any previous symptoms, as in Charles Kelly's case of a boy, aet. 10, who fell down in the street without warning and died in twenty minutes. At the post-mortem a slightly adherent fibrous sac containing hydatid cysts was found in the appendix of the right auricle: through a rent in this sac a cyst as large as a walnut had escaped, and lay against the tricuspid valve, preventing the passage of blood from the auricle into the ventricle.

Dr. Graham^{2.} records the case of a strong and powerful man who, in the act of breaking stones, dropped down dead. Beyond an occasional tightness in the chest, he had complained of nothing amiss in his health. The wall of the left auricle contained a hydatid cyst with/

^{1.} Path. Soc. Trans. Vol. xx. p. 145.

^{2.} Hydatid Disease. Graham. p. 134.

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². Hydatid Disease. Graham. p. 134.

with numerous daughter-cysts. The left ventricle was hypertrophied: the cyst had ruptured into the pericardium.

Brodribb¹ records the case of a lad, the right ventricle of whose heart was so occupied by a hydatid cyst as to cause fatal interference with the passage of blood into the pulmonary artery. Death occurred after a sudden attack of dyspnoea and cardiac oppression.

Sudden death is usually associated with rupture of the sac into the blood-stream or pericardium, but may occur without rupture, as in Goodhart's case referred to above. On the other hand rupture does not necessarily cause sudden death: if the rent be large, so that large cysts escape, sudden death is likely to happen from obstruction of the pulmonary artery or its main branches, i.e. if the sac be in the right ventricle. For example, Rokitansky related a case (quoted by Walshe²) in which an acephalocyst, nearly as large as a hen's egg, escaped from the mother-sac in the/

¹Lancet. 1837-38. p. 628.

²Diseases of the Heart. Walshe. p. 360.

the upper part of the ventricular septum, and caused instant death by becoming impacted in the conus arteriosus and pulmonary artery, reaching almost to the left branch of this vessel.

Where the sac is situated on the left side of the heart and ruptures into the systemic circulation, death is as a rule sudden. Thus Dr. M'Call Anderson¹ records a case which he says is "worthy of record as an example of an extremely rare, if not unique, form of disease, viz. embolism as the result of a hydatid disease of the heart." Here the patient, a man, was suddenly seized one morning with severe abdominal pain and right-sided hemiplegia, and died a few hours later. There had been no previous symptoms, and, at the time of onset of the attack, no cardiac murmurs were made out on examination of the patient. Attached to the lower part of the septum ventriculorum in the left ventricle was a small clot, on removal of which a small irregular slit was observed: this opened into the cavity of a hydatid cyst in the septum measuring in diameter one and a half inches. Death occurred from blocking/

¹ Contributions to Clinical Medicine.
M'Call Anderson. p. 269.

blocking of the Internal Carotid Artery at the base of the brain by a small cyst.

4. If however the rent be small, so that only small cysts escape, then death occurs gradually with preceding symptoms pointing particularly to embolism of the lungs, and may be to secondary cysts therein, as in the case I have described, i.e. where rupture occurs from a cyst situated on the right side of the heart. Three cases very similar thereto have been reported, and of these I shall give brief notes:-

(a) Barclay¹ describes the case of a man, who for two years had suffered from febrile attacks associated with hoemoptysis and rapid breathing, and who finally died from respiratory and cardiac failure.

P.M. The primary cyst was found in the right ventricle. It had ruptured at least two years before death, because the patient spat up a 'skin' at that time. The lungs contained innumerable cysts, one of which had ruptured into the pleural cavity and set up an empyema, this being the immediate cause of death.

(b)/

¹Glasgow Medical Journal. Vol. i. p. 426.

(b) Budd^{1.} describes a similar case, where the patient died of dropsy.

P.M. The primary cyst in the right ventricle was found to be ruptured, and many cysts were found in the branches of the left pulmonary artery, none in the right.

(c) Ducastel^{2.} describes a case of a man who coughed up hydatid cysts for three years, finally developed heart murmurs, and died of gradual heart failure.

P.M. A hydatid cyst was found in the interventricular septum, pointing into the right ventricle. The lungs contained a large number of cysts.

Where rupture occurs from a cyst situated on the left side of the heart, death, as I said before, is generally sudden. (Vide M'Call Anderson's case.) This may not be so however. For example, Bland Sutton^{3.} notes a case of Oesterlen's^{4.} of a young girl of 23 who developed gangrene of one leg: this was amputated and she died of pyaemia.

P.M./

^{1.} Path. Soc. Trans. Vol. x. p. 80.

^{2.} Bull. de la Soc. Anat. Vol. xv. p. 362.

^{3.} Tumours, Innocent and Malignant. Bland-Sutton. p. 479.

^{4.} Virchow's Archiv. bd. xlii. p. 404.

P.M. A cyst the size of a pigeon's egg, situated in the cardiac wall, had ruptured into the left auricle. Hydatid membrane was found in adherent thrombi in the Common Iliac Artery, and an entire vesicle was found in the Deep Femoral Artery.

5. The cyst may cause death gradually after having given clear evidence of cardiac troubles, such as quickening and irregularity of the pulse, palpitation, murmurs, precordial pain, dyspnoea, swelling of the legs, etc., as in a case communicated by Edward Stanley, F.R.S.,¹ of a woman of 40 who for six months had suffered from shortness of breath and precordial pain. One day she ran upstairs and was suddenly seized with violent precordial pain and dyspnoea: she was sick and collapsed. She took to bed and suffered for six weeks from her heart and died.

P.M. A cyst was found at the apex of the heart bulging into the right ventricle. It had not ruptured.

Diagnosis/

¹ Path. Soc. Trans. Vol. xxvii. p. 72.

Diagnosis:-

There are no symptoms nor physical signs peculiar to Hydatid Disease of the Heart. The diagnosis must therefore always be a matter of great difficulty. It generally results in the death of the patient, the condition only being made out on post-mortem examination; but, where there are physical signs in the lungs in scattered foci, together with organic cardiac murmurs which cannot be accounted for by previous history of rheumatism, chorea, etc., especially in a young person who is coughing up hydatid cysts, the diagnosis of Hydatid Disease of the Heart might justifiably be made. It must necessarily be very rarely that such a chain of circumstances will exist.

Treatment:-

It is scarcely to be expected that medical treatment would be of more avail here than in Hydatid disease generally, where the consensus of opinion appears to be that it is of very little value. No more than passing mention need be made of the drugs which/

which some observers have found beneficial. In 1867 Dr. Hjaltalin¹ in the Edin. Med. Journ. brought before the profession the use of Kamala, with which he thought he had obtained good results in the treatment of hydatid disease. On the other hand Dr. M'Gillivray² found that it had no effect whatever beyond producing intestinal irritation, to which action probably the drug owes its reputation as an anthelmintic. Potassium Iodide and Potassium Bromide have also been used, but with doubtful results. Turpentine has been recommended, no doubt on account of its anthelmintic powers and ready diffusibility.

Operative interference seems to be the only possible remedy for hydatid disease, but, as yet at all events, is impossible where the heart is affected, even should such a condition be diagnosed.

Another case which I had is of interest as showing the difficulty of diagnosis of murmurs in the neighbourhood of/

¹ Edinburgh Medical Journal. August, 1867.

² Australian Medical Journal. July, 1872.

of the pulmonary area.

The patient was a woman, aet. 37, a widow. She was admitted to hospital from the Out-patient department as a case of Acute Pericarditis, diagnosed as such by an experienced physician.

Previous History:-

This was rather indefinite and unsatisfactory. Five or six years previously she had been laid up with pains in the joints, chiefly the fingers, for six weeks. There was no history of Rheumatic Fever other than this. No specific history was obtainable.

For eighteen months previously she had been short of breath, with occasional slight hoemoptysis during this time. The feet and legs sometimes swelled at nights. For some months past she had complained of severe precordial pain.

Family History:-

There was nothing of note in the family history.

State on admission:-

The patient looked feverish and ill, was very tremulous/

tremulous all over, and had a continuous, short, dry, irritable cough. There was extreme shortness of breath and cyanosis, and she complained of more or less continuous pain over the precordium and in the left side. There was slight albuminuria, and a certain amount of oedema of the lower extremities. The temperature was 100.8° . The pulse was not as rapid as might have been expected in a case of Acute Pericarditis, being only 90. It felt like the typical 'water-hammer' variety. The walls of the Radial Artery were slightly thickened. There was no inequality in the pulses. To mention the most important points in the physical examination -

Heart:- The heart's apex-beat was in the 6th interspace in the nipple-line. There was no abnormal pulsation in the chest. On percussion the right border of the heart extended to just outside the right lateral sternal line; there was no increase in the normal dulness at the upper border of the heart. A coarse, double thrill was present, simulating/

simulating pericardial friction fremitus, and a loud, grating, to and fro murmur was audible all over the precordium, even without aid of the stethoscope, quite a foot from the chest. The point of maximum intensity was difficult to make out, but was noted at the time as being in the 4th. left interspace.

Lungs:- There was some impairment of resonance on percussion at both bases posteriorly, with some fine crepitations on auscultation.

Abdomen:- The liver was enlarged, the lower edge extending one inch below the costal margin in the nipple line. No other abnormality was made out on physical examination.

Further progress of the case:-

The pain was relieved by counter-irritation in the shape of ice-bags, and later by leeching. Still the physical signs remained much the same, though, if anything, not so exaggerated as when in the upright position/

position. The murmurs had not the usual direction of propagation of any known murmurs: after about a week, during which she did not improve, a soft systolic mitral murmur developed. The liver increased in size and became pulsating: the cyanosis remained marked. Oedema extended upwards, being especially marked in the lumbar region. The patient became very restless and sleepless, and there was increasing dyspnoea and orthopnoea after the slightest exertion or movement of any kind. The albuminuria increased, and latterly she had several attacks of cardiac syncope, and died of cardiac failure with marked signs of respiratory distress.

Diagnosis:-

The physical signs in this case were so anomalous that it was difficult to understand the morbid condition present: the loud to and fro murmur masked everything else: the murmurs were not propagated in the usual direction of aortic murmurs, nor was their maximum intensity over the aortic area, yet the pulse felt/

felt just like the recognised aortic regurgitation pulse and, there being no signs pointing to Aneurism of the Thoracic Aorta, it was thought that the condition present must be Double Aortic Disease with Mitral Regurgitation, and superadded probably a subacute Pericarditis.

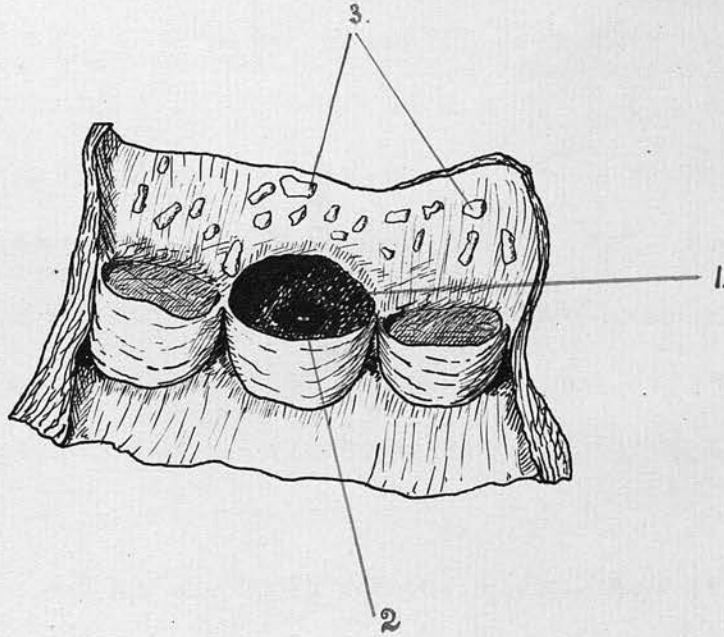
Post-Mortem:-

There were a few small white patches on the pericardium, especially over the right auricle and right ventricle, but no signs of recent pericarditis. There was aortic and mitral incompetence, and extensive atheroma of the whole thoracic aorta. From the anterior sinus of Valsalva, pouching into the pulmonary artery, and adherent to the latter, was a small saccular aortic aneurism of the size of a large hazel-nut, with a small irregular opening of communication into the pulmonary artery.

The presence of Aneurism in this case was interesting from the fact of the patient being a woman.

Dr./

Diagram to illustrate Case of Communicating Aneurism



The Aorta has been laid open.

1. Aneurism of the Sinus of Valsalva.
2. Point of Rupture into the Pulmonary Artery.
3. Patches of Atheroma of the Aorta.

Dr. Browne¹, in his analysis of 173 cases, finds that in only 20 were females affected. Here there was extreme atheroma of the aorta, though the aortic valves themselves, contrary to what is said to be usually the case, looked fairly healthy and free from sclerotic change. According to Gibson, in a large proportion of aortic aneurisms there is some affection of the semilunar cusps: "most frequently such changes are of the nature of sclerosis, obviously having origin in factors analogous to, if not identical with, these which have given rise to the formation of the aneurism."²

Aneurisms at the root of the aorta, involving the sinuses of Valsalva, are often entirely latent, i.e. they may cause no symptoms and give rise to no physical signs: in such cases the aneurism may burst into the pericardium, and suddenly cause the death of a patient, who had previously made no complaints, and who had presented up to the time of death all the appearance of robust health.

Incompetence of the aortic valve is frequently present/

¹ Aneurisms of the Aorta. Browne. London. 1897. p. 4.

² Diseases of the Heart and Aorta. Gibson. p. 835.

present, as was the case here. The incompetence may be the direct result of the aneurism, as, for instance, in those cases in which the base of the aorta is affected and relative incompetence of the aortic valve produced, the aortic segments themselves being healthy: most usually however the two conditions are associated with a common cause, viz. atheroma of the aorta.

Aneurisms of the sinuses rarely attain any size. Bramwell¹ remarks that they kill sooner than aneurisms of any other part. They tend always to descend in the progress of growth, involving in their course the heart or root of the pulmonary artery, as happened in the case described. They are saccular, and by their position are sheltered from influx from the ventricle, while exposed to the maximum force of reflux from the aorta.

Pericarditis is sometimes produced by the irritative pressure of the sac, and, according to Bramwell,² is a highly satisfactory result, seeing that, because of the adhesions thus formed, there is less risk/

¹. Diseases of the Heart. Bramwell. p. 739.

². Ibid. p. 720.

risk of rupture of the aneurism into the pericardium, which is a common termination of aneurisms in this position, where the pericardium is not adherent.

Aneurism of the sinuses causes indefinite symptoms, and owing to its position within the pericardium and its close proximity to the heart, the symptoms are readily confounded with structural or valvular disease of the heart itself: Nixon remarks that " the acoustic signs are no less indefinite from a diagnostic point of view, because, from the close proximity of the aneurism to the orifice of the aorta, a murmur produced by it, whether of influx or efflux, may be easily mistaken for one of the same rhythm caused by obstruction or inadequacy at the aortic valve. The difficulty of diagnosis is further increased by the usual co-existence of atheroma and relative incompetence of the valves and eccentric hypertrophy of the left ventricle." Where there is a communication with one of the chambers of the heart, the physical signs, according to the same writer, are more characteristic, though the symptoms may not be much changed: he notes that/

that a loud murmur, systolic or diastolic, exists, booming or splashing in character, accompanied by a thrill traceable from the root of the aorta along the line of abnormal influx, but not transmitted in the ordinary lines of valvular murmurs: if the two murmurs co-exist, they are fused or converted into a continuous rumble. In the case described the murmurs were loudly heard in the pulmonary area with their point of maximum intensity possibly a little lower down, though, as was said before, this was difficult to distinguish.

The pulmonary area between the second and third ribs to the left of the sternum, has, according to Dr. Balfour, been not inaptly termed the 'region of romance', because of the various interpretations put upon murmurs having their maximum intensity in this position. In his Clinical Lectures on Diseases of the Heart this writer gives notes of interesting cases where loud, rough, systolic pulmonary murmurs were produced over the pulmonary area, where the pulmonary artery/

artery itself was normal, viz. by retraction of the left lung leading to uncovering of the base of the heart, with the production of a systolic pulmonary murmur, this being explained according to Quincke's theory that the systolic murmur is produced by compression of the pulmonary artery by the heart during its systole. I myself have heard similar murmurs in two cases: both of the patients suffered from phthisis, and the left apex had retracted after vomica-formation. "Such cases," Balfour says,² "simulate aneurism of the sinus of Valsalva very closely in the roughness of the systolic murmur, the distinctness of the second sound, and the existence of abnormal pulsation, differing from aneurism first in the entire absence of pain, which, we know, does not always exist even though aneurism be present: and second in the feebleness of the pulsation compared with that of the heart, a sign which, though of undoubted value in the diagnosis of aneurism, is not wholly, nor at any time solely, to be relied upon as of excluding the idea of aneurism/

¹ Edinburgh Medical Journal. Jan. 1871. p. 667.

² Diseases of the Heart. Balfour. p. 211.

aneurism."

^{1.} Bramwell remarks that, in rare and exceptional cases, aneurisms of the sinuses may burst into the pulmonary artery, or into the left auricle of the heart. Stokes^{2.} applies the term 'communicating aneurism' to those aneurisms which perforate, and in which the blood returns into the circulating stream. He states that openings of this sort may occur into the right ventricle, the right auricle, the pulmonary artery, the vena cava, or even the thoracic duct. In these cases there is a combination of symptoms and physical signs, which does not belong to any ordinary case of valvular disease.

The symptoms attendant on rupture into the pulmonary artery, when sudden, appear to be a sensation of something having given way in the cardiac region, with symptoms of nervous shock and subsequent dyspnoea, orthopnoea, cyanosis, occasional nausea and vomiting, syncopal attacks, and increasing oedema of the more distal portions of the body, with engorgement of the lungs/

^{1.} Diseases of the Heart. Bramwell. p. 720.

^{2.} Diseases of the Heart and Aorta. Stokes. p. 552.

lungs and liver, as proved by auscultation and percussion. The pulse is jerking, and feeble in some cases. The physical signs are principally the systolic and diastolic thrill over the second and third left intercostal spaces, and the superficial, harsh, and peculiarly intense sawing and blowing sounds over this area. Where such symptoms and physical signs suddenly arise after some violent effort the diagnosis of rupture of an aneurism into the pulmonary artery might, according to Stokes, safely be made, especially if the patient had also been examined prior to such an accession of symptoms and physical signs; but, where the patient is seen after the rupture has occurred, as was probably the case here, the diagnosis is a matter of considerable difficulty. Again, where this communication is not the result of sudden rupture, but of a more gradual erosive process, "the attendant symptoms may be wanting in the elements of sudden cardiac shock and respiratory distress, which are so striking when abrupt rent of the vessels concerned throws their two channels/

channels into one." In these cases the patient dies with the symptoms of gradually obstructed circulation.

W. Roberts has a case almost identical with the one described: there the communication had been effected, not by sudden rupture, but by a more gradual process: double, loud, rasping, superficial murmurs were heard at an inch from the chest, accompanied by intense tactile thrill. The pulse was markedly jerking and visible in all the superficial arteries.

The fact of an existent diastolic murmur not being conducted downwards to the heart's apex is inferred by F. W. Wade² to be the key to the diagnosis of aortic communication with the pulmonary artery, but Walshe³ argues that in many cases of aortic regurgitation the diastolic murmur is not transmitted downwards to the heart's apex.

In cases of rupture the sphygmographic tracing generally shows a characteristic appearance, viz. great height and perfect verticalness of the upstroke, the downstroke being markedly dicrotous:⁴ unfortunately

a/

¹ British Medical Journal. May 1868.

² Med. Chir. Trans. Vol. xlv.

³ Diseases of the Heart. Walshe. p. 530.

⁴ Ibid.

a tracing was not taken in the case alluded to, as the apparatus was not available.

From the history of the case there was nothing to show that the communication occurred suddenly. In Stokes' Diseases of the Heart and Aorta there is a graphic and full description of an interesting case of communication between the aorta and pulmonary artery: it was one of Prof. Smith's, the patient being a young and muscular man who had always been healthy: there the rupture occurred gradually, and in addition to the usual symptoms, he suffered from frightful dreams and great anxiety: he complained of a fluttering sensation as if, to use his own words, there was a living bird within his chest: the purring murmur could be felt through the bed-clothes, and the sound heard at a great distance from the patient. There was a dilatation of the aorta at its origin, and a small opening of communication with the pulmonary artery at this point, with distinct signs of arteritis/

¹ Diseases of the Heart and Aorta. Stokes. p. 554.

² Dublin Journal. Vol. xviii. p. 164.

arteritis.

It is doubtful how long a patient may survive rupture and communication thereby with another part of the circulation: in Prof. Smith's case the patient survived for three months: in Roberts' case the signs of the affection certainly existed eight weeks before death: but there are three recorded cases where death occurred in four minutes, nine hours, and twelve hours respectively, after the presumed time of rupture. In the cases of gradual rupture, as in the one I have recorded, death occurs from exhaustion through insomnia, restlessness, dyspnoea, pulmonary congestion, and general dropsy.

The third case of which I give notes was one of Congenital Heart Disease.

The patient was a boy, aet. 19; he had no occupation/

occupation.

Previous History:-

The history was that he had always been a 'blue boy' since birth. He had likewise always been short of breath, especially on exertion; he continually complained of the cold, and had been very deaf, though not absolutely so, since infancy. He was subject to recurring attacks of bronchitis.

Family History:-

No other members of the family suffered from the same complaint, nor was there any deformity in any of them.

State on admission:-

In appearance he was cyanosed, at times more deeply so than others: the eyes were prominent, and the conjunctivae slightly injected. He was of an irritable disposition, and mentally not at all bright. He showed marked clubbing of the fingers, toes, and nose, while the nails of the fingers and toes were enlarged and/

and arched. The temperature generally was subnormal. He was poorly enough developed, yet there was no emaciation, as is so frequently present; in this respect Dr. Hunter's amusing and graphic description of his emaciated case of congenital heart disease would scarcely have applied.

The physical examination of the heart and lungs was, put briefly, as follows:-

Heart:- The heart's apex-beat was in the 5th. left interspace, $\frac{1}{2}$ in. internal to the nipple-line. The right border of the heart extended out to $\frac{1}{2}$ in. beyond the right lateral sternal line. There was a coarse systolic thrill and murmur over the pulmonary area: the murmur was audible over a wide area, but with a maximum intensity in the second left intercostal space near the border of the sternum. There was no pulsation in the veins of the neck, nor any such marked physical signs of backward pressure.

Lungs:- The chest was emphysematous and barrel-shaped, with generalised rhonchi on auscultation. There/

There was slight impairment in the percussion-note over both apices, with coarse well-marked moist crepitations on auscultation during inspiration and expiration. Nothing else of importance could be made out on physical examination of the chest.

Examination of the abdomen showed no abnormal physical signs.

The blood showed an increase in the red blood corpuscles to 7,500,000 per cmm.: the white blood corpuscles numbered 11,000 per cmm., and the haemoglobin 105%.

On ophthalmoscopic examination there was great turgidity and sinuosity of the veins of the disc, these being much darker than usual.

The patient ultimately developed a right-sided hemiplegia, and died of exhaustion and cardiac failure two weeks later.

Diagnosis:-

The condition was thought to be one of Congenital Pulmonary/

Pulmonary Stenosis, with probably some deficiency in the ventricular septum.

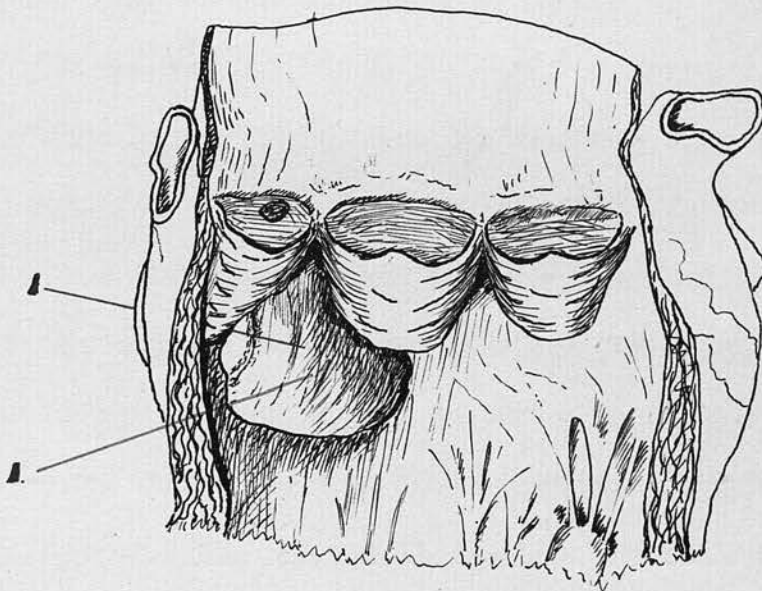
Post-Mortem:-

The heart weighed 18 ounces. The pulmonary orifice was stenosed, though the stenosis was not extreme: there was a deficiency in the inter-ventricular septum situated, as is generally the case, in the so-called 'undefended space', and of about the size of a half-crown piece. The cavity of the right ventricle was large, and its walls markedly hypertrophied: the cavity of the left ventricle was small, and its walls thin and flaccid, relatively to those of the right ventricle.

There were signs of tuberculous affection at the apices of both lungs: the foramen ovale and ductus arteriosus were both closed.

A branch of the Left Middle Cerebral Artery was found blocked by a clot which was undergoing organisation./

Diagram to illustrate Case of Congenital Heart Disease



The Left Ventricle and Aorta have been laid open.

1. The opening in the Inter-Ventricular Septum is here shown.
-

organisation.

Malformation of the Heart is commoner in males than in females. Peacock¹ calculated out of 110 cases, in which the sex of the patient was recorded, that the proportion of males to females was as 61 to 49, or as 55.4% to 44.5% respectively. The explanation of this fact is wanting and remains to be found.

The case described is interesting on account of the age to which the patient had attained. According to Bosanquet², it appears that patency in the foramen ovale is the least serious of the congenital malformations, as small degrees of this deformity are compatible with the attainment of advanced age. He considers that defects in the ventricular septum come next in order of severity, life being occasionally prolonged to the adult period. Cases of pulmonary stenosis, according to the same writer, may also rarely reach adult age, but the majority die in childhood.

Peacock/

¹ Malformation of the Heart. Peacock. p. 165.

² Quain's Dictionary of Medicine. p. 659.
Malformations of the Heart. Bosanquet.

Peacock, on the other hand, thinks that those congenital cases live longest in which there is only a moderate pulmonary contraction with the heart otherwise well-formed: next, he considers, come those with pulmonary contraction and patent foramen ovale, in which the pulmonary contraction is generally greater than in the first class. He analysed 64 cases of pulmonary contraction with imperfect septum (the mean duration of life he found to be practically equal in those cases where, on the one hand, the septum ventriculorum was deficient and the foramen ovale closed, and, on the other, the foramen ovale patent and the septum ventriculorum closed) and the aorta more or less in direct communication with the right ventricle, and, of these, 14 survived the age of fifteen: thus 1 died at fifteen, 2 at sixteen, 1 at seventeen, 1 at eighteen, 1 at twenty, 2 at twenty-one, 1 at twenty-two, 1 at twenty-three, 3 at twenty-five, and 1 at thirty-nine years of age. After these three groups he included the more extreme forms of malformation, into which I need not go.

Another/

Another point of interest in this case was the effect of venesection upon the patient: On one occasion when his breathing was especially bad, and the cyanosis extreme, I performed venesection, extracting 16 ounces of blood, judging from the effect produced on him how much I should withdraw. While the blood was flowing, the patient's colour improved, and he said that he felt much better; he certainly looked better too, and remained so for about a quarter of an hour, after which time he suddenly became cyanosed, almost black in the face in fact, and orthopnoeic. It was only after prompt administration of oxygen and the hypodermic administration of Ether that he was brought round: he had never had so acute an attack of dyspnoea before. Apparently great care must be exercised that more than the required amount of blood is not removed: the actual measurement of the blood in these cases is a matter of considerable difficulty, as, in consistence, ^{the} blood resembles treacle as much as anything else. This was the only case of congenital heart disease on which I had ever performed venesection/

venesection, but I have had excellent results from this means of treatment in chronic valvular disease with failure of compensation. I have notes of several cases which did exceedingly well; of one of these I shall give a brief account in contradistinction to the foregoing:-

The patient was a boy, aet. 15, who was admitted to hospital on May 15th. suffering from extreme shortness of breath, and swelling of the feet and legs. The history was that he had had Chorea two years previously, and that for twelve months he had suffered from cough, shortness of breath, occasional hoemoptysis, and swelling of the legs. On admission there was general anasarca, especially marked in the lower extremities and lumbar region: he was passing scanty urine, and there was considerable albuminuria. He was cyanosed and also orthopnoeic; the superficial veins of the chest and abdomen were distended, and there was pulsation in the veins of the neck. With regard to the physical examination of the chest and abdomen, shortly stated/

stated it was as follows.- The heart's apex-beat was in the 6th. interspace in the anterior axillary line: the right border of the heart was a finger's-breadth to the right of the right lateral sternal line: there was a presystolic thrill over the apex, with presystolic and systolic mitral murmurs, and also a soft tricuspid systolic murmur. There were generalised rhonchi present and the percussion-note was impaired at both bases posteriorly, the breath-sounds being feeble and accompanied by fine crepitations. The liver was enlarged to within two finger's-breadths of the umbilicus, and there was marked ascites. The pulse was rapid and exceedingly irregular. On admission he was bled from the right arm to the amount of 16 ounces, and experienced much relief therefrom. At the same time he was put on Pil. Hyd. Scill. cum Dig. b.i.d. and a diuretic mixture. On May 20th. the abdomen was tapped, and 57 ounces of ascitic fluid was withdrawn. The breathing continued uneasy, and the cough/

cough troublesome, and the signs of effusion, especially at the right pulmonary base, became more marked: this base was accordingly aspirated, and $1\frac{3}{4}$ pints of fluid removed. On the 28th. the ascites was again considerable, and the abdomen was tapped, 31 ounces being removed on this occasion. Subsequent to this the urine increased in quantity to more nearly the normal daily amount, but the breathing and cyanosis again became bad on June 5th. and the right heart showed signs of enlargement as before. He was therefore again bled, 12 ounces being removed: he was greatly relieved thereby, and from this time onwards he rapidly improved, until on June 22nd. the heart's apex-beat was in the 6th. interspace in the nipple-line, the presystolic and systolic mitral murmurs being heard as before: otherwise there were no abnormal physical signs, and he complained of nothing. He refused to remain in hospital as he felt so well, and accordingly walked out on July 6th. looking perfectly fit.

He/

He remained under observation for some months, during which he kept well.

In this case venesection was of the utmost benefit to the patient and seemed, especially after the second operation, to start the rapid improvement which followed. It was instructive also as showing how much more rapidly the patient can be relieved by paracentesis thoracis et abdominis when combined with medical treatment, than by medical treatment alone.

To return to the question of Congenital Heart Disease, just as there may be no general symptoms in organic morbus cordis, even in its most serious forms, so here there may be an entire absence of any clinical phenomena. In illustration of this statement I may cite the case of a Chinese sailor, aet. 21, whom I had occasion to treat for a monkey-bite onboard ship. The wound was clean-cut, being situated on the calf of the leg, and after cleansing with antiseptic lotions I inserted four stitches, and hoped that the wound would/

would heal by first intention. It did not do so, but on the contrary a bad cellulitis of the whole leg supervened, which notwithstanding fomentations did not improve; but foul, thick, greenish-black pus exuded at each dressing. I decided to make free incisions under an anaesthetic, and examined the patient's heart the night before the operation: I was considerably surprised to find, on palpation of the precordial region prior to auscultation, a coarse systolic thrill over the pulmonary area, with a rough, grating, systolic pulmonary murmur on auscultation without aid of the stethoscope. The right heart was considerably enlarged, and the pulmonary second sound was feeble. On examining him more carefully, I noticed his complexion to be slightly cyanotic: the fact of his skin being yellow made the slight cyanosis less noticeable than it might otherwise have been. His fingers and toes (the latter especially) showed distinct clubbing. He said that he had always been short of breath on exertion, but had never thought anything of it.

I/

I thought that, from these various physical signs, I was justified in diagnosing congenital malformation of the heart. The subjective phenomena in his case must have been very slight, because, as a sailor, he was liable to fairly laborious work. I wondered whether this condition could have accounted for the great sluggishness in healing of the wound, which was still discharging when he left the ship some two months thereafter: no doubt the wound had been infected, but the character of the discharge, the extremely unhealthy appearance of the wound, and the length of time in healing were unusual on board ship, where healing is usually so quick in a healthy person.

Cases of this kind therefore can only be made out on physical examination. Openings in the septa, auricular or ventricular, are frequently found on post-mortem examination of patients dying in advanced years. Duroziez¹ has instanced a case of a woman who died of Erysipelas at the age of 76, in whose heart there was a large opening between the two auricles. The/

¹ Traité Clinique des Maladies du Coeur.
Duroziez. Paris. 1891. p. 377.

The diagnosis of the actual condition present must always be largely a matter of speculation. It has been said that where a patient, suffering from congenital malformation of the heart, has survived the age of 15, the likelihood is that the pulmonary artery is contracted. Fagge¹ says,- "The physical signs and symptoms of congenital lesions of the heart are rarely capable of leading to more than a general recognition of their presence, and we must be content to base a further diagnosis upon the age which the patient has attained, and our knowledge of the relative frequency of the several lesions." According to Peacock, "if the evidence of obstruction at the pulmonary orifice be tolerably conclusive, we may safely infer that there is either a deficiency in the septum ventriculorum or a patent foramen ovale, for one or other of these defects almost invariably co-exists with that condition."² Again:- "With an imperfectly divided ventricle the heart may possess its natural form externally, and the defect only be found out on laying open the cavity. Generally, however, and especially if the defect be considerable/

¹ A Text-book of Medicine. Fagge & Pye-Smith.
p. 276.

² Malformation of the Heart. Peacock. p. 194.

considerable, the organ is wider than usual, resembling the quadrangular form of the turtle's heart: this change is more marked when, with the deficiency in the septum ventriculorum, there is some obstruction to the exit of blood from the right ventricle to the pulmonary artery. Here the right ventricle is much hypertrophied: the right auricle also becomes greatly dilated, and its walls much thickened. The left cavities, on the contrary, are relatively smaller and their walls thinner and more flaccid.¹ The hypertrophy of the right ventricle in these cases is due to increased growth, consequent on powerful muscular contractions to overcome the pulmonary obstruction, or to maintain the circulation in the systemic as well as the pulmonary vessels.

The presence of Tuberculosis of the Lungs in the case described was also interesting. Tuberculosis is said to advance rapidly to a fatal issue in many cases of malformation of the heart, but did not do so here. Laennec and Rokitansky asserted that cyanosis and/

¹ Malformation of the Heart. Peacock. p. 38.

and tuberculosis were antagonistic: in the words of the latter,- " Tuberculosis does not exist with congenital vices of formation of the heart or great arterial trunks, which, with their complications, result in venosity and cyanosis," and again, " all cyanoses, or rather all forms of disease of the heart, vessels or lungs, inducing cyanosis of various kinds and degrees, are incompatible with tuberculosis, against which cyanosis affords a complete protection."²

It seems however, that after puberty is attained, and from the ages of 13 to 25, phthisis is the commonest cause of death, a remarkable contrast to its rarity in disease of the orifices of the left side of the heart. In Peacock's 56 cases with different malformations of the heart, which survived the age of eight, 9, or 16.7% died of tubercular affections: this too while about the same period the deaths from tuberculosis amounted to only 9.1% of the total number of the population at large in England and Wales.⁴ Lebert examined/

¹ Path. Anat., Sydenham Soc. Transl. Vol. i. p.316.

² Ibid. Vol. iv. p.251.

³ Malformation of the Heart. Peacock. p.190.

⁴ Registrar-General's Report for 1854.

examined the facts bearing on this question, and found that there was hardly a disease so commonly followed by tuberculosis as pulmonary obstruction.
