cumbency still influences cardiac murmurs. The clue to this further explanation lies, I think, in the relation of the venous hum to posture.

INFLUENCE OF GRAVITY.

Gravity (as Professor Clifford Allbutt has pointed out) seems to cause the increase of the hum in the upright position. Can gravity affect the murmurs in the heart? It seems so, and in order to illustrate the relation of the intracardiac blood currents to gravity, I have prepared Figs. 11 (p. 638) and

Fig. 12.—Diagram of the heart, seen from the left side in the recumbent posture, showing the valvular orifices, etc., as in Fig. 11. *a*, mitral orifice; *b*, tricuspid orifice; *c*, aortic orifice; *d*, pulmonary orifice.

Fig. 12, based chiefly on a careful consideration of the fine drawings in Professor Macalister's *Anatomy*, Professor Braune's *Atlas of Topographical Anatomy*, and Dr. Sibson's article in Reynolds's *System of Medicine*.

Fig. 11 is an attempt to represent the heart (as if transparent) seen with its valves in perspective from the front when the patient is upright. Fig. 12 is similarly an attempt to represent the heart and its valves from the left side when the patient is recumbent. Fig. 10, as just stated, is modified from a frozen section showing the arrangement of the heart cavities in the recumbent position.

From these diagrams it will be seen that :

In the upright position

Gravity should increase the force of flow
from left auricle to left ventricle
" aorta " "
And should decrease the force of flow
from left ventricle to left auricle
" right " " aorta
" left " " pulmonary artery
leaving almost unaffected the nearly horizontal flow between the
right cavities.

Whilst in the recumbent position

Gravity should increase the force of flow
from left ventricle to left auricle
" right " " right (to less degree)
" left " " aorta (slightly)
" right " " pulmonary artery
and should decrease the force of flow
from left auricle to left ventricle
" aorta " " (slightly)
In short recumbency, owing to the action of gravity, should
increase the force of the flow of mitral regurgitation
" " " " tricuspid " (to less degree)
" " " " aortic stenosis
" " " " pulmonary stenosis
and decrease the force of flow of mitral stenosis and
" " " " aortic regurgitation.

In other words, since whatever tends to reduce the force of flow through the valves tends to lessen the murmurs produced at them, and whatever tends to increase the force of flow tends to augment the murmurs, the action of gravity alone appears to explain all the peculiarities observed, except that it does not reduce the murmur of aortic regurgitation.

In the case of aortic regurgitation, however, the force producing the regurgitation is an exceptionally great one—the aortic tension—and in the case of the aortic valves we have the effect of chest depth directly opposed to the effect of gravity.

Taking, then, change of chest depth and gravity together, we should expect to find on recumbency :

Mitral regurgitant murmurs increased,
Mitral stenotic murmurs decreased.
Tricuspid regurgitant murmurs increased.
Aortic stenotic murmurs increased.
Aortic regurgitant murmurs scarcely, if at all, affected.

And these are just the effects which appear to be produced.

Turning next to "haemic" murmurs, it is curious to observe that (assuming the foregoing to be generally true) these behave exactly as if they were produced at the valves to which they correspond. If the apical "haemic" murmur is (as is generally admitted) a real mitral regurgitation, if the pulmonary "haemic" murmur is (as is often denied) a real pulmonary "forward" murmur, and if the aortic "haemic" murmur (which is scarcely discussed) is a real aortic "forward" murmur, we ought to find on recumbency :

The pulmonary murmur greatly increased.
The aortic " " increased.
The mitral " " "

And this is exactly what we do find.

There is an explanation of the pulmonary "haemic" murmur which, so far as I know, has never been put forward, yet may perhaps deserve consideration—namely, that whilst the normal right ventricle, throwing the normal amount of blood into the pulmonary artery, flings wide the valves, and thus encounters no constriction, in anaemia a relaxed right ventricle, propelling a reduced amount of blood, may fail to force quite open the pulmonary valves, and then these valves, though healthy, being held by the pulmonary tension in an intermediate position, may actually, by their position, constitute a constriction and produce a murmur. If this be so, then the explanation of the changes in the murmurs due to change of posture would seem to be complete.

If gravity really exercises the influence which I believe it does, a number of interesting questions arise. To take one instance: we may ask, Does a patient in the last stage of mitral regurgitation assume a forward-leaning position when trying to sleep, in order to bring gravity as much as possible to his assistance, that object being certainly thus attained?

SUMMARY.

1. It seems to me that recumbency tends to increase all "haemic" murmurs except the venous hum, which it tends to obliterate; to increase the murmurs of mitral regurgitation, tricuspid regurgitation, and aortic stenosis; to decrease the murmur of mitral stenosis, and to leave little, if at all, affected the murmur of aortic regurgitation.
2. That the effects of gravity and of change in chest-depth seem to account for the influence of recumbency.
3. And that, therefore, in describing and discussing murmurs which posture modifies the patient's position at the time of observation should be stated.

REFERENCES.

¹ Potain, *Clinique Médicale de la Charité*, quoted by Sansom in Allbutt's *System of Medicine*, vol. v, p. 979. ² Gee, *Auscultation and Percussion*, 1893 edition, p. 149. ³ Allbutt's *System of Medicine*, vol. v, p. 504. ⁴ Allbutt's *System of Medicine*, vol. v, p. 502. ⁵ Braune, *An Atlas of Topographical Anatomy*, translated by Edward Bellamy.

TWO CASES OF CONGENITAL DISEASE OF THE LEFT SIDE OF THE HEART.*

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ALTHOUGH it has frequently been suggested that cases of valvular disease of the left side of the heart may date from fetal life, there has been little *post-mortem* evidence in favour of such a view. Dr. Goodhart once remarked that he had hunted for years for a specimen of mitral stenosis in an infant, both in the *post-mortem* room and in museums, but had not succeeded in finding one. Neither had he met with any medical man who had seen such a specimen.¹ The two following cases illustrate the fact that congenital disease of the left side of the heart may exist, but it does not follow that there is any foundation for the theory that a valvular lesion

* The specimens from these two cases were shown before the Society for the Study of Disease in Children.

such as mitral stenosis when found in a young adult has probably been present since birth. The great mass of *post-mortem* evidence is decidedly opposed to such a view. The interest of these two cases lies, not in the support they may be considered to give to a theory, but in their rarity.

CASE I. *Congenital Mitral Stenosis.*

History.—A male infant, aged 15 months, was brought to the out-patient department of the Bristol Children's Hospital in August, 1901. The child is described in my notes as rather small, anaemic, and presenting some degree of cyanosis. There was no clubbing of the fingers. The mother stated that at the time of birth the child was very blue, and there had been difficulty in "getting warmth into it." Attacks of difficulty of breathing, in which the child was described as going of a "grey colour," had been frequent since birth. The cardiac impulse was in the fifth intercostal space about a quarter of an inch outside the nipple line. A rather high-pitched but not loud systolic murmur was audible all over the cardiac area, but was not conducted far outside the limits of the heart, and was not heard behind. At the base the second sound over the pulmonary area was described as sharp and "clicking" in character, but elsewhere it was rather dull. The only particularly unusual feature noticed was at the apex, where three sounds with something of a *bruit de galop* rhythm were audible. The pulse was 140. On the following week the child was again brought up. It had suffered from repeated attacks of severe dyspnoea, and appeared as if it would not live many days. The child was admitted under the care of Dr. Elliott, and died four days later.

Fig. 1.—Congenital mitral stenosis; the letter M is in the left auricle immediately outside the stenosed mitral orifice; the letter T is in the centre of the tricuspid orifice.

Necropsy.—Only a partial examination was made, the heart being removed by Dr. Forster, but I afterwards went down to the house of the parents, and with their consent removed the aorta beyond the situation of the ductus arteriosus. The heart was much hypertrophied, and weighed 4½ oz. The enlargement was mainly of the right ventricle. The pulmonary artery also was fully double the size of the aorta. On cutting open the auricles the tricuspid orifice was seen to be very large for a child of 15 months, while the mitral orifice was extremely stenosed. The tricuspid orifice measured nearly 2 cm. across, but the mitral only 5 mm. The segments of the pulmonary and aortic valves were normal. Neither the septum ventriculorum nor the foramen ovale was patent. The stenosis of the mitral orifice was of somewhat curious character. As in ordinary cases of mitral stenosis the chordae tendineae were much thickened and shortened, and the flaps of the valve drawn together, but there was also a perforated diaphragm resembling the diaphragm of a microscope, formed by a circular membrane, the base of which was attached to the bases of the mitral flaps. There was thus produced a double stenosis, one at the bases of the mitral flaps and the other at their extremities. The aorta showed a very marked constriction immediately above the point of entry of the ductus arteriosus. It measured there only ½-inch in diameter. The ductus arteriosus was patent but of

small size, and would only admit a glass rod about the size of a lady's hat pin.

Family History.—The existence of mitral stenosis led to more careful inquiries being made into the medical history of the mother. She had not suffered from rheumatism, either during her pregnancy or at any other time. None of the other children, four in number, had been affected with rheumatism. One child had died apparently of diphtheria.

The records of cases of mitral stenosis which I have succeeded in finding have not been numerous. Sansom mentions a case in a child, aged 2 months. The mitral valve was thickened, and presented granulations along the edges of the cusps, and refers also to a somewhat similar case recorded by Ayrolles.² Carmichael has met with a case where in a girl, aged 3 years, extreme mitral stenosis was found. Two orifices not more than ½-inch in diameter were present. The ductus arteriosus was patent in this case.³ Another example is recorded by Cotton, in which granulations were present both on the mitral and upon segments of the aortic valve.⁴

As will be seen from the description of the case recorded above, the stenosis differed in one important detail from ordinary cases of mitral stenosis due to endocarditis. How the perforated diaphragm was produced is not quite clear. So far as I am able to understand the changes which occur in the auriculo-ventricular valve-segments during their developments, I do not see how a diaphragm could arise at the bases of these segments. The processes of endocardium which arise, and later, to some extent, atrophy, are situated at the extremities of what afterwards become the segments of the mitral valve. On the other hand, it is also difficult to understand how endocarditis could have produced the diaphragm, since this membrane did not occupy the usual site of rheumatic vegetations. In the aortic valve rheumatic vegetations may occasionally be seen to form a band which unites the segments, but I hardly think a band produced in this way can have been present here. Although it is not clear how this membrane could have been produced, it seems probable that endocarditis led to some change in the relative positions of the portions of the segments which develop from the ventricular wall and from the endocardium, and it is the endocardial portion which persisted as the diaphragm.

In this case the existence of mitral stenosis was not suspected during life. No presystolic murmur was heard, and the triple sound audible at the apex was not of the rhythm commonly present in mitral stenosis.

It may be mentioned, also, that the patency of the ductus arteriosus was not suspected. I listened carefully in the second and third left intercostal spaces for the undulating but continuous murmur present in some cases of patent ductus arteriosus, but could hear only the systolic murmur that was audible elsewhere over the cardiac area. The narrowness of the lumen of the patent duct probably was the explanation of the absence of this interesting and curious murmur, which, it may be remarked in passing, has resembled, in the two cases in which I have heard it, the mixed sounds of the engine-room of a screw steamer.

CASE II. *Congenital Aortic Stenosis.*

History.—A female child, aged 4½ months, was brought to the Bristol Royal Infirmary in May, 1898, suffering from bronchitis and bronchopneumonia. It was seen by Dr. Watson Williams, and admitted. The child died the following day.

Necropsy.—The body was of average size and fairly well nourished. Broncho-pneumonia and bronchitis were present in both lungs, but the condition of the heart was the chief point of interest. It was much enlarged, the enlargement being chiefly of the left ventricle, which measured 1½ inch from apex to base, and its wall ¾-inch in thickness. The segments of the aortic valve were found to be much thickened and adherent to one another. The right posterior segment was most affected, and was fully 2 mm. in thickness. It was adherent to the segments on either side, which were also irregularly thickened, but to a slighter degree. The thickening in the segments was situated chiefly upon the ventricular aspect. Above they were comparatively smooth. The chordae tendineae of the mitral valve were also slightly thickened, but the orifice was not stenosed. The anterior muscular pillar presented a feature of some interest. It was more than half converted into fibrous tissue, and microscopic sections from other parts of the cardiac wall showed some fibrosis. The other valvular orifices were normal, and the heart presented no other defects. There was nothing noteworthy in any of the other organs.

Family History.—A visit to the mother elicited the interesting fact that about three months before the birth of the child she had suffered from pain and swelling in the left knee, which lasted a few days. She had not been kept in bed, but had been obliged to use a walking stick to aid her in getting about the house. She was about 36 years of age, but had suffered from no previous attacks of rheumatism.

Congenital disease of the aortic valve appears to be more common than similar disease of the mitral orifice. Dr. G.

Carpenter quotes Ranchfuss as having recorded 24 cases of congenital aortic stenosis, the majority of which appear to have been due to endocarditis.⁵ Cases in which two segments of an aortic valve are adherent to one another, and apparently have been so since birth, are not very rare. Whether such a

Fig. 2.—Congenital aortic stenosis; the valve segments are thickened and adherent to one another.

condition is the result of an endocarditis or is a malformation may not be obvious. It is a curious fact that when two aortic segments only are present the valve is far more likely to become affected during adult life with chronic endocarditis than when it is possessed of the normal three segments. Dr. Parkes Weber⁶ argues from this that in some of the cases of disease of the aortic valve occurring in adults, where two cusps are found adherent, the adhesion which is thought to be the consequence of endocarditis has in reality existed from fetal life. This may be so; but I have met with specimens in the *post-mortem* room which have illustrated various stages in the development of such adhesion during adult life. An inflammatory nodule appears at the junction of the attachment of two segments. This swelling generally spreads more or less equally into the adjacent segments, and may unite them together by a mass of considerable size, which, with other nodular thickenings that may have subsequently appeared, sooner or later becomes calcareous. The disease is one of adult life—in one instance only have I seen the nodular swellings under the age of 20—yet it is possible that a similar inflammation may attack the valves of the fetus. This would be especially probable if one could connect the valvular disease with syphilis; but of such an association it seems to me there is little if any evidence. While, however, other causes of fetal endocarditis besides rheumatism may exist, in the case of congenital disease of the aortic valve recorded above, the thickening, although well marked, resembled that commonly seen in rheumatism, and the history given by the mother leaves little doubt that rheumatism was the cause.

In closing I wish to express my thanks to Dr. Elliott and to Dr. Watson Williams for allowing me to use these cases.

NOTE.—Another case of congenital disease of the heart of some interest has recently occurred in the *post-mortem* room of the Bristol Royal Infirmary. In the heart of a female infant, aged 4 months, the rare lesion of a wide communication between the aorta and pulmonary artery, situated immediately above the valves, was present. Besides this, however, very large vegetations were attached to the segments of the aortic and pulmonary valves. The mother had never suffered from rheumatism, but during her pregnancy there had been offensive smells in the house and a few weeks after her confinement, the drains, which had been found to be in a bad state, were repaired by the landlord. The possibility

of the fetal endocarditis having been infective in nature suggests itself.

REFERENCES.

¹ BRITISH MEDICAL JOURNAL, 1891, vol. ii, p. 255. ² Med. Soc. Trans. vol. xiii, p. 147. ³ Edin. Hosp. Repts., vol. ii, p. 298. ⁴ Trans. Amer. Pediac. Soc., 1900, vol. xii, p. 142. ⁵ Congenital Diseases of the Heart, p. 44. ⁶ St. Bart's Hosp. Repts., vol. xxxv, p. 147.

ON THE TREATMENT OF TRAUMATIC ANEURYSM BY PROXIMAL LIGATURE:

WITH SIX SUCCESSFUL CASES.

By CECIL BIRT,

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It is usually laid down in surgical treatises that the correct treatment of traumatic aneurysm is to incise the tumour and apply ligatures to the ends of the artery when found. In the revised tenth edition of Erichsen's *Surgery*, it is stated that the result of departing from this first principle of treatment of diffused traumatic aneurysm in the axilla is well shown by three recorded cases in which the subclavian was tied for subcutaneous rupture of the axillary artery. Two died of gangrene and secondary haemorrhage respectively; the third recovered after amputation at the shoulder-joint.

In gunshot injuries the difficulty of the procedure is great, and can only be sufficiently realized by those who have attempted the operation in the living or made dissections in the dead of the widespread lesions caused by high velocity bullets.

The following cases, which have been admitted to No. 19 Stationary Hospital, Harrismith, Orange River Colony, during the past six months, show that proximal ligature alone has been sufficient to bring about recovery in every instance in which this plan has been adopted.

CASE I.—*Gunshot Wound: Diffused Traumatic Axillary Aneurysm: Ligature of Third Part of Subclavian: Recovery.*—A trooper was struck by a Lee-Metford or Mauser bullet half an inch internally to the angle of the anterior fold of his right axilla. The wound of exit was at the corresponding site behind. The artery was injured, as the radial pulse was lost. The nerves were not severed, since he retained his rifle in his grasp for some minutes. Paralysis of the limb soon came on. On the third day after the injury when he arrived here he had great extravasation of blood in his axilla, arm, and side of his chest; paralysis of the forearm was complete; pain was severe. By the sixth day the swelling had rapidly increased. The hollow of the armpit was replaced by a diffused expansile pulsating tumour. The arm was tense, livid, and ecchymosed in places; his suffering was much aggravated. I therefore tied the third part of the subclavian artery. His symptoms were at once relieved. Twelve days later, when the dressings were removed, the wounds were healed, the tension in his axilla and arm had subsided, and the aneurysm was firmly consolidated. The symptoms of pressure on the nerves were gradually abating when he left.

CASE II.—*Gunshot Wound: Diffused Traumatic Axillary Aneurysm: Ligature of Third Part Subclavian: Recovery.*—A yeoman was wounded at a few yards' range by a rifle bullet which entered midway between the right coracoid process and the angle of the anterior fold of his right axilla and emerged below the spine of his right scapula. He lost much blood. Major R. Caldwell, R.A.M.C., who was near him when he was shot, thought the haemorrhage would prove fatal. He controlled it by compressing the subclavian artery. Forty-eight hours afterwards on his arrival at this hospital the bleeding had ceased. There was a diffuse axillary aneurysm taking an upward direction, causing much swelling beneath the pectorals as well as in the armpit. The right radial artery was pulseless. His upper limb was completely paralyzed and anaesthetic. His pain was great. The third part of the subclavian was therefore tied. In 8 days' time the operation wound was healed and the tumour had become reduced in size and solid. He was regaining feeling and power in his forearm and hand. On his being invalided to England he was making rapid progress towards a complete recovery.

CASE III.—*Gunshot Wound: Diffused Traumatic Brachial Aneurysm: Ligature of Third Part Axillary Artery: Recovery.*—Dr. Allan Campbell, whose case this was, has kindly per-