She desquamated profusely, and made an easy and complete recovery.

About 15 per cent. of cases were stated to have had measles before. Erythema nodosum on the front of the tibia occurred in numerous cases after measles in children of rheumatic tendency.

Treatment.

Copious and frequent application of nearly saturated borax solution to mouth, eyes, and nose. Rest in bed ten days after appearance of rash and confined to house for two weeks unless complications ensued. Jacket linseed poultices I found of great value in the pulmonary complications, followed by a Gamgee jacket in protracted cases. Brandy, in failing pulse and emaciated cases, and often when no other food could be taken, was of the utmost value. Cough mixtures were given as a routine practice containing squills, ipecacuanha, belladonna, potassium and ammonium carbonate, and liquorice. Tinct. nucis vomicae was added when necessary. I give full notes of the following case, as one of great interest, showing the ten days' struggle from the onset of the rash of a healthy child:

Girl, aged 18 months. Called to her on June 24th. She had the usual history and had had slight diarrhoea for two days. The rash was well out when I saw her. On June 28th, fifth day of rash, the temperature was 101° F., respirations 30, and snuffling in type; pulse 130. Diarrhoea continued in spite of treatment. Poulticing of chest was tried. On June 30th the breathing got worse and she began to be restless. The chest sounds were loud and harsh on inspiration with bronchitic rhonchi. No dullness. On July 2nd a few streaks of blood appeared in motions, and although diarrhoea was much improved the child became semicomatose with great restlessness. Cough was not very severe. Temperature never high, 101° to 102° F. On July 3rd diarrhoea was under control, but in the evening, while at stool, on giving a cough, the mother noticed a swelling in the face and sought me late at night. I found the child collapsed, dusky, and breathing distressfully. The left cheek appeared swollen like a large gum-boil. The right cheek was slightly puffy. A further examination revealed a goitrous-like neck, which extended round to the back on both sides and tapered off to the spines of the scapulae. On feeling these swellings their true nature was at once apparent by the crackling feel under the fingers—surgical emphysema. Chest back and front were hyper-resonant. I did not think the child would survive the night. Next day the emphysema had blown out both cheeks to the zygoma and tapered off quickly into the scalp. It passed round to the inion and back of neck. The child died July 3rd at 11.30 a.m.

Necropsy, July 4th, 1911.—Well nourished body; rigidity passed off; buttocks irritated by discharges; surgical emphysema, the knife passing through subcutaneous tissues with crackling sound, like cutting raw blown veal. Some air had penetrated beneath the pectoral muscles. On removing the sternum the external surface of the pericardium was seen to be studded with air vesicles, like bunches of very small grapes. The lungs also, but more discrete. In the lungs the visceral pleura was blown up, forming the blebs. No fluid in pleural sacs. In one place the lung tissue for about $\frac{1}{2}$ in. was as if cut in two and connected by two thin transparent membranes. The anterior aspect of the upper lobes in both lungs—from anterior margin to half the extent of the lung surface—was most affected, and the lower pole. The apices and posterior border were little affected. The "dog's tongue" was blown out into a bladder. The middle lobe of right lung was similarly affected to the upper lobes. The lower lobes of both lungs contained scarcely any blebs, and presented a nearly normal appearance, except for a bleb or two. (The air pressure, therefore, was least in apices, posterior border, and lower lobe.) The roots of the lungs were a mass of blebs. One root-bleb in the left chest had burst. (Pneumothorax was not diagnosed during life; both lungs were equally hyper-resonant.) From the roots of the lungs the blebs were continuously present into the pericardial surface (external) and mediastinal tissues in all directions, passing upwards into neck and face, and down along the aorta through diaphragm, getting more scarce, but a few blebs reached around the kidneys, and even behind the rectum. The pericardial sac contained 3iv of yellowish albuminous fluid. The interior of sac and heart surface presented a boiled appearance, otherwise normal. Mitral valves: Opalescent film patches on valve; free border thickened and red, with smooth lentil-sized nodules (2)—swelling, not deposit. Aortic valve normal. Tricuspid and pulmonary valves similar to mitral, but no nodules. A firm white clot adherent to the auricular appendix passed into and filled the left ventricle, adhering to the papillae, and passing $1\frac{1}{2}$ in. into the aorta. Right heart same condition, reaching from auricle to beyond the bifurcation of the pulmonary artery. Section of lung showed no solid raised patches, large or small. Plugs of muco-purulent matter could be expressed from the bronchioles. The lungs were spongy, and frothy bloody fluid exuded. Trachea contained a moderate layer of reddish muco-purulent matter. Larynx similar, with some caked on drab coloured parts. Epiglottis swollen, and also vocal mechanism (vocal cords unrecognizable). Spleen dark and firm. Liver, boiled appearance. Kidneys also, and they showed fetal lobulation. Blood dark and fluid. Stomach showed hyperaemic patches of mucosa at entrance and exit,

and contained yellowish fluid. A foot along the jejunum a small patch of hyperaemia, and occasionally a patch, until one reached the last 4 in. of the ileum, where there was marked hyperaemia and a little ecchymosis. The caecum and ascending colon were similarly severely involved. Patches of the same nature, but slighter, occurred in the rest of the intestine. No peritonitis, no fluid in abdomen. No enlarged glands. Appendix normal. Very little air had penetrated along the mesenteric vessels. Brain and membranes showed venous congestion only. The blood taken *post mortem* showed an intense leucocytosis, mainly polymorphs; numerous pneumococci were free in the blood and in the leucocytes. A smear from the laryngeal wall showed plentiful supply of *Micrococci tetrageni* and pneumococci. Also long streptothrix chains (unbranched), eight to ten times as long as the pneumococci chains; each member of the chain a bacillus with beaded appearance, and as long as six pneumococci, and Gram-staining. Tracheal smear showed the same fauna. The smear from the exudate from the bronchiole showed pneumococci only, as also did the pericardial fluid.

ANEURYSM OF THE SUPERIOR MESENTERIC ARTERY, WITH RUPTURE.

By ALEX. H. GIFFORD, M.R.C.S.ENG., L.R.C.P.LOND.

SENIOR MEDICAL OFFICER, ADDINGTON HOSPITAL, DURBAN.

ANEURYSMS of the abdominal aorta, near the coeliac axis, are described as occurring in nearly equal proportions communicating with the anterior and posterior parts of the aorta, but I can find no description of an aneurysm of the superior mesenteric artery. The following is a case of aneurysm of the superior mesenteric artery associated with an aortic aneurysm, with rupture into the lesser sac of the peritoneum.

History.

A Kaffir woman, aged about 33 years, came into hospital complaining of pain and throbbing in the epigastric region, where she had "noticed" a lump for a week. The pain in the abdomen had been felt for three months; it had no relation to the taking of food, but her appetite had been very bad; the bowels had been constive for the same period. There was no history of injury in this region, and no history of syphilis.

Physical Signs and Symptoms.

A tumour the size of an orange was seen and felt in the epigastric region, slightly to the left of the middle line; it was firm, and incompressible on moderate pressure, with a somewhat irregular conformation; there was a strong expansile impulse synchronous with the heart's beats. The tumour was immovable, both from above downwards and laterally. On light percussion there was no resonance. The mass was firmly pushed against the anterior abdominal wall.

Two days after admission the epigastric pain was diminished, but two days later there was a return of this symptom, with a constant severe pain, which was referred to the spine in the lumbar region, and to the sides of the abdomen about the same level. The patient died eight days after admission.

Necropsy.

At the *post-mortem* examination a fusiform aneurysm of the abdominal aorta was found at and around the origin of the coeliac axis and superior mesenteric arteries.

The opening of the coeliac axis artery lay in the right side of the aortic aneurysmal sac, and was normal in size and shape; below this and involving the origin of the superior mesenteric artery was a large orifice, 2 in. in length and $1\frac{1}{2}$ in. in width, opening into a larger, irregular, oblong-shaped aneurysmal cavity, the measurements of which were 4 in. from right to left, 3 in. from above downwards, with a depth of 2 in. from before backwards. In addition there was a conical prolongation downwards and to the right, which was continuous with the antero-duodenal portion of the superior mesenteric artery. Another prolongation of the cavity extended upwards below, then to the right of and finally above the coeliac axis artery and its branches. This prolongation extended up as far as the inferior surface of the liver, and it was at its upper part that the rupture occurred into the lesser sac of the peritoneum.

This large aneurysmal cavity was bounded anteriorly by the head, neck, and body of the pancreas, and by the posterior layer of the lesser sac of the peritoneum; posteriorly the cavity was formed by muscles and various tissues matted together, superiorly the liver, and inferiorly the transverse part of the duodenum and the mesentery bounded the cavity, whilst the right side was formed by the junction of the anterior and posterior walls.

The rupture took place into the lesser sac by a transverse opening $\frac{1}{2}$ in. in length at the upper part of the cavity, just under the liver, the edges being much thickened and somewhat valvular.

The blood, after rupturing into the lesser sac of the peritoneum, had found its way through the foramen of Winslow into the peritoneal cavity, where about two handfuls of clot and a pint of serum were found at the *post-mortem* examination.

The interesting points of this case were the facts:

1. That the aneurysm must have been one occurring in the superior mesenteric artery in its course behind the pancreas, remnants of the arterial wall being found lying flat on the posterior wall of the cavity.
2. That the gradual dilatation of the large aneurysmal cavity had exercised a traction on the aorta itself, so that a fusiform aneurysm was formed.
3. That there was a rotation of the aorta, in that the opening of the coeliac axis artery and what must have been the opening of the superior mesenteric artery originally, were found on the right side of the aortic aneurysm.
4. That there must have been a gradual and persistent dissection in the upward direction to the lower surface of the liver where the rupture eventually took place, as is evidenced by the thickening and induration of the walls.

For permission to publish this case I am indebted to the Medical Superintendent of Addington Hospital.

ELECTRO-CARDIOGRAPHY AND ITS IMPORTANCE IN THE CLINICAL EXAMINATION OF HEART AFFECTIONS.

By THOMAS LEWIS, M.D., M.R.C.P.,*

LECTURER IN CARDIAC PATHOLOGY, UNIVERSITY COLLEGE HOSPITAL MEDICAL SCHOOL; PHYSICIAN TO OUT-PATIENTS, CITY OF LONDON HOSPITAL.

(Continued from page 1423.)

PART II.

The Auricular Summit in Mitral Stenosis.

THE summit *P*, which corresponds to the contraction of the auricles, is small and pointed or rounded in most normal electro-cardiograms from lead *II*. As a rule its height does not extend for more than one scale division. In mitral stenosis, where there is hypertrophy of the auricular musculature, this summit is considerably modified and is of diagnostic importance. It is generally broad, flat-topped, and bifurcates in its centre (the last-named feature is not of necessity pathological), its height is greatly increased, it often extends to two scale divisions (Fig. 6; see also Fig. 4 *II* of previous article) and may amount to three or even more millimetres.

Fig. 6.—An electro-cardiogram (lead *II*) from a case of mitral stenosis. It shows the exaggerated *P* summit, which is broad, flat and divided. This picture is common in mitral stenosis.

The sign is of chief clinical value in cases of mitral stenosis in which murmurs are absent or obscure, and in such cases galvanometric examination may decide an otherwise doubtful diagnosis.

"As-Vs" Interval.

The normal heart beat consists of a sequence of chamber contractions. The contraction begins in the auricle and having passed through it is transmitted to the ventricle through the specialized tract of tissue known as the auriculo-ventricular bundle; the ventricle having received both its charge of blood and impulse from the auricle, contracts. There is an appreciable delay between the contractions of auricle and ventricle in a normally acting heart, and the length of interval between the onsets of the contractions is taken as a measure of the functional efficiency of the bundle in question. This interval is readily ascertained in human electro-cardiograms. It is represented by the distance from the commencement of the auricular summit *P* to the commencement of the ventricular summit *R*. In normal electro-cardiograms this *P-R* interval measures between 0.12 and 0.18 second.

But in pathological hearts the interval is often greatly increased; it often reaches 0.3 second (Fig. 7) and may extend on occasion to as much as 0.4 second. Prolongation of this interval is the first of a series of extremely

interesting and important phenomena which are spoken of collectively as "heart-block," phenomena which are due to impairment of the tissue functions in the tract which

Fig. 7.—An electro-cardiogram from a case of subacute infection of the bladder. The *P-R* interval is prolonged; there is hesitancy in the transmission of the impulse from auricle to ventricle, and consequently evidence of damage of the tissues joining the auricle and ventricle. The disturbance in conduction was the sole sign of myocardial disturbance in this patient at the time when the curve was taken.

conveys the contraction impulse to the ventricle. The impairment of bundle function produces delay or hesitancy of the impulse transmission, which can be discovered with certainty only in graphic records taken from the heart. It leads to no disturbance of the beats which is evident during ordinary clinical examination. Yet we cannot afford to neglect it. It is a frequent sign in heart disease; it may be a forerunner of grave disturbances of the heart's mechanism, which will be spoken of later; it is in itself always serious. The length of the *P-R* interval is one of the important prognostic signs in mitral stenosis, for such hearts when regularly exhibiting prolongation of the interval are hard hit, and the damage is not necessarily confined to the auriculo-ventricular bundle, but is usually disseminated throughout the heart muscle. A prolongation of the *P-R* interval is not at all an infrequent sign during the course of acute infections, especially those of rheumatic type. It may be that a patient who exhibits some slight enlargement of the heart, or in whom a systolic murmur of rheumatic origin is present, develops a sore throat or slight febrile attack or soreness of the joints. At the same time prolongation of the interval may be noticed. It may even occur apart from other manifestations of infection. Whenever it develops it is a sign of considerable consequence, for it indicates involvement of the heart muscle. Clinical medicine has recognized lesions of the heart valves for many years; it has looked almost in vain for signs of early muscle damage. Advanced rheumatic disease of the heart has essentially a long history behind it; it probably results from repeated infection and repeated slight damage of the valves and muscle. Transitory prolongation of the interval between auricular and ventricular contractions is one of the very few signs which we possess of these invasions. The greater part of the ventricular musculature may be spoken of as silent, in the same sense that large areas of the cerebral substance are termed silent. The auriculo-ventricular bundle, like the pyramidal tracts of the spinal cord, gives, when damaged, early indications of such damage, though the lesion may not be, and usually is not, confined to it. The electro-cardiogram thus serves an extremely useful purpose, for it not only gives us an accurate index of the manner in which the functions of the auriculo-ventricular bundle are fulfilled, but it also throws light upon the condition of the myocardium as a whole. A single manifestation of bundle deficiency is spoken of at the present time: others will be noted at a later stage.

Lesions of the Bundle Branches.

The auriculo-ventricular bundle divides into two large branches near the ventricular septum and these run and arborize under the endocardium before joining the muscle of the ventricle. Just as lesions of the main stem of the bundle give rise to electro-cardiographic signs, so do lesions of its branches; and a number of such lesions may be recognized to-day, and their significance may be appreciated. A lesion of a bundle branch results, not in a prolongation of the conduction interval, but in profound modification of the manner in which the auricular impulse is distributed to the ventricle through the conducting system as a whole; the result is a profound modification of the type of electric curve which represents ventricular contraction, for the type of electric curve is chiefly dependent upon the point or points at which contraction starts in the muscle from which it is recorded.

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