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CLINICAL NOTE ON A CASE OF COMPLETE DEXTROCARDIA.

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There is at present under the care of the writer, a soldier's son, aged 12 years, whose heart lies completely in the right side of his chest. On inspecting the chest an impulse is seen in the fourth right intercostal space and anterior axillary line, that is, outside the right nipple line; a second area of impulse, less marked but more diffused, is noted about the third right intercostal space and right para-sternal line. Palpation communicates a somewhat heaving impulse at the first named spot, synchronous with the radial pulse and first heart sound; in the second area mentioned the impulse is scarcely appreciable. Percussion gives a roughly quadrilateral area of deep dulness limited towards the left by the sternum from the third to the fifth chondro-sternal junctions; the lower limit lies along the fifth right rib; the outer limit is a vertical line joining the fourth and fifth ribs two fingers' breadth beyond the right nipple line; the upper limit is less well defined, but reaches a curved line drawn from the summit of the outer limit to the third right chondro-sternal junction; the area of superficial dulness is less by about two fingers' breadth. The normal heart sounds are heard over the dull area, most loudly and clearly at the first-named point of impulse; there are no adventitious sounds. The muscles of the right shoulder are wasted, the upper part of the chest in front is flattened, the intercostal spaces are narrow, and expansion is less than on the left side. The percussion note is impaired over the back and front of the upper part of the chest, and auscultation reveals bronchial breathing, bronchophony and crepitant râles over the upper lobe of the lung; the altered breath sound and râles are especially marked in the supra-spinous fossa and in the apex of the axilla. The left side of the chest is larger than the right side, the upper part is more rounded and prominent, the shoulder is higher, the intercostal spaces are wider, expansion is greater, and the percussion note gives greater resonance than on the right side; there is no area of dulness, and the expiratory murmur is audible and prolonged. The patient suffers from cough, hæmoptysis, dyspnoea, sleep sweats and other signs of phthisis, from which he is undoubtedly suffering; it was on account of an attack of hæmoptysis that he first sought advice; neither he nor his parents were aware of the dislocation of the heart, nor does he suffer anything which could be directly attributed to it. The upper
lobe of the right lung and probably the apex of the middle lobe are acutely diseased; the left lung is in a condition of complementary emphysema, and the altered position of the heart is almost certainly consequent on the pulmonary disease.

Abdominal examination, with a view to ascertaining the position of the liver, is not very satisfactory; neither decided liver dulness nor gastric tympany can be obtained in either the right or left hypochondrium; there is undoubtedly a greater sense of resistance on the left side, and the percussion note has less tone; this may possibly be due to unusual descent of the diaphragm. Low down, in the right posterior axillary line, there is a small area of impaired resonance, probably hepatic; it is curious that in a case where it would be expected to find an enlarged liver a difficulty is found in locating that organ satisfactorily by ordinary clinical methods.

Dextrocardia is either congenital or acquired. The former gives rise to no symptoms, and is usually discovered accidentally; it is generally associated with transposition of other viscera, hence the importance of ascertaining the position of the liver; if the liver is found transposed in a case of dextrocardia it is fair to assume that the position of the heart is congenital.

Congenital dextrocardia is not, however, invariably associated with transposition of other viscera, so that it is not safe to conclude that because the liver is in its normal position the dextrocardia is necessarily pathological. In this particular instance, the question is not of great importance, either in connection with diagnosis or prognosis, owing to the very serious pulmonary mischief. But the interest lies in the fact that Babcock appears to regard the congenital form as predisposing to pulmonary tuberculosis; he quotes, in his work on "Diseases of the Heart and Arterial System," the case of a lady who developed phthisis two years after the diagnosis of transposition of the viscera had been made. Acquired dextrocardia is complete or partial; the case under consideration is unusually complete. In "Auscultation and Percussion," Gee says, "And whatever may be the case in dislocations of the heart rapidly produced, there is no doubt that chronic disease can displace the heart so that its very apex beat shall be felt in the right nipple line." Further on he adds that in displaced hearts the impulse seen to the right of the natural position is often some part of the ventricle other than the apex. There is a considerable difference of opinion with regard to the anatomy of the condition. According to some the heart being fixed at its base is, in process of transposition, rotated on its long axis, so that more either of the right or left ventricle than is natural comes to the front; simultaneously, the apex is said to be swung across the front of the chest to a variable extent, the heart even assuming a horizontal position in extreme cases. Others maintain that the heart is neither rotated nor the apex swung over, but that the heart is moved bodily over with the mediastinum,
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retaining approximately its normal relation to the diaphragm, and that
the impulse found to the right is that of the right auricle, the apex lying
behind the sternum. Clinical and post-mortem evidence is adduced in
support of both contentions; there is, unfortunately, nothing in the present
case to indicate the anatomical condition with certainty. There is general
agreement as to the causes of displacement to the right in acquired cases.
The causes given include increased pressure in the left pleural cavity,
owing to the presence of fluid or air, and tumours to the left of the heart;
phthisis, cirrhosis, and old pleurisy of the right lung can also cause the
displacement, owing in part to contraction of pleuro-pericardial adhesions.
With removal of the cause of increased intrapleural pressure, the heart
may return to its normal position, but traction by pleuro-pericardial adhe-
sions causes permanent and the most extreme degrees of displacement.

NOTES ON A CASE OF ACUTE YELLOW ATROPHY OF THE
LIVER, WITH REMARKS ON THE ETIOLOGY AND TREAT-
MENT OF THE DISEASE.

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Patient.—Driver A. R., R.F.A. Was admitted on February 9th, 1903,
apparently suffering from a mild attack of catarrhal jaundice. On Feb-
uary 18th, 1903, he developed mental symptoms, and was transferred to
a special ward. He came under my charge on February 25th, 1903. He
was slightly jaundiced, his skin being lemon-colour and conjunctivre
faintly yellow. Urine slightly bilious; it did not give a very typical
"play of colours" with Gmelin's test. No albuminuria. A few gono-
cocci.

Alimentary System.—Tongue slightly furred, yellowish-white; teeth
good; no aural sepsis. Liver and spleen tender, but not enlarged. Stools
white and scybalous.

Circulatory and Respiratory Systems.—Normal.

Nervous System.—He looks dull, half asleep, in fact, in a state of
mental torpor, but he can be roused into sufficient intelligence to answer
questions relating to his case. Pupils regular, react both to light and
accommodation; no nystagmus; superficial and deep reflexes normal.
Organic reflexes, incontinence of urine and faeces at times.

Blood Examination.—Showed a polymnuclear leucocytosis. No malarial
parasites.

Temperature.—102° F. on admission, then normal until three days
before death, when it rose to 103° F.

Pulse.—Slow, 56, regular, low tension; the same on both sides; artery
not thickened.