A CASE OF BERI-BERI HEART DISEASE

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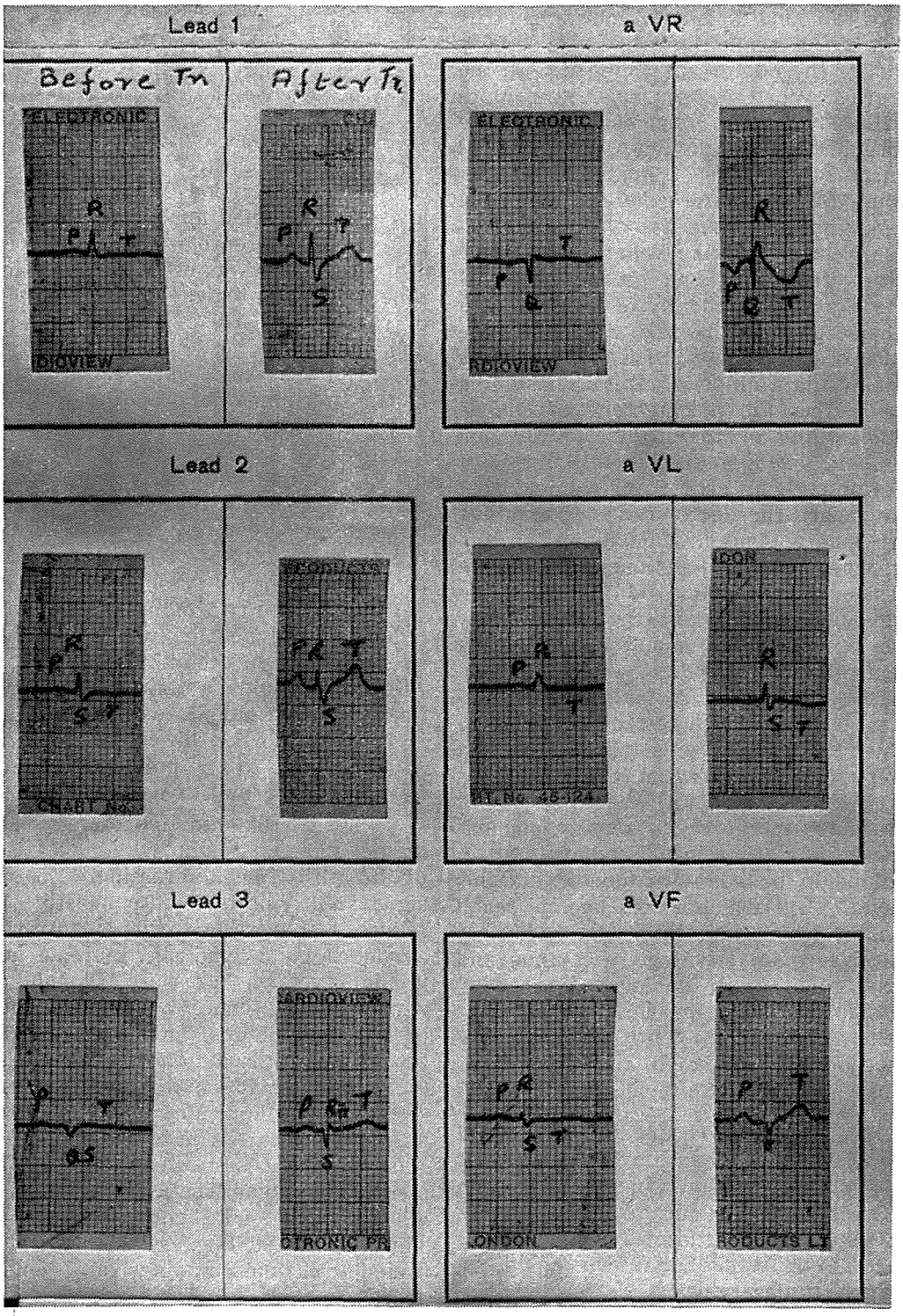
A thirty year old male was referred for progressive heart failure of unknown etiology. His personal history was as follows: At 15 he had joined the Royal Navy, playing Rugby football and taking part in athletics. There was not, therefore, at that time anything to suggest either congenital or rheumatic cardiac lesions. Nine years ago, he passed his "medical", and joined the Fleet Air Arm. Three years later he began to complain of palpitations which were attributed to emotional problems, because exhaustive investigations in a naval hospital had failed to show any abnormality. Since one year he has found himself progressively getting out of breath on exertion — a symptom which he attributed to heavy smoking (80 cigarettes per day). Since two months his exertional dyspnoea had become worse and he noticed swelling of the ankles. No history of chest pain could be obtained. After close questioning he admitted that for the last eight years he had been a heavy drinker of whisky (up to one bottle a day) and of beer (up to 20 bottles daily).

Physical examination showed a thin man of medium height, who got easily out of breath while undressing. The usual signs and symptoms of congestive heart failure were present, that is, exertional dyspnoea, mild cyanosis, anorexia, raised jugular venous pressure, basal rales, hepatomegaly. There was pitting oedema of the lower limbs. Palpation revealed a biventricular thrust. There was a regular tachycardia of 130/m and grade 2 systolic (ejection type) murmurs over the apex and base of the heart. The blood pressure was 140/0. Urinalysis showed a trace of albumen but no sugar. The E.C.G. showed low-voltage of the QRS complex and flat or slightly inverted T-waves. The X-rays confirmed the clinical findings of an enlarged heart.

We were dealing, therefore, with a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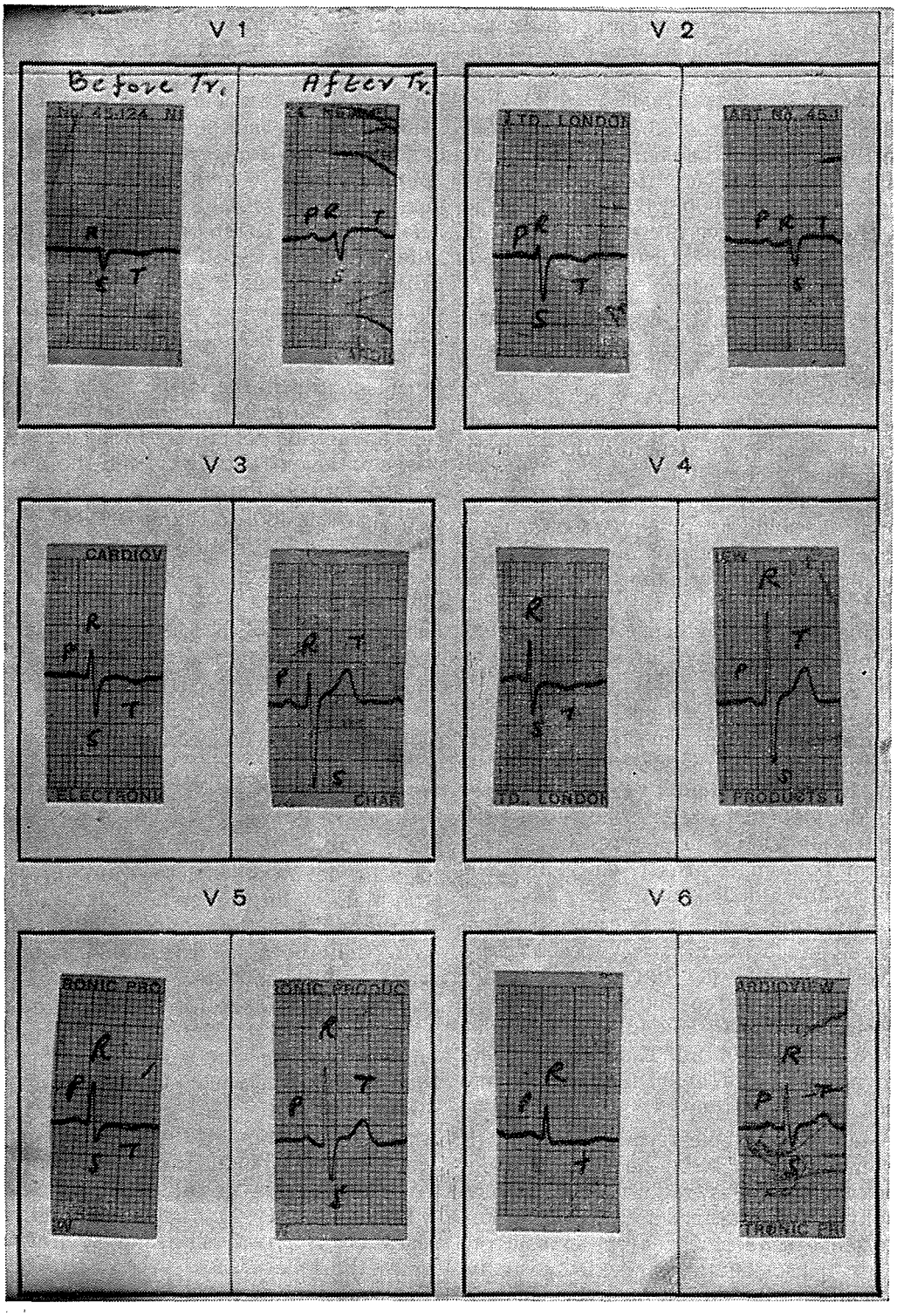
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case of congestive heart failure with a blood pressure of 140/0 and an abnormal electrocardiogram showing no specific pattern. Since aortic incompetence was not present the diagnosis was one of high-output failure with a collapsing pulse which was not due to an aortic lesion. Clinical and laboratory findings ruled out hyperthyroidism and anaemia (radio-iodine uptake and metabolic rate were normal, there were 4,8000,000 r.b.cs. per ml and 92% Hgb) and we were therefore left with the following possibilities of high-output failure:

1. A—V fistula; 2. Severe liver failure; 3. Beri-beri heart disease. At this stage the following investigations were carried out: skeletal X-ray survey, radio-iodine uptake, metabolic rate estimation, liver function tests, electrophoretic pattern study, serum cholesterol, serum electrolytes, serum calcium and phosphorous estimation, alkaline phosphatase, transaminase and haemochromatosis tests all being normal. Wassermann test was negative. Pyruvic acid estimation showed an abnormal rise after glucose: 25 mgm, normal being 1 mgm. Blood urea was 10 mgm. per 100 ml.

A diagnosis of beri-beri heart disease was made and the patient was treated accordingly. He was put on vitamin B_1 100 mgm by intramuscular injection three times daily for one week, followed by 150 mgm. orally daily. He was also given Digoxin 0.25 mgm. twice daily and 100 mgm. hydrochlorticoside weekly.

After one month's treatment the patient felt much better, was able to return to work, did not get out of breath, the murmurs disappeared and his blood pressure was 130/85. His blood urea had risen to 30 mgm. per 100 ml. The electrocardiogram now showed a normal T-wave and higher voltage of the QRS complex.

Mechanism of heart failure in Vit. B_1 deficiency: Since B_1 is the co-enzyme of carboxylase and is required for normal carbohydrate metabolism and utilisation, its absence renders the heart muscle unable to utilise lactate pyruvate normally. This leads to diminution of myocardial O_2 extraction, insufficient energy production and functional failure. In addition there is an accumulation of the vasodilating intermediate catabolites of glucose (pyruvic, lactic and other keto-acids) which cause widespread peripheral arterioral dilatation. This acts like a large arterio-venous fistula augmenting the venous return to the right ventricle and making demands upon the left ventricle for increased output, thus putting a burden on a heart already working at a disadvantage (Konstam G. & Sinclair H. M. 1940). Cardiac catheterisation in a patient with beri-beri heart shows an average cardiac output of 16 litres per minute (normal 4—7 litres) and an O_2 consumption of 355 ml/m (normal average 760 ml/m). Alcoholics who take more of their calories in the form of alcohol (which requires B_1 for its metabolism) and do not eat enough B_1 containing foods, eventually develop heart failure through the mechanism described above.

I would like to thank Dr. R. I. S. Bayliss M.D., F.R.C.P., Physician in the Westminster Hospital, London, for the biochemical tests carried out in the investigation of this case.

Reference

KONSTAM G. & SINCLAIR H. M. (1940), Brit. Heart J., 2, 231.

STAPEDECTOMY WITH A SPRING PROSTHESIS

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In deafness due to otosclerosis the footplate of the stapes becomes ankylosed to the niche of the oval window in such a way that the stapedovestibular joint progressively disappears.

The treatment for this form of deafness is surgical. The stapes is removed and the gap between the incus and the oval window is bridged by means of a prosthesis. The operation is called a stapedectomy and the prosthesis may be either a polythelene tube or Teflon. The oval window is usually closed by means of a vein graft ,but Prof. H. Schuknecht uses as his prosthesis a plug of fat taken from the lobe of the ear (which occludes the oval window) and stainless steel wire which is attached to the incus. Another