VENTRICULAR TACHYCARDIA ASSOCIATED WITH THYROTOXICOSIS

BY

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Though Parry, in 1786, described irregular, as well as forcible and rapid, action of the heart in exophthalmic goitre, it was not until the development of electrocardiography that the nature of the arrhythmias associated with thyrotoxicosis could be clearly distinguished. Auricular fibrillation accounts for most of these arrhythmias—20% in a series of 1,000 cases described by Ernstene (1938). Auricular flutter (Phillips and Anderson, 1927), heart-block (Kremer and Laplace 1936), and bundle-branch block (Digilio, 1938) have been described but are rare. Ventricular tachycardia does not appear to be a recognized arrhythmia in thyrotoxicosis.

In a series of 107 cases of ventricular tachycardia (Armbrust and Levine, 1950) there were no cases attributed to thyrotoxicosis. Coronary disease accounted for the majority, but rheumatic heart disease and congenital heart disease were responsible for some. In 13 cases no aetiological cause was found.

Parkinson and Papp (1947) described a condition termed "repetitive ventricular paroxysmal tachycardia"—runs of ventricular tachycardia interspaced between runs of sinus rhythm. One of their patients, aged 32, with this condition over a period of two and a half years, was found to be mildly thyrotoxic when seen 10 years later, though she then had a normal electrocardiogram with sinus rhythm.

We here describe a case of ventricular tachycardia in a woman with thyrotoxicosis.

Case Report

A woman aged 31 was admitted to hospital complaining of severe palpitations for 24 hours. The attack had been sudden in onset and was associated with some dyspnoea and general weakness. She had noticed similar attacks in the past two months, but they had been less severe, lasting only a few hours. There was no history of rheumatic fever, nor had she been taking digitalis.

On examination there was no oedema or finger-clubbing, and her neck veins were not distended. The pulse rate was over 200 a minute at the apex and her blood-pressure was 100/60. Slight cardiac enlargement and fine crepitations at the lung bases were present, but no hepatic enlargement. An E.C.G. revealed ventricular tachycardia (Fig. 1).

Procainamide, 1 g., was given orally and repeated in four hours. After this treatment the electrocardiogram showed normal sinus rhythm (Fig. 2).

Further investigation did not reveal any evidence of organic heart disease; however, she did have a persistent tachycardia with a sleeping pulse rate of 90–100, and moist warm hands, with some tremor. Her thyroid gland was enlarged and the clinical diagnosis of thyrotoxicosis was confirmed by radioactive iodine studies. Her thyrotoxicosis

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was controlled by potassium perchlorate, and no further attacks of palpitations occurred in the subsequent nine months.

Discussion

No recognized cause of ventricular tachycardia could be found in this case. Ischaemic heart disease appears unlikely in view of the patient's age, sex, and normal electrocardiogram after the attack. There was no clinical evidence of rheumatic or congenital heart disease. She had not received digitalis.

One might argue that this patient had idiopathic ventricular tachycardia unconnected with thyrotoxicosis, but as the attacks ceased after control of the thyrotoxicosis it seems reasonable to incriminate thyrotoxicosis as the cause of the tachycardia.

It is well recognized that thyroxine acts directly on the myocardium. McIntyre (1931) produced tachycardia in denervated dogs' hearts by thyroxine, and experimental evidence suggests that it increases the sensitivity of cardiac muscle to adrenaline. Rosenblum, Hahn, and Levine (1933) produced thyrotoxicosis in animals by thyroid feeding and then studied the effects of adrenaline infusions on their cardiac rhythm by electrocardiogram. Paroxysmal ventricular tachycardia was produced in all animals with doses of adrenaline too small to alter the cardiac rhythm of normal controls. If such a mechanism is responsible for arrhythmia in thyrotoxicosis, it is difficult to explain why ventricular tachycardia is so rare. Furthermore, Burn, Gunning, and Walker (1957) have shown that adrenaline inhibits the production of experimental auricular fibrillation. It seems unlikely, therefore, that adrenaline sensitivity plays a major part in the arrhythmias of thyrotoxicosis.

FIG. 1.—E.C.G. on admission, showing ventricular tachycardia in standard leads.

FIG. 2.—Standard lead 3 following procainamide, showing sinus rhythm.

The occurrence of non-pathogens showed a different pattern; in 11 cases where they were present at the beginning none were found at the final visit. The details are given in Table II.