PAROXYSMAL TACHYCARDIA FOLLOWED BY TEMPORARY INVERSION OF THE T WAVES

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It is now generally recognized that inversion of T waves such as is met with in coronary disease can occur in a number of other conditions also, for example, in myocardial disease due to toxæmia or hypertension, from large doses of digitalis, from the physiological effects of taking cold drinks, as a result of the position of the heart, and even from changes of posture especially in persons of the neuro-circulatory asthenic type.

Attention has, however, only recently been given in the literature to the fact that electrocardiographic features simulating serious coronary disease may in certain cases occur as a transient feature after paroxysmal tachycardia. The condition, furthermore, is of more than mere academic importance, since if not recognized, an erroneous diagnosis of coronary disease may easily be made especially if the attack of paroxysmal tachycardia passes unnoticed or is thought to have been an acute coronary lesion. Either these changes after paroxysmal tachycardia are very rare or it would seem possible that cardiologists have been familiar with their occurrence but have not recorded them.

White *et al.* (1941) in a review of various conditions other than organic disease causing inversion of the T wave in lead II makes no mention of paroxysmal tachycardia, and in a search of British and American papers only eighteen previous cases appear to have been described. Graybiel and White (1934) reported two cases of paroxysmal ventricular tachycardia in young robust adults, one followed by inverted T I and the other by inverted T II and T III; there was no sign of organic disease, and there was a gradual return to a normal cardiogram. Campbell and Elliott (1939) described two cases of paroxysmal tachycardia, one being ventricular, that gave transient changes simulating coronary thrombosis: one of these gave a history of diphtheria, and the other was of long standing and had been suffering from increasing dyspnoea during the intervals between attacks; both died at a later date. Though these two cases differ in their organic nature from the other cases described and from the present one, which except for the electrocardiographic abnormality seemed normal in other respects, Campbell and Elliott nevertheless laid stress on the transient nature of the cardiographic changes following the tachycardia. Cossio, Sabathie, and Berconsky (1941) described four cases, and Campbell (1942) a further three cases that were clinically benign. Currie (1942) described a further case probably of ventricular origin in a girl aged thirteen, followed by inversion of T waves, and a return to a normal cardiogram in one month. Dubbs and Parmet (1942) added a further case, Geiger (1943) one case, Zimmerman (1944) three cases, and Ward (1946) one case. The present case was one of paroxysmal auricular tachycardia and was clinically benign.

PRESENT CASE

A woman, aged 27, was admitted to the Meath Hospital, Dublin, on February 16, 1945. The history was that quite suddenly when she was walking three days previously her heart began to beat very heavily and she found her breathing became difficult. She also felt inclined

to vomit but was unable to, and experienced a throbbing sensation in her throat. She stated that she had a similar attack but milder in character a year previously. There was nothing of importance in the family history. Physical examination was normal except for some bad teeth, the rapid heart action, and a suspicion of œdema in her legs. The heart rate was extremely rapid and regular so that it was impossible to estimate the rate of the pulse at the

FIG. 1.—(A) Paroxysmal auricular tachycardia (16/2/45). (B) Inversion of T I and T II six days after cessation of attack (24/2/45). (C) More normal appearance of T I and T II two days later (26/2/45). (D) Completely normal T I and T II sixteen months later (20/6/46). In each case one complex has been inked in to mark it more clearly.

wrist, but when taken at the apex beat by auscultation it was found to be approximately 240 a minute, a figure that actually agreed with the rate as timed on the electrocardiogram. The method of tapping with the foot synchronously with the sound as heard on auscultation advised by Levine for timing cases of rapid heart action was found helpful in this case as otherwise it would have been very difficult to time at all accurately the extremely rapid heart rate.

Attempts to control the rapid heart action by the ordinary simpler methods were unsuccessful but the attack finally stopped between 7 a.m. and 1 p.m. on the morning of February 18, 1945, after 24 grains of quinidine sulphate had been given, spread over a period of 36 hours, the attack having lasted in all approximately four-and-a-half days. All other investigations both cardiological and otherwise were negative, including the Wassermann reaction.

Subsequent convalescence was uneventful except that the patient complained that she had a very slight attack on February 26 which she stopped herself by ocular pressure. The patient was discharged on March 21, 1945.

The electrocardiograms demonstrate the transient occurrence of inversion of T waves in leads I and II. Fig. 1A on February 16, the day of admission, shows typical paroxysmal auricular tachycardia. Fig. 1B on February 24, six days after the cessation of the attack, shows typical inversion of T I and T II. Fig. 1C taken on February 26, two days later, shows the T waves no longer inverted. Fig. 1D on June 20, 1946, sixteen months later when the patient felt perfectly healthy except that she was pregnant at the time, shows a normal cardiogram.

DISCUSSION

The importance of not confusing a benign condition like the above with serious cardiac disease has already been stressed. It is of interest to consider the possible ætiological factors that may play a part in producing the abnormal electrocardiographic findings. Clearly even if the condition is less rare than it would seem, only a small minority of cases of paroxysmal tachycardia are associated with the abnormal type of cardiogram. Campbell suggests two possible explanations (1) "chemical or other changes in the myocardium as a result of a prolonged attack" and adds that "in this sense the changes would, to some extent, be a measure as to the severity and seriousness of the attack, and might indicate the need for adequate rest" or (2) "that it depends on changes in the position of the heart, and that as the diaphragm sinks, or as the stomach empties, the heart returns to its more normal position with the return to its normal electrocardiographic pattern." While he mentions in support of this view that it would bring the case into line with some of the other T wave changes reported from alterations in the position of the heart, he is inclined to discount this latter theory from the fact that the time taken for recovery from the electrocardiographic changes is measured in days rather than in hours. He sums up his views by saying that the condition "does not indicate any organic disease, but is a completely reversible process indicating some degree of exhaustion or strain of the heart muscle." The title of his paper "Inversion of T waves after long paroxysms of tachycardia" suggests that one of the most dominant factors is the length of the attack, and Currie also refers to this causative factor in support of which was the fact that in his case the changes in the cardiogram were less in an attack of short duration compared with one of longer duration. In the present case the attack lasted probably between four and five days and duration may have been a factor in the changes, but it is also worth noting that the rate of 240 a minute is an exceptionally high one, and since rapid heart action is well-recognized as a strain on the heart, it is possible that in this case the high rate of striking may have played an important part in determining the cardiographic changes. That such changes should occur is not surprising when one remembers that pulsus alternans and anginal pain are quite commonly experienced in paroxysmal tachycardia of

benign type, and that inversion of the T wave in lead I is considered as the cardiographic equivalent of pulsus alternans in cases of coronary disease.

From the practical point of view it is hoped that by the recording of this and other cases, otherwise healthy people may be prevented from being condemned to a life of cardiac invalidism.

SUMMARY

A case of paroxysmal tachycardia with temporary inversion of T waves in leads I and II is reported. This brings the total number of such cases published in British and American journals to nineteen. Stress is laid on the possible influence of the very high rate of tachycardia in this particular case as an ætiological factor in the electrocardiographic changes. The ease with which these changes may be confused with coronary disease is emphasized.

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