Case reports

British Heart Journal, 1973, 35, 220-222.

Unusual effect of direct current shock in ventricular tachycardia associated with myocardial infarction

Elieser Kaplinsky, Naomi Kaplinsky, and Henry N. Neufeld

From the Heart Institute, and Department of Medicine, The Chaim Sheba Medical Center, Tel-Hashomer, and the Tel Aviv University Medical School, Israel

A case of acute myocardial infarction and ventricular tachycardia is presented. Low energy synchronized direct current discharge caused a reversal of polarity of the QRS vector with only minor change in its rate. A second larger synchronized discharge of 50 W sec abolished the ventricular tachycardia and restored sinus mechanism. The relation between this observation and a similar phenomenon, previously described only in the experimental animal subjected to coronary ligation, is discussed.

The introduction of cardioversion was a major breakthrough in the treatment of cardiac arrhythmias. Apart from its therapeutic value, the investigation of the effects of direct current shocks shed new light on the basic mechanisms of cardiac rhythm disturbances.

Recently, in a patient with acute myocardial infarction who developed ventricular tachycardia, a striking inversion of the polarity of the QRS complexes in all leads was observed after a synchronized direct current discharge of 10 watt-seconds (W sec). To our knowledge, this is the first clinical demonstration of this phenomenon which hitherto has been described in the experimental animal only.

Case report

An 84-year-old woman with a history of coronary artery disease was admitted to hospital because of chest pain and palpitations. On admission she was in severe distress. The blood pressure was 100/75 mmHg and signs of mild peripheral and pulmonary congestion were present. The heart rate was 160 a minute and regular with no murmurs. The electrocardiogram revealed broad and regular QRS complexes at a rate of 160 a minute. She was treated with lignocaine and ajmaline intravenously with no effect except for slowing of the ventricular rate. The patient became comatose and anuric, the systolic blood pressure dropped to 80 mmHg, and she was transferred to the cardiology unit for cardioversion. At this stage the ventricular rate was 137 a minute. After a 10 W sec discharge there was a minor change in the heart rate. However, the QRS vector shifted almost 180

degrees (Fig. 1). She was given a second discharge, this time of 50 W sec and normal sinus rhythm was immediately restored. The electrocardiogram in sinus rhythm showed complete left bundle-branch block. After the cardioversion, the patient's condition improved considerably. Three weeks later, however, she died suddenly in the general medical ward. The necropsy revealed an extensive antero-septal myocardial infarction of two to three weeks' duration.

Discussion

The ventricular origin of the arrhythmias in our patient was suggested by the association with acute myocardial infarction and by the severe clinical condition which improved dramatically upon conversion to sinus rhythm. The possibility of supra-ventricular tachycardia with aberrant conduction seemed unlikely, even though not completely ruled out at the beginning. The QRS configuration after conversion to sinus rhythm, though displaying complete left bundle-branch block, was dissimilar to the configuration during the tachycardia.

The mechanism of cardiac arrhythmias associated with acute myocardial infarction has been studied extensively by Wolff, Veith, and Lown (1968). They described the existence of a vulnerable period for ventricular tachycardia in dogs during the acute phase after coronary artery ligation. The energy needed for the cardioversion of the ventricular tachycardia of the vulnerable period was very low. Such low energy direct current synchronized discharges could also produce, in the experimental

FIG. 1 Leads I, II, and III of the electrocardiogram taken before (A) and after a 10 W sec discharge (B). Mean QRS axis in the frontal plane is -40° in A and $+130^\circ$ in B. Notching in the ST segment in leads I and II of A may represent retrograde atrial activation.

FIG. 2 Lead II of the electrocardiogram taken 5 days after ligation of the anterior descending coronary artery in a dog. Ventricular tachycardia at a rate of 250 a minute was produced by a low energy discharge in the vulnerable period. As can be seen in the tracing, a 2.0 W sec discharge did not abolish the ventricular tachycardia, but the QRS complex became opposite in direction to the one before the electrical shock. The rate remained unchanged.

animal, 180 degrees shift in the direction of the ventricular tachycardia with or without changing its rate (Fig. 2). Experience in coronary care units suggests the existence of a similar vulnerability to ventricular tachycardia in the human patient with acute myocardial infarction (Lown *et al.*, 1967). This is based on the mode of initiation of the arrhythmias and the extremely low electrical energy needed for restoration of sinus rhythm. On occasion, even a blow to the chest has been able to revert the arrhythmias (Bornemann and Scherf, 1969). The energy used for converting the ventricular tachycardia to sinus rhythm in our patient was moderately low: 50 W sec. However, the response to the discharge of 10 W sec was most interesting. It revealed a feature previously described only in the experimental ventricular tachycardia of the vulnerable period, namely, the reversal of the QRS vector with little change in the ventricular rate (Fig. 1). In retrospect this served as further evidence for the ventricular origin of the tachycardia in our patient.

Both the low energy needed for cardioversion of ventricular tachycardia of acute myocardial infarc-

tion and the reversal of polarity are most probably associated with large re-entry cycles around the infarction area. Being a surface phenomenon, it might explain the low energy needed for abolishing the ectopic rhythm. Furthermore, the reversal of polarity is also in agreement with the re-entry theory. The low energy discharge might reverse direction of the re-entry cycles around the infarcted region and thus radically change the QRS vector.

References

- Bornemann, C., and Scherf, D. (1969). Paroxysmal ventricular tachycardia abolished by a blow to the precordium. *Diseases of the Chest*, **56**, 83.
- Lown, B., Vassaux, C., Hood, W. B., Jr., Fakhro, A. M., Kaplinisky, E., and Roberge, G. (1967). Unresolved problems in coronary care. *American Journal of Cardiology*, **20**, 494.
- Wolff, G. A., Veith, F., and Lown, B. (1968). A vulnerable period for ventricular tachycardia following myocardial infarction. *Cardiovascular Research*, **2**, 111.

Requests for reprints to Dr. Elieser Kaplinisky, Heart Institute, Chaim Sheba Medical Center, Tel Hashomer, Israel.