THUMPING OF THE PRAECORDIUM IN VENTRICULAR FIBRILLATION

D. C. F. HARWOOD-NASH, M.B., CH.B. (CAPE TOWN), *formerly House Physician, Groote Schuur Hospital*

Thumping of the praecordium is a well-established method of cardiac resuscitation in ventricular standstill, and the subject has recently been reviewed by Scherf and Bornemann.[1] Its use in the termination of ventricular fibrillation has not been sufficiently stressed, however. The purpose of this paper is to describe the use of praecordial thumping and external cardiac massage in reviving a patient with ventricular fibrillation caused by quinidine intoxication.

CASE REPORT

M. McD., a Coloured female aged 48 years, was admitted to Groote Schuur Hospital for treatment of mild cardiac failure associated with atrial fibrillation. Thyrotoxicosis, the underlying cause of her illness, had been partially controlled by 2 doses of I^{131} at spaced intervals.

On examination she was thyrotoxic, with tachycardia, atrial fibrillation, exophthalmus, sweaty palms, and a firm, enlarged thyroid. Iron-deficient anaemia with a haemoglobin level of 8·5 G. per 100 ml. was also present. A slightly elevated jugular venous pressure (3 cm. above the sternal angle), a 5-cm. hepatomegaly, and basal crepitations, were regarded as manifestations of mild heart failure. Her blood pressure was 140/90 mm.Hg. There was no cardiomegaly and no murmurs were present.

Digitalization was begun with 3 gr. of digitalis folia, 8-hourly for 6 doses, followed by a maintenance dose of 1 gr. *b.d.* This did not produce any toxic effects until the second day of maintenance digitalis, when she became nauseated and vomited twice. The atrial fibrillation was slowed and there were no ventricular ectopic beats. Digitalis was discontinued for 48 hours, after which the maintenance dose of 2 gr. daily was recommenced.

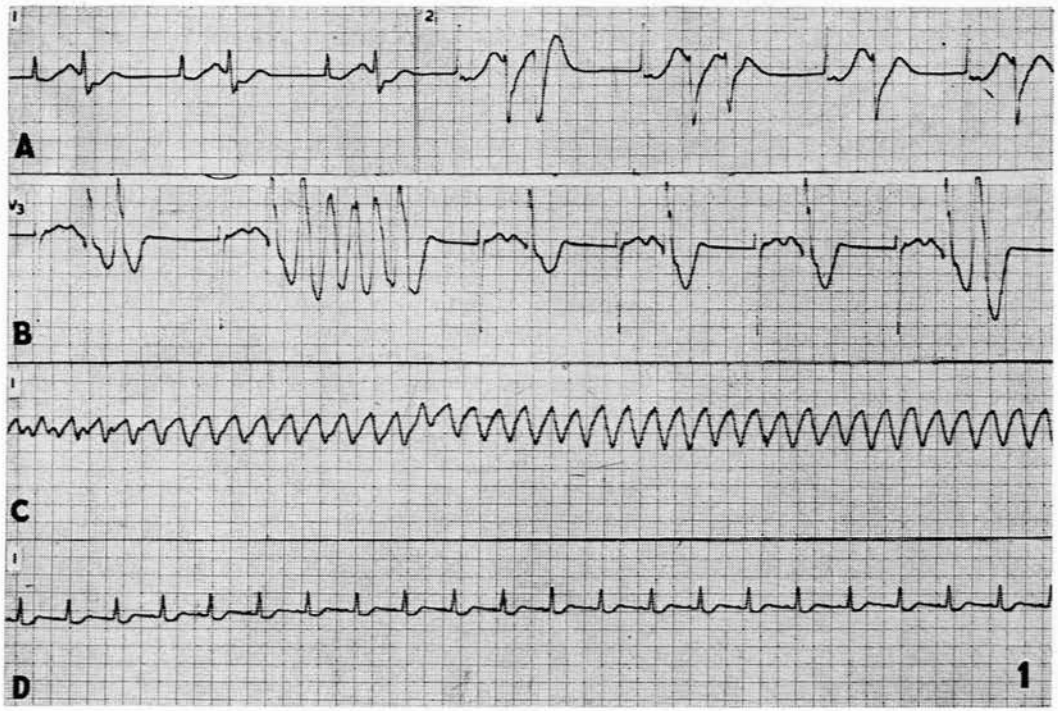

Fig. 1. A. Record taken just before the Stokes-Adams attack, showing nodal rhythm with retrograde conduction to the atria, and ventricular ectopic beats, single or paired.
B. Recorded just before the Stokes-Adams attack as well, showing a small bout of paroxysmal ventricular tachycardia.
C. Showing ventricular flutter-fibrillation during the attack.
D. Record taken after the thumping of the praecordium, showing the restored sinus rhythm, with a quinidine effect in the ST segment.

Conversion of the arrhythmia with quinidine was now attempted; the first course of quinidine, 6 gr. 2-hourly for 5 doses, with electrocardiographic control, was given without success. No toxic effects were noted. The following day a further course of quinidine was given, 9 gr. 2-hourly for 2 doses, and 6 gr. 2-hourly for 3 doses. Again no ill effects were noted clinically or electrocardiographically.

At 9 p.m. that day (i.e. 5 hours after the completion of the

course of quinidine) she felt nauseated, and vomited, and coupling of the pulse caused by ventricular ectopic beats was noted. Digitalis was discontinued; she had a quiet night and slept well. At 6.30 the next morning, however, she looked pale and shocked, and was extremely nauseated, but not hypotensive. The pulse was irregular with intermittent coupling. The electrocardiogram (Fig. 1 A and B) showed nodal rhythm with retrograde conduction to the atria, ventricular ectopic beats, small paroxysms of ventricular tachycardia, and marked quinidine effect.

As the electrocardiographic recording was completed she gave a gasp, became extremely pale, and collapsed in a Stokes-Adams syncopal attack. No pulse was felt, and no cardiac sounds were heard. Since the electrocardiograph machine was still connected, it was switched on and the tracing showed ventricular fibrillation (Fig. 1 C).

Three powerful blows were delivered to the chest over the heart, and a few rhythmical external thoracic compressions were performed. A facial flush appeared, the patient started breathing, and the pulse returned at a normal regular rhythm. The whole episode lasted approximately 2 minutes. The electrocardiogram now showed normal sinus rhythm (Fig. 1 D).

Six grams of potassium chloride, in 3 divided doses, were given to counteract digitalis overdosage, and a regular close watch was maintained. Sinus rhythm continued until 11 a.m., when a further Stokes-Adams attack occurred. Praecordial thumping and external cardiac massage again successfully reverted the arrhythmia within 3 minutes.

The patient remained very drowsy, but conscious, for the next 2 days, after which she recovered completely. Intermittent coupling, caused by ventricular ectopic beats, disappeared after a few hours. At no time was electrical defibrillation used.

Twelve days later the patient was discharged with a sinus rhythm, apparently none the worse for the Stokes-Adams attacks.

DISCUSSION

Ventricular fibrillation is a frequent cause of sudden death, but it is only in recent years that the reversibility of this arrhythmia has been recognized. Before resorting to external or internal defibrillation, the simple method of thumping of the praecordium should always be used. As in this patient, a few vigorous thumps over the heart may terminate ventricular fibrillation and restore normal cardiac action.

External cardiac massage as an effective method of reverting cardiac standstill has only been stressed in the past few years, and is now replacing the courageous and dangerous method of thoracotomy and direct cardiac massage.

A number of authors[1,3,4] have presented series of cases in which external cardiac massage, and mouth-to-mouth breathing when indicated, was successful in maintaining an effective circulation after ventricular standstill, thus allowing time for additional equipment and personnel to be mobilized. In all the patients in whom ventricular fibrillation was encountered, restoration of effective cardiac action (whether ultimately successful or not) was aided by electrical defibrillation.

The potential complications of thumping the chest and/or external cardiac massage, i.e. fracture of the ribs, haemothorax, haemopericardium, liver injury and marrow embolism, are thought to be offset by the now apparent efficacy of this method of resuscitation in the fatal condition of ventricular fibrillation.

The role of quinidine in the production of ventricular fibrillation has not been sufficiently emphasized. Quinidine has been well known as a cause of sudden death, but the mechanism has been obscure.[5] Rainier-Pope *et al.*[2] from this clinic have recently reviewed the subject and reported 2 patients who developed ventricular fibrillation. The case history of a third patient is reported here, also with a fortunate outcome. Quinidine is a potentially dangerous drug and should be used with great care. Thyrotoxic subjects may be particularly sensitive to this drug.[6]

SUMMARY

A patient with thyrotoxic atrial fibrillation is reported, in whom ventricular fibrillation developed during attempted conversion of the arrhythmia with quinidine. Ventricular fibrillation was reversed by prompt, vigorous thumping of the praecordium together with external cardiac massage.

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REFERENCES

1. Scherf, D. and Bornemann, C. (1960): Amer. J. Cardiol., **5**, 30.
2. Rainier-Pope, C., Schrire, V., Beck, W. and Barnard, C. N. (1962): Amer. Heart J. (in press).
3. Kouwenhoven, W. B., Jude, J. R. and Knickerbocker, G. C. (1960): J. Amer. Med. Assoc., **173**, 1064.
4. Baringer, J. R., Saltman, E., Jones, W. A. and Friedlich, A. L. (1961): New Engl. J. Med., **265**, 62.
5. Thomson, G. W. (1956): Circulation, **14**, 757.
6. Sokolow, M. and Perloff, D. B. (1961): Progr. Cardiovasc. Dis., **3**, 316.