children (Boyle *et al.*, 1961 ; Benzing and Kaplan, 1963). During the last 15 years, with the decline in the incidence of inadequately treated septicaemia, more attention has been focused on the suppurative pericarditis which may complicate coronary

(1966). Two of these patients had received corticosteroids during the 48 hours before death. A review of the literature has revealed six further post-mortem reports of this complication (Tedeschi *et al.*, 1950 ; Miller and Edwards, 1951). Suppurative pericarditis had not been considered during life and had been an unexpected post-mortem finding.

Corticosteroids are advocated under certain circumstances in the treatment of the post-infarction (Dressler, 1959) and post-cardiotomy syndromes (Robinson and Brigden, 1963). The post-infarction syndrome was diagnosed in this patient during the seventh week of his illness when he was critically ill with hypotension, pericarditis, and pleurisy. He was treated with prednisone, but in retrospect this may have been more harmful than beneficial.

It is suggested that this patient's infarction became infected, that a myocardial abscess developed, and that this drained spontaneously into the pericardial cavity. Delay in recognition of this complication until pericardial aspiration was performed was partly attributable to lack of awareness of the condition. It is to be hoped that by reporting this case history this rare complication may be recognized more promptly and correct treatment instituted. Treatment with appropriate systemic and local antibiotics may be successful, but if recurrent tamponade occurs early surgical pericardial drainage is essential.

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CASE REPORT

A man aged 75 had been in the ward in March 1967 with myocardial infarction and left bundle-branch block. He was readmitted on the evening of 18 July with further cardiac pain and was found to be in supraventricular tachycardia, 180 per minute with right bundle-branch block. He was given digoxin 1 mg. intravenously without effect, and by the next morning was hypotensive and in failure. He was anaesthetized with nitrous oxide, oxygen, and halothane and given a synchronized direct current shock (100 Wsec.).

The following is a summary of the events which ensued during the next 25 minutes: supraventricular tachycardia with right bundle-branch block—(100 Wsec. synchronized shock)—ventricular tachycardia—ventricular fibrillation—(400 Wsec. shock)—nodal rhythm, 60/min.—A-V dissociation—(external cardiac massage)—multiple extrasystoles—ventricular fibrillation—(sodium bicarbonate 190 mEq, 400 Wsec. shock)—ventricular standstill—(external cardiac massage)—A-V dissociation—ventricular fibrillation—(intravenous infusion, sodium bicarbonate, 400 Wsec. shock)—asystole—(external cardiac massage)—idioventricular rhythm—supraventricular tachycardia with right bundle-branch block—(150 Wsec. synchronized shock)—ventricular fibrillation—(400 Wsec. shock)—idioventricular rhythm—(external cardiac massage)—supraventricular tachycardia without right bundle-branch block—sinus rhythm with ventricular extrasystoles.

At the end of this exciting and worrying session the patient immediately regained consciousness and asked if the procedure had been successful. An hour later he was in left bundle-branch block, as

FIG. 1.—Twelve-lead electrocardiograms taken on: A, 24 January 1966 ; B, 7 March ; C, 29 April ; D, 14 March 1967.

artery disease. Katz (1964) described the histories of two men in whom necropsy showed coronary thrombosis, myocardial abscess, and a purulent pericardial effusion. Similar necropsy findings in a woman aged 74 have been described by Korns (1966) and in two women aged 64 and 73 by Horeau *et al.*

Physical Stimulation of the Heart

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It has been known for some years (Scherf and Bornemann, 1960 ; Semple *et al.*, 1963 ; Don Michael and Stanford, 1963) that stimulation of the heart by a precordial blow can sometimes induce spontaneous rhythm from ventricular standstill. Surgeons also have realized that operative cardiac arrest could sometimes be countered in the past by needle stimulation of the ventricle.

We have had experience in several patients of "starting up" the arrested heart by "thumping" the precordium, and in one of these patients with complete heart block ventricular function was restored from standstill on over 20 occasions within the space of an hour by this simple procedure, while preparations were being made for transvenous pacing. However, to our knowledge physical stimulation has never been shown to be capable of reversing arrhythmias other than asystole. Some patients previously restored from cardiac arrest by this simple method may well have been in ventricular fibrillation, but usually there has been no E.C.G. confirmation to distinguish this from ventricular standstill.

That this physical method may sometimes be effective in less severe arrhythmias is shown in the following case.

he had been after the myocardial infarction in March 1967. Thereafter he was maintained on lignocaine infusion 1 mg./min. to a maximum of 500 mg. in 24 hours and remained in sinus rhythm for 48 hours with only occasional extrasystoles. By this time the infusion had been discontinued and he felt well, was in sinus rhythm 80/min., and the blood pressure was 120/70.

At this time he suddenly relapsed to the original dysrhythmia—supraventricular tachycardia with right bundle-branch block. On this occasion, as an alternative to further electrical cardioversion but while being monitored on the "cardioverter," he was given a sharp blow on the sternum with no effect. A similar heavy thump with the ulnar side of the clenched fist on the precordium at the cardiac apex immediately induced sinus rhythm with left bundle-branch block (see Fig.). Over the next few days he had runs of ventricular extrasystoles, bigeminy, and multifocal extrasystoles requiring further lignocaine infusion followed by tabs. phenytoin 100 mg. t.i.d.

A, Tracings showing supraventricular tachycardia with right bundle-branch block in a selection of leads. B, Effect of a precordial blow (↑) showing sinus beats, ventricular extrasystoles, and eventually left bundle-branch block. C, Stabilization in left bundle-branch block a few seconds later.

One week later, while he was still being monitored, his rhythm again relapsed to supraventricular tachycardia with right bundle-branch block, and once more sinus rhythm was easily induced by a single precordial blow. A tendency to recurring ventricular extrasystoles persisted, however, and the phenytoin dose was increased to 400 mg. daily.

On 7 August, three weeks after the original electrical cardioversion, he again developed tachycardia. He was no longer being monitored, but an E.C.G. tracing showed supraventricular tachycardia, this time with left bundle-branch block. Once more a precordial thump induced sinus rhythm and left bundle-branch block with multifocal extrasystoles, which settled on administration of antiarrhythmic drugs lignocaine and phenytoin. He had no further episodes of arrhythmia, his E.C.G. showing the same picture of left bundle-branch block as it had done after the initial myocardial infarction in March 1967.

By 21 August he was getting up in the ward. Clinically and radiologically his heart was enlarged, but, on daily diuretic, there was no evidence of failure. He was still having fairly frequent ventricular extrasystoles, but was well enough to be discharged from hospital on 30 August, continuing on phenytoin 100 mg. t.i.d. as an antiarrhythmic drug.

Haemodynamic Effects of Balloon Septostomy in Tricuspid Atresia

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Tricuspid atresia has been regarded as one of the rare congenital cardiac anomalies, but it must be considered in the differential diagnosis of every infant with cyanotic heart disease. Campbell (1955) stated that tricuspid atresia is present in about 5% of patients with cyanotic congenital heart disease; Keith *et al.* (1958) placed the incidence at 3.2%.

The physiological consequences of the anomaly are manifold. Arterial desaturation is caused by complete mixing of systemic venous and pulmonary venous blood in the left atrium. The degree of unsaturation and cardiac handicap depends on the

He was readmitted on 6 September again in supraventricular tachycardia and with evidence of congestive failure. On three occasions over the next 48 hours a precordial thump converted this rhythm to sinus rhythm. On each occasion the procedure was performed with the patient monitored on the cardioverter, and with the shock paddles prepared and an intravenous drip available for giving sodium bicarbonate or other drugs in the event of more serious arrhythmia developing. In view of the continuing cardiac irritability with multifocal extrasystoles he was then put on a procainamide continuous infusion, 4 g. in 24 hours, which had the effect of almost clearing the ventricular ectopic beats.

COMMENT

Thus physical stimulation of the heart, like electrical stimulation and drugs such as digitalis and quinidine, would seem to have the ability to induce and reverse arrhythmias. In our patient it seems that the direct-current precordial shocks given during the first attack of arrhythmia were too powerful. It will be interesting to discover whether this simple physical method can be used as an alternative to electrical cardioversion in other types of arrhythmia. It has the advantage that no anaesthetic is necessary, but we would strongly advise that the patient's E.C.G. be monitored, with a defibrillator to hand, when the stimulus is given. There is evidence that such a blow can produce an ectopic beat which, by falling in the vulnerable T-wave zone, might conceivably induce ventricular fibrillation (Smirk and Palmer, 1960). This could account for the very rare and otherwise inexplicable sudden death in the boxing ring.

It seems that response to electrical and physical stimulation is intensified in the diseased and hypoxic myocardium and by certain drugs such as digitalis (Scherf and Bornemann, 1960), and a single blow, instead of eliciting a single ectopic beat as it does in the normal heart, might well induce unwelcome tachycardia or ventricular fibrillation in the presence of such cardiac instability.

Unfortunately there is no way of synchronizing the stimulus. Thus until more is known about the possible dangers of this treatment its use should be restricted to cardiologists with experience in treating acute arrhythmias, employing all the emergency precautions normally taken with planned electrical precordial shock.

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pulmonary flow and the size of the atrial septal opening. If the atrial septal defect is small it causes raised right atrial pressure, and this in turn results in raised systemic venous pressure and severe congestive failure. In such cases enlargement of the atrial septal defect should decrease central venous pressure and thus relieve the congestive heart failure. We report here the application of the technique of atrial septostomy in a small infant with tricuspid atresia and transposition of the great vessels with an extremely small intra-atrial communication.

CASE REPORT

A full-term male infant born on 9 February 1967 and weighing 7 lb. 6 oz. (3,350 g.) was noted to be slightly cyanosed at birth. For two weeks, while the infant was nursed at home, he became dyspnoeic and more cyanosed after feeds. He was admitted to