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ARRHYTHMIAS FOLLOWING CARDIAC SURGERY*

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One of the many new problems created by cardiac surgery is the management of post-operative disturbances of rhythm.

The following observations are based on 48 cases seen during the last 18 months at the Brompton Hospital, where it is our practice to give digitalis before operation to all cardiac cases.

Clinically the problems fall into three main groups: (1) Cases of "tachycardia" which give rise to concern during the first post-operative week; (2) the restoration of sinus rhythm after mitral valvotomy in cases with established atrial fibrillation before operation; (3) cases in which atrial flutter developed after closure of an atrial septal defect.

The management of heart block after closure of ventricular septal defects and problems of resuscitation associated with ventricular fibrillation are not included.

GROUP 1: "TACHYCARDIA" DURING FIRST POST-OPERATIVE WEEK

This group accounted for 40% (19 cases) of the arrhythmias seen. Clinical and electrocardiographic examinations rarely fail to establish the nature of the arrhythmia; it is, however, much more difficult to determine whether there is a specific factor responsible.

The common disturbances of rhythm occurring at this time are (*a*) sinus tachycardia, (*b*) supraventricular tachycardia, (*c*) atrial fibrillation, and (*d*) multiple ectopic beats, coupling, and nodal rhythm.

(*a*) SINUS TACHYCARDIA is inevitable after any operation and normally starts to subside between the second and fifth post-operative day. Persistent tachycardia at a rate of over 130 beats per minute after the first 24 hours may give rise to some concern. Fluid imbalance and haemorrhage are the first things to think of, and the latter is not always easily excluded, particularly when bleeding is confined within the pericardium. The pericardium can seal off remarkably quickly after surgery, and a small haemorrhage into the pericardial sac may cause tachycardia without obvious signs of tamponade. Similarly, a small haemothorax may keep the pulse rate high. The commonest cause of persistent tachycardia, however, is incomplete expansion of the lung, and there is usually a dramatic fall in pulse rate after this has been corrected by physiotherapy or bronchoscopy.

There still remain a considerable number of patients in whom no adequate explanation can be found for the tachycardia. In these cases it may represent an unusually prolonged reaction to the general metabolic disturbance caused by the operation, but if it fails to subside by the end of the first post-operative week the possibility of haematoma or infection should be considered. Sepsis, pulmonary embolism, and the post-cardiotomy syndrome have not, in my experience, been the cause of persistent tachycardia during the first four or five post-operative days.

If the tachycardia persists, increasing the dose of digitalis may slow the atrial rate. This pharmacological action is more widely appreciated in America than in this country. Sedation may be useful, but quinidine has no place in the treatment of post-operative sinus tachycardia.

There is a small group of cases of congenital heart disease in which persistent post-operative tachycardia may be sinister. These are cases of pulmonary stenosis with or without a normal aortic root, in which the stenosis has been inadequately relieved or where a secondary functional stenosis or infundibular shutdown is embarrassing the ventricle. In Fallot's tetralogy persistent tachycardia may indicate infundibular shutdown which can lead to syncope and death, and in pure pulmonary stenosis obstruction to right ventricular outflow from a secondary functional stenosis may cause acute right heart failure. Such cases should be nursed in an oxygen tent and heavily sedated. Recent work on syncope in Fallot's tetralogy suggests that morphine is the best drug for relaxing infundibular tone (Wood, 1958; Braudo and Zion, 1959).

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(*b*) and (*c*) SUPRAVENTRICULAR TACHYCARDIA AND ATRIAL FIBRILLATION.—Occurring at or shortly after operation this is rarely a difficult problem. Any of the predisposing causes of sinus tachycardia may be responsible. In atrial fibrillation in spite of pre-operative digitalis the initial ventricular rate is always fast, around 120–140 per minute. Digitalis should be increased, usually by mouth, until adequate slowing has been achieved. In supraventricular tachycardia digitalis should again be given to increase the degree of atrio-ventricular block from the usual 2:1 to 3 or even 4:1, thus slowing the ventricular rate. Sinus rhythm may return without further treatment. If the arrhythmia persists and quinidine is required this should be withheld until the third or fourth post-operative week (vide infra).

One group of cases which seem particularly prone to atrial fibrillation are elderly patients who have extensive resection of a lung and in whom the pericardium has been opened. These patients almost invariably revert to sinus rhythm spontaneously.

(*d*) MULTIPLE ECTOPIC BEATS, COUPLING, AND NODAL RHYTHM.—These are usually due to excessive digitalis, but ventricular ectopics are more common after an extensive ventriculotomy than after atrial surgery. Withdrawal of digitalis and additional sedation may be expected to correct these arrhythmias. During the early post-operative period quinidine should be avoided as more than one case of closed aortic valvotomy has been lost associated with quinidine therapy at this time (Brock, 1959).

GROUP 2: ATRIAL FIBRILLATION AND RESTORATION OF SINUS RHYTHM AFTER MITRAL VALVOTOMY

All cases in this group have established atrial fibrillation before surgery. Those developing atrial fibrillation at or shortly after surgery have already been discussed.

After successful mitral valvotomy it is our practice to attempt to restore sinus rhythm in all cases, irrespective of the duration of atrial fibrillation. Using quinidine sulphate we revert 50% of cases, but in about half of these fibrillation recurs before the patient leaves hospital or shortly afterwards (Wood, 1954).

We do not use anticoagulants when attempting to restore sinus rhythm after successful valvotomy because the risk of post-operative embolus is negligible. The rarity of this complication is attributed

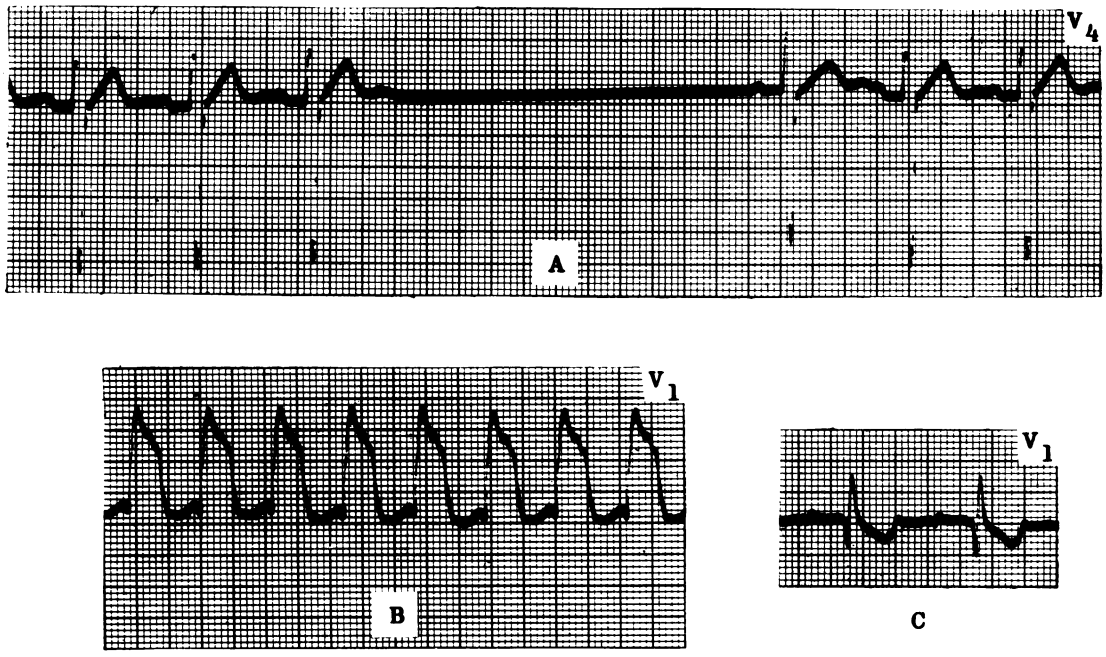

FIG. 1.—A, Sino-auricular block after a total daily dose of 13 grains quinidine sulphate (woman, 64 years, rheumatic heart disease, dominant mitral incompetence and resistant supraventricular tachycardia). B, Atrial flutter with a ventricular response of 145/min. and widening of QRS to 0·24 sec. occurring after a total daily dose of 32 grains quinidine sulphate. (Man, 20 years, developed atrial flutter after closure of an atrial septal defect.) C, Same patient as in B, showing his usual QRS complex in V_1 of 0·08 sec.

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to the removal of the left atrial appendix, washing out all fresh clot from the left atrium at the operation, and the improved flow of blood through the left heart.

It is our usual practice to start quinidine during the third post-operative week. The patient is fully digitalized and confined to bed. After a test dose of 3 grains we give 3, 5, and 8 grains at six-hourly intervals on the first day, 8 grains t.d.s. on the second and third days, 10 grains t.d.s. on the fourth day, 8 grains every two hours for four doses on the fifth day, and 10 grains every two hours for four doses on the sixth day. An electrocardiogram is taken before treatment on the sixth day and again before the final dose of quinidine on that day.

Toxic symptoms, which include severe nausea, vomiting, diarrhoea, tinnitus, deafness, disturbances of rhythm, and widening of the QRS complex on the electrocardiogram, must be heeded and treatment stopped, otherwise sooner or later sudden death may occur. Two examples of arrhythmias due to quinidine are illustrated in Fig. 1. The main factors mitigating against successful restoration of sinus rhythm are an incomplete valvotomy, mitral incompetence (pre-existing or created at operation), and active pericarditis at the time of quinidine therapy. The restoration of sinus rhythm is important and should be achieved whenever possible since atrial fibrillation alone can reduce the resting output by one third.

Group 3: Atrial Flutter after Closure of Atrial Septal Defects

In a total of 100 cases in which the atrial septal defect was closed using hypothermia, post-operative atrial flutter occurred in nine.

In most patients it stopped spontaneously, but two patients required quinidine and a further two have defied all treatment and atrial flutter has now persisted for over two years in each. Half the cases that develop this complication do so in the first post-operative week, the remainder do so in the third to sixth post-operative week. The atrial rate varies between 250 and 375 per minute with an average of 294 per minute. All patients were taking digitalis when atrial flutter occurred.

As with so many other arrhythmias no cause was found although there were a number of associated factors. Atrial flutter never followed the closure of a small defect. The size of the defect in the affected cases varied between 3.5×3.5 cm. and 6.0×8.0 cm. Two of the nine cases had severe post-operative pericarditis, a third had a pulmonary infarct, and a fourth developed thrombosis of transposed anomalous veins. The age and sex of the patient, the development of ventricular fibrillation at operation, and the length of time the circulation was occluded did not appear to be significant factors. This complication, however, cannot be ignored if 2% of patients who have had otherwise successful operations develop persistent atrial flutter.

Summary

The significance and management of the common arrhythmias occurring during the first week after cardiac surgery are described.

The problems of restoring sinus rhythm after mitral valvotomy and the association of atrial flutter with the surgery of atrial septal defect have been discussed.

The patients studied were under the care of Dr. Paul Wood and one of the following surgeons: Sir Russell Brock, Mr. N. R. Barrett, Mr. O. S. Tubbs, Mr. W. P. Cleland, or Mr. M. Paneth. I am most grateful to these members of the Brompton Hospital staff for their help and co-operation. I am particularly indebted to Dr. Paul Wood for his help in preparing this paper.

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