Endocardial pacing in a patient with sinus node dysfunction and absent right superior vena cava

DAVID R RAMSDALE, RICHARD G CHARLES
From the Regional Cardiothoracic Unit, Broadgreen Hospital, Liverpool

SUMMARY A patient with sinus node dysfunction and absent right superior vena cava underwent stable temporary and permanent endocardial pacing via a left sided superior vena cava. Active fixation of the electrode and epicardial pacing were not necessary.

Persistence of the left superior vena cava occurs in 0-5% of the population, and 10% of these individuals also lack a patent right superior vena cava. Since such patients may have associated structural and functional abnormalities of the conduction system they may require implantation of a cardiac pacemaker. Problems, however, have arisen after insertion of transvenous pacing electrodes via persistent left sided superior vena cavae, and active electrode fixation or elective epicardial pacemaker implantation have been advocated as methods of choice especially if the abnormal anatomy is diagnosed preoperatively. We report a patient with a persistent left but absent right superior vena cava and sinus node dysfunction in whom both temporary and permanent endocardial pacing were simple, successful, and uncomplicated and active fixation of the electrode was unnecessary.

Case report

A 58 year old man was admitted for an oesophagectomy because of a benign peptic stricture. He had no cardiac symptoms and in particular no dizziness or syncope. On admission he had an irregular slow pulse (43 beats/min) and a blood pressure of 110/80 mm Hg. There were no cardiac murmurs, and the remainder of the physical examination was normal. He was currently taking no medication.

An electrocardiogram on admission showed a slow sinus rate, junctional escape rhythm, and sinus arrest with junctional escape impulses. Ambulatory Holter monitoring confirmed a profound sinus bradycardia with night rates as low as 33 beats/min. In addition, there were multiform atrial and ventricular extrasystoles and prolonged periods of sinus arrest lasting up to 4-5 s with nodal escape impulses. There were no associated symptoms, although the most serious bradycardias occurred during sleep. The radiograph
Endocardial pacing in a patient with sinus node dysfunction and absent right superior vena cava

Fig. 2 Radiograph showing permanent pacemaker electrode (E) lead passing from the right subclavian vein to the apex of right ventricle (RV) via the left superior vena cava, coronary sinus, and right atrium.

showed a normal sized heart and no pulmonary disease.

A prophylactic temporary pacing electrode was inserted via a right subclavian vein puncture before his scheduled oesophagectomy. During the procedure it was noticed that the pacing lead crossed the midline and entered a persistent left sided superior vena cava, which drained into the right atrium via a large dilated coronary sinus. This was confirmed by contrast venography of the right subclavian vein. There was no right sided superior vena cava (Fig. 1). Emerging from the coronary sinus, the lead formed a sharp angle as it entered the right ventricle across the tricuspid valve. A stable position was easily achieved with a threshold of 0.35 V at a pulse width of 0.5 ms. After successful oesophagectomy a multiprogrammable permanent pacemaker was implanted in the right pectoral region. A 62 cm Sorin S80 sintered platinum tip endocardial electrode (wedge flange) was inserted via a second right subclavian vein puncture and advanced along the same course as the temporary lead. Again there was no difficulty in obtaining a satisfactory stable position in the right ventricle (Fig. 2) and a pacing threshold of 0.49 V at a pulse width of 0.5 ms was achieved. The endocardial R wave measured 8.6 mV.

Nine months after pacemaker implantation the patient was still without symptoms, and an electrocardiogram and chest x-ray film confirmed normal pacemaker function and a stable electrode position.

Discussion

Normally, during the second month of intrauterine life the left brachiocephalic vein develops as an oblique cross anastomosis between the left and right anterior cardinal veins. Blood from the head, neck, and upper limbs thus channels into the right common cardinal vein, which together with the terminal portion of the right anterior cardinal vein becomes the superior vena cava. In 0.5% of the population failure of development of the cross anastomosis leads to persistence of the left anterior and common cardinal veins and a left sided superior vena cava. In 10% of these cases an anastomosis develops between the two anterior cardinal veins, but drainage is from the upper right to the lower left. This may lead to an atretic or absent right sided superior vena cava as the lower part of the right anterior cardinal vein regresses and the right subclavian and internal jugular veins drain into a left superior vena cava via a right brachiocephalic vein. Such an anatomical anomaly was recognised in our patient only at the time of temporary pacemaker implantation for associated sinus node dysfunction. Contrast venography and thoracotomy confirmed the abnormality.

Abnormal development of the right horn of the sinus venosus and right anterior cardinal vein may considerably displace the anatomical location of the sinoatrial node and result in sinus node dysfunction. Camm et al described symptomatic sinus node dysfunction in a patient with absent right and persistent left superior vena cava. Our patient with the same anatomical abnormality was asymptomatic, and evidence of sinus bradycardia, nodal escape rhythm, and ventricular escape impulses was detected only by routine electrocardiography before surgery for an unrelated problem. Holter monitoring was useful in identifying potentially serious sinus arrest and in providing the indication for permanent pacemaker implantation. The P wave vector of the dominant rhythm was, however, normal, and an electrophysiological study confirmed normal impulse initiation from the high right atrium.

In our case, temporary pacemaker implantation was necessary before major thoracic surgery, and the passage of the electrode through the right subclavian vein, left superior vena cava, coronary sinus, right atrium, and right ventricle was simple and uncomplicated. The electrode remained stable for 14 days.

0-5 ms was achieved. The endocardial R wave measured 8.6 mV.

Nine months after pacemaker implantation the patient was still without symptoms, and an electrocardiogram and chest x-ray film confirmed normal pacemaker function and a stable electrode position.

Discussion

Normally, during the second month of intrauterine life the left brachiocephalic vein develops as an oblique cross anastomosis between the left and right anterior cardinal veins. Blood from the head, neck, and upper limbs thus channels into the right common cardinal vein, which together with the terminal portion of the right anterior cardinal vein becomes the superior vena cava. In 0.5% of the population failure of development of the cross anastomosis leads to persistence of the left anterior and common cardinal veins and a left sided superior vena cava. In 10% of these cases an anastomosis develops between the two anterior cardinal veins, but drainage is from the upper right to the lower left. This may lead to an atretic or absent right sided superior vena cava as the lower part of the right anterior cardinal vein regresses and the right subclavian and internal jugular veins drain into a left superior vena cava via a right brachiocephalic vein. Such an anatomical anomaly was recognised in our patient only at the time of temporary pacemaker implantation for associated sinus node dysfunction. Contrast venography and thoracotomy confirmed the abnormality.

Abnormal development of the right horn of the sinus venosus and right anterior cardinal vein may considerably displace the anatomical location of the sinoatrial node and result in sinus node dysfunction. Camm et al described symptomatic sinus node dysfunction in a patient with absent right and persistent left superior vena cava. Our patient with the same anatomical abnormality was asymptomatic, and evidence of sinus bradycardia, nodal escape rhythm, and ventricular escape impulses was detected only by routine electrocardiography before surgery for an unrelated problem. Holter monitoring was useful in identifying potentially serious sinus arrest and in providing the indication for permanent pacemaker implantation. The P wave vector of the dominant rhythm was, however, normal, and an electrophysiological study confirmed normal impulse initiation from the high right atrium.

In our case, temporary pacemaker implantation was necessary before major thoracic surgery, and the passage of the electrode through the right subclavian vein, left superior vena cava, coronary sinus, right atrium, and right ventricle was simple and uncomplicated. The electrode remained stable for 14 days.
throughout the perioperative period. A permanent pacing electrode with a wedge flange tip (Sorin S80) was inserted via the same route and with similar ease and remains stable nine months postoperatively. Furthermore, we have been similarly successful with this electrode in pacing a patient with complete atrioventricular block via a left superior vena cava. In this case a separate right sided superior vena cava was detected by venography.

Reports of electrode displacement after pacing via a left superior vena cava\(^6\)\(^7\)\(^1\) have caused some concern that the abnormal anatomy may preclude transvenous pacing without active fixation of the electrode. There is, however, no good evidence that the cause of the electrode displacement is the abnormal anatomy by itself. Amikam \textit{et al}\(^{14}\) have suggested that initial incorrect positioning of the electrode may have been responsible in two cases,\(^6\)\(^1\)\(^1\) whereas in a third\(^7\) electrode displacement was suspected only during generator replacement for battery failure. Our experience is similar to that of previous workers,\(^1\)\(^2\)\(^6\)\(^7\)\(^1\)\(^7\) who have also been successful in pacing patients via a left sided superior vena cava using more modern, passively fixed, tined (Medtronic 6961) and wedge flange (Cordis 322-620) leads. This should be reassuring when the anatomical abnormality is discovered for the first time during the passage of such a lead. We would not recommend atrial loop formation as described by Amikam \textit{et al},\(^{14}\) Ronnevik \textit{et al},\(^{17}\) and Rose \textit{et al}\(^{18}\) since this may give rise to lead instability.\(^9\) If difficulty is encountered in obtaining or maintaining a stable electrode position in patients with a single left superior vena cava then either the electrode should be actively fixed or an epicardial pacemaker implanted.

References
2 Winter FS. Persistent left superior vena cava. Survey of world literature and report of 30 additional cases. \textit{Angiology} 1954; 5: 90–132.
6 Kukral JC. Transvenous pacemaker failure due to anomalous venous return to the heart. \textit{Ches} 1971; 59: 458–61.