Intracardiac Electrocardiogram in Endomyocardial Fibrosis

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Endomyocardial fibrosis is a common form of heart disease in tropical Africa. In Uganda it accounts for an incidence of approximately 12 per cent in the Cardiac Clinic at Mulago Hospital (D'Arbela and Somers, 1964). The established disease is characterized by fibrosis of the mural endocardium at one or more of three sites: the apex of the right ventricle, the posterior wall of the left ventricle, and the apex of the left ventricle. In endomyocardial fibrosis, unlike fibro-elastosis, the normal layers of the endocardium are destroyed and replaced by thick scar tissue. Recent pathological studies suggest that collagen degeneration is a characteristic process (Connor *et al.*, 1967, 1968). Cardiac auto-immunity reactions have also been shown in patients who have died of endomyocardial fibrosis (van der Geld *et al.*, 1966).

The clinico-pathological syndromes in endomyocardial fibrosis depend on the location of the lesion, predominantly right, predominantly left, or biventricular. The main clinical and haemodynamic features of endomyocardial fibrosis are now well known (Shillingford and Somers, 1961; Parry, 1964; World Health Organization, 1965; Emslie-Smith, 1966; Somers, Brenton, and Sood, 1968b; Somers *et al.*, 1968a; Fowler and Somers, 1968). The diagnosis is usually made on clinical grounds but can be confirmed, where facilities are available, by selective ventricular angiocardiology (Cockshott, 1965). The ordinary electrocardiogram usually shows abnormal P waves, a low-voltage QRS, and non-specific changes in the T wave; it is not diagnostic (Williams and Somers, 1960; Somers *et al.*, 1968b).

The intracardiac electrocardiogram recorded during routine cardiac catheterization yields useful additional information in the investigation of congenital and rheumatic heart disease (Emslie-Smith,

1955; Watson, 1964). We report here a study of the application of intracardiac electrocardiology to the investigation and diagnosis of endomyocardial fibrosis affecting the right ventricle predominantly.

SUBJECTS AND METHODS

Twenty-one patients were investigated by cardiac catheterization, one of them twice. There were 10 male and 11 female patients, and their ages ranged from 10 to 30 years. Of the 21 patients, 16 were of Rwanda/Burundi origin, immigrant people in Uganda, in whom the incidence of endomyocardial fibrosis is known to be high (D'Arbela and Somers, 1964). The diagnosis was confirmed in all 21 patients; in 17 the diagnosis was made by right ventricular selective angiocardiology, in 3 at necropsy, and in 2 at pericardiotomy to exclude constrictive pericarditis. Both latter patients had presented with pericardial effusion followed by the signs of "constrictive" heart disease, and had been studied early in the series before the facility of routine cine-angiocardiology had become available. All the patients had severe right ventricular endomyocardial fibrosis except one whose disease was classified as moderate.

Standard cardiac catheterization was performed through the right saphenous vein in all patients except one in whom an antecubital vein was used. The electrode Courmand catheters (United States Catheter and Instrument Corp.) carried a nickel-silver ring electrode at the very tip and were used in sizes 7F and 8F. The catheterization was controlled by image-intensified screening with television display. The intracavitary pressure pulses and the intracardiac electrocardiogram were continuously monitored on an oscilloscope, and simultaneously recorded on a direct-writing 6-channel recorder (Sanborn), together with a control lead II electrocardiogram. Very special attention was paid to situations in which it was judged that the tip of the catheter lay against the endocardium: this was suspected by a feeling of resistance to the onward passage of the catheter, with either the characteristic S-T elevation of ventricular endocardial contact in the intracardiac electrocardiogram or damping of the ventricular

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pressure pulse, or both. Selective right ventricular single-plane cine-angiocardio-grams were made using either sodium iothalamate (Conray 420) or meglumine/sodium iothalamate (Cardio-Conray) as the contrast medium. In 4 patients an endocardial biopsy was made with the Konno biotome (Konno and Sakakibara, 1963). It was found that when the proximal end of the endocardial biotome was appropriately connected to the electrocardiograph its tip could be used as an exploring electrode.

RESULTS

General Observations on the Intracardiac Electrocardiogram in Right Ventricular Endomyocardial Fibrosis. In severe endomyocardial fibrosis of the right ventricle the dense white scar tissue may be several millimetres thick and is thickest in the apical region. It may extend towards the tricuspid ring and encase the papillary muscles. The apex of the cavity may be obliterated by scar tissue and the right ventricular cavity may be shrunken and contracted. The infundibulum of the right ventricle is usually dilated (Fig. 1). During the catheterization of such a ventricle the catheter appears to move directly from the region of the tricuspid valve to the infundibulum, and right ventricular selective angiocardio-grams confirm the absence of the usual apical cavity (Fig. 2).

In view of these gross anatomical changes, it was surprising to find that the QRS complex of the electrocardiogram, recorded from a position free in the cavity of the right ventricle, was generally quite normal. In 10 patients the right ventricular QRS complex was of the form rSr' , or rS with slight notching, but of normal width. This, how-

FIG. 1.—Transverse section of a heart with severe right ventricular endomyocardial fibrosis, viewed from above. The cavity of the fibrosed, shrunken right ventricle (RV) and the dilated infundibulum (I) are shown. The cavity of the left ventricle is also seen (photograph by courtesy of Dr. D. H. Connor).

ever, is a common finding in the intracardiac electrocardiogram from the right ventricular cavity of patients with congenital and rheumatic heart disease, and in only one of these patients did the

FIG. 2.—Single frame from a postero-anterior right ventricular selective angiogram in a case of severe right ventricular endomyocardial fibrosis. The contrast medium outlines the inflow tract and infundibulum of the right ventricle but the apex (A) is obliterated. Because of tricuspid incompetence the contrast medium has regurgitated into the right atrium.

scalar electrocardiogram show the pattern of even incomplete right bundle-branch block. In one patient the right ventricular QRS had the form *QRS* (see Fig. 4). Late R waves of this type are occasionally found in sites near the tricuspid valve in patients with rheumatic and congenital heart disease (Emslie-Smith, 1956).

With right ventricular endomyocardial fibrosis involving the papillary muscles, chordae, and leaflets of the tricuspid valve, tricuspid incompetence is common, and often there is enlargement of the right atrium, sometimes to aneurysmal dimensions. Six of our patients had atrial fibrillation or atrial flutter at the time of catheterization, but from many of the others very large P waves were recorded from the cavity of the right atrium: in 7 cases the amplitude of the intra-atrial P wave was over 3 millivolts and in 2 it measured 5 millivolts. All these patients were shown to have huge right atria on angiocardiology. The mean right atrial pressures in the 6 patients with atrial flutter or fibrillation were all over 20 mm. Hg. In the 15 patients with sinus rhythm, the mean right atrial pressures were over 20 mm. Hg in 7 patients. In the remaining patients the pressures were above 14 mm. Hg except in one patient whose pressure was 11 mm. Hg.

FIG. 3.—X-ray film taken during the catheterization of a heart with severe right ventricular endomyocardial fibrosis. The tip of the catheter lies at the shrunken "apical" region of the right ventricle.

Localization of Catheter Tip. In severe right ventricular endomyocardial fibrosis, with an obliterated apex, a dilated infundibulum, a huge dilatation of the right atrium, and tricuspid incompetence, the pressure pulses from the pulmonary artery, the right ventricular cavity, and the right atrium become progressively distorted, and tend more and more to resemble each other. The form of the pressure pulse can then no longer be relied upon to indicate the position of the catheter tip (Somers *et al.*, 1968a). The anatomical distortion of the heart may be so great that localization of the catheter tip by screening is highly inaccurate (Fig. 3). We found, however, that the form of the intracardiac electrocardiogram clearly indicated the position of the catheter tip (Fig. 4). This information was immediately available at cardiac catheterization, when the electrocardiogram was continuously monitored on the oscilloscope, and proved extremely useful in the later interpretation of the permanent records (Fig. 5).

Endocardial Contact Potentials. When an intracardiac electrode touches the normal endocardium, the intracardiac electrocardiogram is altered in a characteristic way. There is S-T elevation in records from the right ventricular endocardium and SaTa elevation in records from the endocardium of the right atrium. Pressure on the catheter tip, such as is inevitable during the forward passage of a catheter in cardiac catheterization, readily increases the height of the S-T elevation. As the S-T elevation increases, the S wave diminishes, and ultimately the complex may resemble a monophasic action potential with the R wave represented by a small notch on the upstroke. These normal endocardial contact potentials were seen during the catheterization of the infundibulum of the right ventricle in almost all our patients (Fig. 6), and contact of the electrode with the endocardium of the "apical region" of most of the patients also produced contact potentials of the usual kind. In 7 patients, however, contact and pressure sufficient to damp out the pressure pulse failed to produce the characteristic contact changes in the intracardiac electrocardiogram or only produced a minimal change in the S-T segment (Fig. 7-9).

Localization of Biotome. When the metal tip of the Konno biotome was used as an exploring electrode the presence of a ventricular endocardial contact potential was found to be useful in establishing that the biotome was in position against the ventricular endocardium (Fig. 10).

FIG. 4.—Pressure pulses (*above*) and intracardiac electrocardiogram (IEG) (*below*) from the right atrium (RA), the right ventricle (RV), and the pulmonary artery (PA) of a patient with severe right ventricular endomyocardial fibrosis. The pressure tracings are similar, but the intracardiac electrocardiograms from the three sites are distinct and characteristic. The intracardiac P waves from the right atrium are huge, the last one measuring 3.5 mV. (IEG recorded at normal sensitivity: 1 cm. = 1 mV.)

DISCUSSION

The routine monitoring and recording of the intracardiac electrocardiogram is of great value in the cardiac catheterization of patients with endomyocardial fibrosis. Apart from the high voltage P waves so understandably recorded from the cavity

of the huge right atrium, the characteristic complexes recorded from the various parts of the right heart and pulmonary artery were similar to those recorded from patients with rheumatic and congenital heart disease. During withdrawal of the catheter the usual more or less abrupt transition

FIG. 5.—Intracardiac electrocardiogram (*above*) and pressure pulses (*below*) recorded during withdrawal of the catheter tip from the pulmonary artery to the right ventricle, and from the right ventricle to the right atrium, in 2 patients with severe endomyocardial fibrosis of the right ventricle. When the electrode crosses the pulmonary valve the QRS-T complexes become much larger; when it crosses the tricuspid valve large intra-atrial P waves appear. The pressure pulses remain similar.

FIG. 6.—Intracardiac electrocardiogram (IEG) and pressures recorded from the infundibulum of the right ventricle in 2 patients, showing contact potentials of normal type (a) without damping, (b) with complete damping, of the pressure pulse. As the catheter is withdrawn in (b) an undamped pressure pulse and a normal right ventricular intracardiac QRS-T complex return. (IEG sensitivity half normal in (a), one-fifth normal in (b).)

between one type of complex and another was found. These transitions marked the passage of the tip of the catheter from one chamber to another with a clarity that could not be achieved by fluoroscopy or by scrutinizing the form of the pressure pulse.

In severe right ventricular endomyocardial fibrosis the pressure pulses in the right ventricle and the right atrium are often identical. Usually there is a flattened systolic wave followed by a “dip and plateau”, but sometimes the anatomical distortion

FIG. 7.—Intracardiac electrocardiogram (IEG) (*above*) and pressures (*below*), recorded during the withdrawal of the electrode from contact against the endocardium of the apical region of the right ventricle in a patient with right ventricular endomyocardial fibrosis. The first four pressure pulses are severely damped, the last two, though abnormal, are undamped. All the intracardiac QRS-T complexes resemble those normally seen only from a position free in the cavity of the right ventricle. Compare with Fig. 6.

FIG. 8.—Withdrawal of the catheter tip from contact against the apical region of the right ventricle in a patient with right ventricular endomyocardial fibrosis. Though at first the pressure pulse is severely damped, the electrocardiogram is unchanged throughout, and shows only slight S-T elevation. (IEG sensitivity half normal.) Compare with Fig. 6.

is such that the pressure pulse from both the atrium and the ventricle has an *a* and *v* wave with identical timings in each chamber. Fig. 11 shows an example of this "atrialization" of the right ventricle, where the intracardiac electrocardiogram gives the only indication that the identical pressure pulses are from two different cavities. There is an analogy here with Ebstein's anomaly of the tricuspid valve, where the intracardiac electrocardiogram can localize the catheter tip in the true atrium or in the atrialized part of the right ventricle (Lowe *et al.*, 1968).

When there is atrial fibrillation or atrial flutter the transition in the curves as the catheter tip crosses the tricuspid valve is strikingly marked by the abrupt appearance of fibrillation or flutter waves.

There is still much to be learned about the abnormal haemodynamics in endomyocardial fibrosis. In any haemodynamic work it is essential to know with complete certainty from which cardiac cavity the pressure recordings are being made. As a result of our study we believe that in many cases of severe endomyocardial fibrosis this certainty can only be attained by the use of the intracardiac electrocardiogram. In our laboratory right heart catheterization in endomyocardial fibrosis is now routinely carried out with an electrode catheter (Somers *et al.*, 1968a).

Everyone with a practical experience of intracardiac electrocardiography is familiar with the characteristic changes in the records that result from contact of the electrode with the endocardium of the atrium or the ventricle. In its fully developed form, when there is a certain amount of pressure against the endocardium, the QRS-T complex resembles the kind of monophasic action potential that can be produced experimentally by injury. Hellerstein and Katz (1948) suggested that these complexes could result from either an injury current of rest or else from focal intraventricular block. They argued that when the monophasic action potential arose from a depressed baseline and reached its highest point at the original baseline it was probably the result of a resting injury current. When there was no depression of the baseline the monophasic action potential was probably the result of localized focal intraventricular block. Where the baseline was depressed and the monophasic action potential rose above the original baseline, a combination of both these effects would be likely. During cardiac catheterization this latter situation is the usual one. In their experiments on animals, Hellerstein and Katz found that firm pressure of the electrode against the endocardium for 5 seconds produced changes that disappeared completely

FIG. 9.—Intracardiac electrocardiogram (*above*) and pressure pulses (*below*) recorded from the right ventricle of a patient with right ventricular endomyocardial fibrosis. In (a) the catheter tip is in contact with the endocardium: the pressure pulse is severely damped and there is only a minimal S-T elevation and a prolonged ST-T segment in the intracardiac electrocardiogram. In (b) the catheter tip is free in the cavity of the right ventricle. (IEG sensitivity one-fifth normal.)

FIG. 10.—X-ray taken during cardiac catheterization to show the Konno endocardial biotome in the right ventricle.

FIG. 11.—Intracardiac electrocardiogram (*above*) and pressure pulses (*below*) recorded from the right ventricle and from the right atrium of a patient with severe right ventricular endomyocardial fibrosis. The pressure pulses from both chambers are of atrial form (*av*), but the intracardiac electrocardiograms from each chamber are distinct and characteristic.

immediately the pressure was removed. If the pressure was maintained for up to 4 minutes the S-T elevations in the intracardiac electrocardiogram remained longer, but only when this pressure was repeatedly exerted on the same spot was there any evidence at necropsy of endocardial injury. Many subsequent workers have confirmed that even when there is S-T elevation measuring several millivolts in the intracardiac electrocardiogram there is no change in the S-T segment in records from surface leads. Levine *et al.* (1949) stated that if this large S-T deviation represented a current of injury its extent must be localized. Kossmann *et al.* (1950) preferred the term "demarcation potential", and considered that it was the result of a partial or complete block of the depolarization process near the pressure-making electrode. Watson (1964) considered that these S-T elevations should be referred to as contact, rather than injury, currents.

In severe right ventricular endomyocardial fibrosis the contracted "apical region", lined with its thick fibrous tissue, often lies high up and just to the left of the spine in the postero-anterior fluoroscopic view (Fig. 3). During cardiac catheterization from the groin, the tip of the cardiac catheter was often occluded by the endocardium of this scarred region so that the pressure pulse was

completely damped. The electrode on the catheter formed a ring at the extreme tip, so when the pressure pulse was completely damped it was highly likely that the ring electrode and the endocardium were firmly in contact. When the pressure pulse is completely damped in this way during the cardiac catheterization of patients with rheumatic or congenital heart disease, the pressure of the electrode usually produces well-marked contact potentials in the intracardiac electrocardiogram. In 7 of our patients the contact potentials recorded in this situation were almost absent, or minimal. Thus, about one-third of the patients with severe endomyocardial fibrosis were considered to show abnormally slight contact potentials. When the catheter was occluded by the endocardium of the infundibulum, however, normal contact potentials were recorded. This is in keeping with the fact that in endomyocardial fibrosis the thick endocardial scarring is confined to the apical region and tapers towards the tricuspid ring, the infundibulum being free of disease.

It is impossible to measure the amount of pressure exerted on the catheter during catheterization, and full control observations on patients without a greatly thickened, tough endocardium have not been made. Mural thrombosis is common in endomyocardial fibrosis, and the pressure pulse could perhaps occasionally have been completely damped by thrombus without the electrode actually touching the endocardium. For these reasons, though we think that the contact potentials recorded from the thickened endocardium were abnormal, we do not suggest that the finding of these abnormal contact potentials would form the basis of a satisfactory diagnostic test.

All but one of our patients were judged to have severe endomyocardial fibrosis of the right ventricle. It was therefore, perhaps, surprising that the QRS complexes recorded from the right ventricular cavity should have been essentially normal. Not only is there a great thickening of the endocardium in severe endomyocardial fibrosis, but the sub-endocardial layers of the myocardium may be abnormal too. It has been suggested (Prinzmetal *et al.*, 1954) that the subendocardial layers of the normal myocardium play no part in the genesis of the electrocardiogram. Our finding of normal QRS complexes from the cavity of the right ventricle in patients with severe right ventricular endomyocardial fibrosis perhaps lends some support to this view.

SUMMARY

The intracardiac electrocardiogram, recorded by a ring-electrode at the extreme tip of the catheter,

has been studied during right-sided cardiac catheterization in 21 patients with proved endomyocardial fibrosis. In right ventricular endomyocardial fibrosis the record is essentially similar to that found in patients with rheumatic and congenital heart disease, except that contact potentials recorded from areas of severe scarring often seem to be modified. During haemodynamic studies of patients with severe right ventricular endomyocardial fibrosis, the changes in pattern of the intracardiac electrocardiogram often offered the only reliable indication of the position of the catheter tip.

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