Ventricular arrhythmias

A guide to their localisation

P M HOLT,* C SMALLPEICE,‡ P B DEVERALL,† A K YATES,† P V L CURRY*

From the Departments of *Cardiology and †Cardiothoracic Surgery, Guy's Hospital, London

SUMMARY An electrocardiographic atlas of ventricular tachycardias was produced by pacing 27 epicardial sections of the heart and the mitral papillary muscles to simulate focal ventricular arrhythmias and simultaneously recording their 12 lead electrocardiographic appearances. One hundred and twenty nine patients undergoing cardiac surgery were studied. In five patients all 27 epicardial sites were paced at operation and in 124 individual sections were paced postoperatively with temporary epicardial wires and the electrocardiograms analysed in terms of frontal and horizontal plane QRS axis, maximum limb lead QRS amplitude, and QRS duration. Each ventricular region paced produced a distinctive 12 lead electrocardiographic pattern.

Simulated right ventricular arrhythmias had either inferior frontal plane QRS axes (from the anterobasal region) or superior frontal plane QRS axes (from the apical and posterior right ventricular sections). Horizontal plane QRS axes were directed leftwards, with some posterior shift in the anteroapical regions. Simulated arrhythmias from the base of the left ventricle (anteriorly and laterally) had inferior frontal plane QRS axes and anterorightward horizontal plane QRS axes. Left ventricular arrhythmias with a superior frontal plane QRS axis were readily distinguished by their horizontal plane QRS axes: posterorightwards from the anterior and anterorightwards from the posterior left ventricular sections. Standard errors of the paced QRS axes for the various epicardial sections paced postoperatively ranged from 3.0° to 6.0° using the frontal plane axis. The electrocardiogram was most accurate in localising ventricular arrhythmias from the anterior left ventricle and least accurate for those arising from the inferior right ventricle. The appearance of the paced electrocardiograms was slightly modified by underlying disease such as myocardial infarction and left ventricular hypertrophy.

This atlas may be useful in comparing the localisation of ventricular tachycardia with the site of underlying cardiac disease and may facilitate mapping in patients with refractory ventricular tachycardia requiring ablation (either surgical or by high energy impulses).

Deductive electrocardiography allows approximate localisation of ventricular arrhythmias using the principles of axis analysis with its inherent limitations. An alternative method of localising ventricular tachycardia from the conventional 12 lead electrocardiographic appearances is by a direct comparison with arrhythmias arising from known sites—that is, from an atlas of ventricular arrhythmias.

An atlas can be produced by pacing several selected

parts of the heart to mimic ventricular tachycardia. The surface electrocardiographic appearances resulting from ventricular stimulation at different sites were first investigated by Kraus and Nicolai in 1907.¹ Shortly after this, Lewis noted the similarity between the electrocardiographic appearances produced by stimulating the right and left ventricles and those obtained from cutting the left or right bundle branches respectively.²⁻⁴ Oppenheimer and Stewart in 1926,⁵ Barker *et al* in 1930,⁶ and Abramson *et al* in 1937⁷ furthered the knowledge in this field, but it was with the advent of more sophisticated electrophysiological techniques that later workers showed that pacing the site of earliest ventricular activation

Requests for reprints to Dr P Holt, Cardiac Department, Guy's Hospital, St Thomas Street, London SE1 9RT.

‡Present address: Queen Elizabeth Hospital, Edgbaston, Birmingham.

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Fig. 1 Diagram of the anterior and posterior ventricular epicardium divided into 27 sections, 12 anteriorly and 15 posteriorly in (a) the sternocostal aspect and (b) the diaphragmatic aspect. RCA, right coronary artery; LCA, left coronary artery; Cx, circumflex; LAD, left anterior descending; SA, sinoatrial; LA, left atrium; RV, right ventricle; LV, left ventricle.

reproduces very closely the QRS morphology of the spontaneous tachycardia under investigation.⁸⁻¹⁰ In this study we used this technique of pace mapping, which was adopted initially to localise ventricular tachycardia during surgery,¹¹ to produce an electrocardiographic atlas of ventricular arrhythmias arising from certain surfaces of the heart but not as yet of those arising from the His-Purkinje trunk and its main branches.

Patients and methods

The electrocardiographic appearances of ventricular arrhythmias were recorded in two separate studies.

VENTRICULAR ARRHYTHMIAS FROM DIFFERENT SITES IN THE SAME PATIENT (OPEN CHEST)

Five patients undergoing cardiac surgery for the man-

agement of their valvar or ischaemic heart disease were included in this study. None of the patients with ischaemic heart disease had had a myocardial infarction. At open heart surgery, the ventricular epicardium was considered to consist of 27 sections (Fig. 1), 12 anterior sections (eight right ventricular and four left ventricular) and 15 posterior sections (seven right ventricular and eight left ventricular). The heart was paced from each section using a small bipolar electrode (Plastimed, Paris) with 4 mm between poles. This electrode could be attached to the appropriate section and the heart returned to its normal position within the thorax during the brief period of ventricular pacing mostly at heart rates between 100 and 150 beats/min, which were constant for each patient. Pacing was performed at 1.0 V above threshold values, usually 2.0–3.0 V. During ventricular pacing the electrocardiogram was recorded to document the characteristic appearances of arrhythmias arising from each section, as represented in the six standard limb leads and three atypical chest leads, V4R, V5, and V6 (all in the open thorax). The electrocardiograms were recorded during inspiration and expiration.

VENTRICULAR ARRHYTHMIAS FROM THE SAME SITE IN DIFFERENT PATIENTS (CLOSED CHEST)

In 124 adult patients undergoing heart surgery a temporary pacing wire was inserted to produce focal activation of one of the 27 epicardial sections (Fig. 1). In many patients the surgeons found sections 10 and 11 difficult to distinguish during implantation of the temporary epicardial wires, and these regions were considered as one. The same problem occurred with sections 15 and 16 on the posterior right ventricle, which were also regarded as one.

The temporary pacing wires used have a small exposed section of wire approximately 2–3 cm distal to the introducing needle. The pacing wire was inserted via the ventricular epicardium as a single stitch, until the exposed section was buried immediately below the epicardium, leaving the two ends of the wire protruding. This gave a well localised pacing stimulus. The wire was sutured on to the epicardium so that it did not become detached in the early postoperative period yet was easily removed at the end of the pacing study. The leads of the temporary epicardial wires were brought out by a separate incision and the chest closed in the usual manner. Five to seven days postoperatively the wires were paced using a Devices box at a rate of 10 beats/min above the patient's resting rate and a voltage 1.0 V above the threshold voltage. A 12 lead electrocardiogram was recorded during pacing with the patient supine and during quiet respiration.

In five patients this technique was also used to define the 12 lead electrocardiographic appearances of

Fig. 2 Electrocardiographic appearances (eight leads) obtained by pacing the anterior ventricular epicardial sections in one patient directly during operation. The arrows summarise the limb lead QRS complexes: their direction represents the limb lead QRS axis, their length indicates the mean maximum QRS amplitude, and their width the QRS duration. RV, right ventricle, LV, left ventricle.

Table 1 Results of electrocardiograms produced on pacing the anterior and posterior ventricles at operation

Section	No of patients	Limb lead QRS axis			Maximum limb lead QRS amplitude (mV)			QRS duration (s)
		Mean	Range	SE	Mean	Range	SE	
Anterior ventricles								
Right:								
1	5	+15°	0° to +30°	4.74°	0.96	0.80-1.0	0.02	0.12
2	5	+99°	+90° to +105°	3.67°	1.08	1.0-1.2	0.04	0.12
4	5	-6°	0° to -15°	3.67°	0.97	0.8-1.1	0.05	0.12
5	5	+63°	+45° to +75°	5.6°	0.82	0.7-0.9	0.04	0.12
7	5	-42°	-30° to -60°	5.6°	0.94	0.7-1.1	0.05	0.12
8	5	-6°	0° to -15°	3.67°	0.56	0.4-0.8	0.07	0.10
10/11	5	-69°	-60° to -75°	3.67°	0.96	0.8-1.1	0.04	0.12
Left:								
3	5	+129°	+120° to +135°	3.67°	1.07	0.8-1.3	0.09	0.12
6	5	+142°	+135° to +150°	3.67°	1.04	0.8-1.3	0.09	0.12
9	5	-171°	-150° to -180°	6.0°	0.60	0.5-0.7	0.03	0.10
12	5	-139°	-120° to -150°	4.74°	0.80	0.7-1.0	0.05	0.12
Posterior ventricles								
Right:								
15/16	5	-9°	0° to -30°	6.0°	0.94	0.8-1.0	0.02	0.12
19	5	-33°	-15° to -45°	5.6°	0.69	0.5-0.8	0.03	0.12
20	5	-30°	-15° to -45°	4.74°	0.78	0.7-0.9	0.04	0.12
23	5	-63°	-60° to -75°	3.0°	0.9	0.7-1.0	0.03	0.12
24	5	-54°	-45° to -60°	3.67°	0.76	0.6-0.9	0.05	0.12
27	5	-93°	-75° to -105°	5.6°	1.08	1.0-1.2	0.04	0.12
Left:								
13	5	+159°	+150° to +165°	3.67°	0.96	0.8-1.1	0.05	0.12
14	5	-45°	-30° to -60°	4.74°	0.88	0.8-1.0	0.04	0.12
17	5	-147°	-135° to -165°	5.6°	0.84	0.6-1.0	0.06	0.12
18	5	-57°	-45° to -60°	3.0°	1.08	0.9-1.2	0.06	0.12
21	5	-141°	-135° to -150°	3.67°	0.88	0.7-1.0	0.05	0.12
22	5	-78°	-60° to -90°	5.6°	1.12	1.0-1.2	0.04	0.11
25/26	5	-108°	-90° to -120°	5.6°	1.08	0.9-1.2	0.04	0.12

ventricular arrhythmias arising from the mitral valve papillary muscles. The temporary pacing electrode was inserted from the endocardial aspect into the papillary muscle and the lead drawn through to the epicardial surface until the bared active tail of the electrode was left in the papillary muscle below the surface in its muscular body. The electrodes were fixed by a small epicardial suture to prevent migration of the electrode tip.

The temporary electrodes were removed after one week by simple traction without complication, and as such was a simple modification of the routine practice of leaving a temporary epicardial electrode after open heart surgery to overcome any episodes of bradycardia that may complicate the early postoperative period.

Patients

The electrocardiographic appearances of between four and seven patients were recorded per site (mean 4.98 patients/site). The 124 patients included in this study were aged between 16 and 64 years; 68 were men and 56 women. Eighty nine (71.7%) had ischaemic heart disease, 38 of whom had had previous myocardial infarction, and 27 (21.7%) valvar heart disease. Of the latter group, 10 underwent aortic valve replacement, 10 mitral valve replacement, and seven replacement of both valves. A further four patients required surgery

for valvar and coronary artery disease, two had repair of secundum atrial septal defects, one repair of a ventricular septal defect, and one section of a left side Kent pathway (with an otherwise normal heart).

The five patients in whom pacing of the mitral valve papillary muscles was performed were aged 50-66 years; three were men and two women undergoing mitral valve replacement, three for predominant mitral regurgitation and two for mixed mitral valve disease.

No patient had spontaneous ventricular tachycardia or a left ventricular aneurysm. None was undergoing repeat coronary artery bypass grafting.

ASSESSMENT OF ELECTROCARDIOGRAPHIC APPEARANCES

Paced QRS complexes from ventricular activation were described in terms of the following features: (a) frontal plane QRS axis; (b) horizontal plane QRS axis; (c) maximum limb lead QRS amplitude; and (d) QRS duration. The frontal plane axis of the main QRS deflection was calculated conventionally by reference to all six limb leads. A final correction of +15° or -15° was made if the original calculation was based on a lead whose deflection was not completely equiphasic. The horizontal plane QRS axis was similarly calculated by reference to all precordial leads and

Fig. 3 Electrocardiographic appearances (eight leads) obtained by pacing the posterior ventricular epicardial sections, again in one representative patient, directly during surgery. The arrows summarise the limb lead QRS appearances as in Fig. 2.

was at right angles to that precordial lead showing the most equiphasic deflection. Each feature was influenced by the site of ventricular activation and is represented diagrammatically by an arrow that has direction, length, and width (QRS axis, amplitude, and duration respectively) in subsequent Figures.

Results

OPEN CHEST STUDY

Anterior ventricles—Figure 2 shows the representative electrocardiographic appearances in the six limb leads and two precordial leads produced by direct epicardial stimulation of the anterior ventricles in five patients. Pacing the right ventricular outflow tract produced a limb lead QRS axis of $+105^\circ$. Moving the site of stimulation closer to the right ventricular apex produced an anticlockwise swing in the limb lead QRS axis, such that paced arrhythmias from the apex had a frontal plane QRS axis of -75° . Changes in QRS amplitude also occurred, amplitude being greatest when the ventricles were activated from their poles (apex and base) and smallest in sections 8 and 9 (Fig. 1) adjacent to the interventricular septum, an electrocardiographic low amplitude centre of the ventricles. Pacing the left ventricular epicardial sections immediately adjacent to the left anterior descending coronary artery changed the limb lead QRS axis from $+120^\circ$ at the base to -120° at the apex, a clockwise swing of 120° . Again a reduction then restoration of the QRS amplitude occurred. Table 1 shows the details of the limb lead QRS axes in all five patients. The QRS duration of simulated ventricular arrhythmias was narrowest (mean 0.10 s, sections 8 and 9) near the septum, especially in the mid zones anteriorly, and widest laterally.

Posterior ventricles—Figure 3 shows representative electrocardiograms produced by epicardial stimulation of the posterior ventricles. Pacing the posterior right ventricular base produced a limb lead QRS axis of 0° . Moving the pacing site nearer to the posterior right ventricular apex via the free wall again produced a stepwise anticlockwise swing in axis to -90° . A similar trend was produced on pacing those posterior left ventricular sections adjacent to the interventricular septum. Pacing the remaining left ventricular sections altered the limb lead QRS axis from $+165^\circ$ at the base to -120° at the apex (Table 1). There was a slight tendency to a reduction in QRS amplitude at the mid sections of the posterior ventricles, but no definitive picture emerged of an electrocardiographic low amplitude centre as was apparent anteriorly. The duration of paced QRS complexes of simulated ventricular arrhythmias was 0.11–0.12 s. Increasing the pacing rate or pacing energy produced no appreciable changes in the QRS morphology.

Changes in ventilation during pacing produced slight

alteration in the limb lead QRS morphology only at maximum inspiration and expiration in two patients. Deeper S waves were present in leads I and aVL on maximum inspiration than on expiration. Therefore pacing as described in the methodology for open chest patients was performed only during normal respiration. The three precordial leads V4R, V5, and V6 recorded with the chest open were only sufficient to determine the bundle branch block pattern and added little further localising information.

CLOSED CHEST STUDY

Table 2 summarises the appearances of the limb lead QRS complexes produced on pacing the ventricular epicardial sections in the 124 patients in the second study.

Anterior ventricles—Pacing the right ventricular outflow tract produced a mean limb lead QRS axis of $+75^\circ$ changing to a mean value of $+51^\circ$, -12° , and -66° as the right ventricle was paced stepwise from outflow to apex in a line parallel to the interventricular septum. A similar anticlockwise swing was present in the remaining anterolateral right ventricular sections. Pacing the anterior left ventricular apex produced a mean limb lead QRS axis of -153° with a mean maximum limb lead QRS amplitude of 0.7 mV. As the pacing site was moved upwards the anticlockwise swing of the QRS axis continued, ending in a mean limb lead QRS axis of $+123^\circ$ at the left ventricular outflow tract, encompassing an arc of almost 360° . Figure 4 shows electrocardiograms from individual patients, and the tracings are representative of the results obtained on pacing the same section in different patients. Complexes of minimum amplitude and duration were encountered in positions 5, 8 (right ventricle), and 9 (left ventricle), again giving rise to an electrocardiographic "centre" to the map. Crossing the interventricular septum from right to left was associated with the development of a small R wave in lead V1 equivalent in conventional terminology to a transition from left to right bundle branch block appearances. The differences in limb lead axes between Figs. 2 (first study) and 4 (second study) are probably due to inconsistencies in electrode placement. Figure 5 summarises the precordial lead QRS axes produced on pacing the anterior ventricular sections.

Posterior ventricles—The limb and precordial lead complexes obtained on pacing the posterior ventricular sections can be analysed in the same way and their appearances are shown in Table 2 and Fig. 6. Pacing the posterior right ventricular apex produced a mean limb lead QRS axis of -81° , which swung anticlockwise to mean values of -63° , -42° , and -7.5° as the pacing site was moved stepwise up to the base of the heart adjacent to the posterior interventricular sep-

Table 2 Results of electrocardiograms produced on pacing anterior and posterior ventricles postoperatively

Section	No of patients	Limb lead QRS axis			Maximum limb lead QRS amplitude (mV)			QRS duration (s)
		Mean	Range	SE	Mean	Range	SE	
Anterior ventricles								
Right:								
1	5	+9°	0° to +15°	3.67°	0.88	0.7-1.2	0.08	0.12
2	5	+75°	+60° to +90°	4.74°	1.28	1.1-1.5	0.07	0.12
4	5	-21°	0° to -30°	6.0°	0.84	0.7-1.1	0.09	0.12
5	5	+51°	+45° to +60°	3.67°	0.62	0.5-0.7	0.05	0.10
7	5	-39°	-30° to -45°	3.67°	1.62	1.1-2.0	0.27	0.12
8	5	-12°	0° to -15°	3.0°	0.65	0.4-0.9	0.10	0.10
10/11	5	-66°	-60° to -75°	3.67°	1.4	1.0-1.7	0.14	0.12
Left:								
3	5	+123°	+120° to +135°	3.0°	1.66	1.3-1.9	0.10	0.12
6	5	+153°	+150° to +165°	3.0°	1.0	0.8-1.2	0.07	0.12
9	5	-177°	-165° to -180°	3.0°	0.5	0.3-0.7	0.04	0.10
12	5	-153°	-150° to -165°	3.0°	0.74	0.5-0.9	0.05	0.12
Posterior ventricles								
Right:								
15/16	4	-7.5°	0° to -15°	4.3°	1.07	0.7-1.3	0.13	0.12
19	5	-42°	-30° to -45°	3.0°	1.32	0.8-1.9	0.15	0.12
20	5	-36°	-30° to -45°	3.67°	1.58	0.8-2.4	0.32	0.12
23	5	-63°	-60° to -75°	3.0°	1.5	0.8-2.0	0.23	0.12
24	5	-51°	-45° to -60°	3.67°	1.9	1.7-2.5	0.15	0.12
27	7	-81°	-75° to -90°	3.0°	2.2	1.5-3.0	0.33	0.12
Left:								
13	4	+124°	+120° to +135°	3.75°	0.95	0.8-1.1	0.06	0.12
14	4	-49°	-45° to -60°	3.75°	1.55	1.0-2.0	0.29	0.12
17	5	-144°	-135° to -150°	3.67°	0.82	0.5-1.4	0.16	0.12
18	5	-69°	-60° to -75°	3.67°	1.08	0.8-1.7	0.16	0.12
21	5	-129°	-120° to -135°	3.67°	0.94	0.7-1.4	0.12	0.12
22	5	-81°	-75° to -90°	3.67°	1.58	1.3-2.0	0.14	1.12
25	5	-117°	-105° to -120°	3.0°	1.34	0.8-1.9	0.2	0.12
26	5	-93°	-90° to -105°	3.0°	1.68	1.4-2.3	0.16	0.12

tum. Similar frontal plane axes were seen on pacing the left ventricular sections adjacent to the interventricular septum. The remaining left ventricular sections produced a sort of "mirror image" in which the mean limb lead QRS axis swung anticlockwise stepwise from -117° to +124° from apex to atrioventricular groove. A region of low voltage complexes was not found posteriorly, which contrasts with the findings on pace mapping the anterior ventricles. Figure 7 shows the horizontal plane QRS axes. Posterior left ventricular sites could be distinguished from anterior left ventricular sites by reference to the horizontal plane axes.

The appearances obtained in this study using the temporary epicardial wires for postoperative pacing produced results very similar to those of the first study.

Mitral valve papillary muscles

Pacing the anterior papillary muscle of the mitral valve produced a mean limb lead QRS axis of -170° and maximum limb lead QRS amplitude of 1.17 mV, with a mean duration of 0.127 s. The corresponding values produced by pacing the posterior papillary muscle of the mitral valve were -90°, 1.05 mV, and 0.12 s respectively (Table 3, Figs. 8 and 9).

Variation between patients

Pacing the same site in different patients often produced similar 12 lead electrocardiographic appearances despite the wide differences in their physical features and those of their resting 12 lead electrocardiogram in sinus rhythm.

Effects of pre-existing cardiac disorders

Myocardial infarction—Thirty eight of 124 patients in whom temporary epicardial wires were paced postoperatively had previous myocardial infarction. Sixteen patients had had previous anterior myocardial infarction, 20 previous inferior infarction, and two previous posterior myocardial infarction. The presence of inferior myocardial infarction made no appreciable difference to the paced electrocardiograms. Previous anterior or posterior infarctions did not appreciably affect the limb lead appearances of the paced electrocardiograms but principally altered those of the precordial leads. Previous anterior myocardial infarction caused a diminution of the initial R wave amplitude in the precordial leads of the paced complexes, whereas a previous posterior myocardial infarction increased the height of the paced precordial lead R wave. The loss of initial R waves can be seen in Fig. 6, section 19, where the electrocardiogram was recorded

Fig. 4 Twelve lead electrocardiographic appearances for the anterior ventricular sections recorded by pacing temporary epicardial wires postoperatively. Each electrocardiogram and the values above are taken from one patient, and are examples of the values recorded in that group in whom the same section was paced. The arrows summarise the limb lead QRS appearances as in Fig. 2.

Fig. 5 Diagram showing by means of arcs the range of horizontal lead axes encountered in patients with temporary epicardial wires inserted in the anterior sections. Stippled areas represent precordial lead axis during sinus rhythm; hatched areas represent precordial lead axis during pacing. The radius of the arcs represents the mean maximum amplitude of the precordial lead QRS complexes. RV, right ventricle; IVS, interventricular septum; LV, left ventricle.

in a patient with previous anterior infarct (see adjacent section 20).

Bundle branch block—Right bundle branch block occurred infrequently. Only six patients had right bundle branch block on their resting electrocardiogram: five had temporary epicardial wires placed in the posterior right ventricle and one in the posterior left ventricle. It did not affect the limb lead or precordial lead axis or the duration of the paced ventricular complexes. Six patients had pre-existing left bundle branch block. Three had temporary epicardial wires positioned in the anterior right ventricle, one in the posterior right ventricle, and two in the left ventricle, one anteriorly, the other posteriorly. The paced electrocardiograms appeared uninfluenced by the presence of left bundle branch block on the resting electrocardiogram.

Left ventricular hypertrophy—Ten of 124 patients included in the second study had the voltage criteria of left ventricular hypertrophy on their resting electrocardiograms. Five of these had epicardial wires

inserted into the anterior right ventricle and three into the posterior right ventricle. Two patients had posterior left ventricular pacing wires. Left ventricular hypertrophy did not affect the limb lead or precordial lead QRS axes or the QRS duration of the paced complexes. The QRS amplitude of the paced complexes was, however, larger in those with left ventricular hypertrophy.

Discussion

Using pace mapping we have produced a working atlas of ventricular arrhythmias, an application of pace mapping that was envisaged in 1979.¹¹ The 12 lead electrocardiographic appearances from ventricular activation vary according to the site paced. Pacing the same site in different patients gives rise to similar electrocardiographic appearances. It is now possible not only to identify the ventricle of origin but also to localise ventricular arrhythmias to a region of that ventricle using the conventional 12 lead electrocar-

Fig. 6 Electrocardiograms from individual patients in whom the posterior ventricular sections were paced representing the results obtained when that same section is paced in different patients. The paced tracing of section 19 has small initial R waves in V1-V4 and was taken from a patient with a previous anterior myocardial infarction (see text). RV, right ventricle; LV, left ventricle.

Fig. 7 Diagrammatic summary of precordial lead axes obtained in patients with temporary epicardial wires inserted in the posterior ventricular sections. Stippled areas represent precordial lead axis during sinus rhythm; hatched areas represent precordial lead axis during pacing; broken arrow denotes patients with bundle branch block on the resting electrocardiogram. RV, right ventricle, LV, left ventricle, IVS, interventricular septum.

Fig. 8 Electrocardiograms on pacing the (a) anterior and (b) posterior mitral valve papillary muscles recorded from individual patients but representative of the results obtained from the whole group of patients in whom the mitral valve papillary muscles were paced.

Fig. 9 Diagram showing the precordial lead QRS axes produced on pacing the anterior papillary muscle (top) and posterior papillary muscle of the mitral valve.

diagram. Since the mechanism¹² of a ventricular arrhythmia has no bearing on its final electrocardiographic appearances the atlas can be applied to the localisation of focal, triggered, and re-entry types. The most important of the 12 electrocardiographic leads in identifying the segment from which the tachycardia arises in the long axis of the heart is lead II. This is shown in Figs. 2 and 4 by the transition of the main QRS deflection from positivity to negativity in this lead as the site of simulated ventricular tachycardia moves from the base to the apex of the heart respectively.

The electrocardiographic effects produced by a transition of simulated ventricular tachycardia loci across the ventricles from right to left are best seen in the precordial leads and also in lead I, in which the paced QRS deflection becomes more negative as the site of stimulation is moved from right to left. The transition of the paced QRS in lead I from positivity to negativity on stimulating the posterior ventricles does not occur at the anatomical landmark of the interventricular septum but is shifted somewhat towards the left ventricle. This confirms Abramson *et al*'s findings.⁷

Our results show that ventricular arrhythmias with an inferior frontal plane QRS axis and a posterior leftward horizontal plane QRS axis originate from the anterobasal region of the right ventricle. Arrhythmias with a superior frontal plane QRS axis need careful scrutiny of their precordial lead appearances. Those with a posteroleftward horizontal plane QRS axis originate from the anterior and apical regions of the right ventricle and those with a leftward horizontal

QRS axis from the posterior right ventricle. Attention to QRS duration further improves discrimination.

Certain pairs of right ventricular sections (4 and 7; 10/11 and 27; 19 and 20), however, produced paced electrocardiograms with very similar appearances. Therefore arrhythmias from these sites can be reliably resolved into only three and not six sections from their surface 12 lead electrocardiograms. In addition, the electrocardiogram from section 19 (Fig. 6) is very similar to that for section 7 (Fig. 4) and was obtained from a patient with previous anterior myocardial infarction. The resultant loss of R waves in the paced precordial lead complexes (V1-4) shifts the horizontal axis, in this example from section 19, into the same range as that obtained by pacing sections from the anterior ventricles.

Simulated arrhythmias from the base of the left ventricle anteriorly and laterally have an inferior frontal plane QRS axis and an anterorightward horizontal plane QRS axis. Left ventricular arrhythmias with a superior frontal plane QRS axis are readily distinguished by analysing their precordial leads; those with a posterorightward horizontal QRS axis originate from the anterior left ventricle and those with an anterorightward horizontal QRS axis from the posterior left ventricular sections. Arrhythmias from only two left ventricular sections (3 and 13) show appreciable overlap of their electrocardiographic appearances and are therefore not readily distinguished by the surface 12 lead electrocardiogram.

In this study we analysed the results of pacing small numbers of patients from a large number of ventricular sites in an attempt to give a detailed electrocardiographic atlas. Pacing more patients from fewer sites would have allowed better evaluation of the reproducibility of the electrocardiographic morphology and possibly the effects of underlying disease. Comparatively small changes in the site of pacing can, however, produce electrocardiographic changes, especially in the left ventricle. Therefore pacing fewer larger sections in the same patients would considerably diminish the electrocardiographic detail of the atlas.

Josephson *et al* reported the unusual presence of a left bundle branch block QRS morphology in the surface electrocardiogram during ventricular tachycardia that, according to the site of earliest recorded activa-

Table 3 Results of electrocardiograms produced by pacing the mitral valve papillary muscles postoperatively

Papillary muscle	Frontal plane QRS axis	Limb lead QRS amplitude (mV)	Limb lead QRS duration (mV)
Anterior	-165°	0.7	0.14
	-165°	1.4	0.12
	-180°	1.4	0.12
	-170°	1.17	0.13
Mean	-90°	1.0	0.12
Posterior	-90°	1.1	0.12
	-90°	1.05	0.12
Mean	-90°		

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tion, was arising from the left ventricle.^{13 14} Virtually all these patients, however, had left ventricular aneurysms associated with the arrhythmia. The development of a ventricular aneurysm may appreciably disrupt the spread of the depolarisation front and cause variable activation patterns in the same tachycardia. Great caution must, therefore, be exercised in interpreting the surface electrocardiograms of those patients with ventricular tachycardia and ventricular aneurysm. Further experience is needed with pace mapping studies in such cases. None of the patients included in our study had a left ventricular aneurysm.

Following our preliminary communication¹¹ Waxman and Josephson and Josephson *et al* also studied electrocardiographic patterns produced by pacing different sites on the left ventricular endocardium and compared them with operative findings.^{15 16} They studied 14 patients using percutaneous electrodes, but not all of the selected mapping sites were paced in each patient. Localisation of the pacing tip was judged by multiplane fluoroscopy with its inherent inaccuracies.

Our observations from epicardial pace mapping studies corroborate their findings during endocardial studies of large R waves in the precordial leads on pacing the posterior left ventricular sites and small R waves and deep S waves in leads V1–6 on pacing the anterior left ventricular sections. The same trend of predominantly superiorly directed limb lead QRS axes from apical and posterior left ventricular sites was also confirmed. Because it was impossible to pace all possible sites, particularly the endocardial surface and the septum, this atlas strictly refers to arrhythmias arising from the epicardial surface.

Our work and previous studies have shown that the site of origin of ventricular tachycardia may be inaccurately identified if attention is confined to the arrhythmia appearances in only a limited number of electrocardiographic leads.¹⁴ The so called left or right bundle branch block classification¹⁷ is insufficiently precise. The entire 12 lead electrocardiogram of a ventricular tachycardia should be scrutinised. Any factors that influence QRS axes (for example, previous myocardial infarction) should be taken into account.

This atlas of ventricular arrhythmias has clinical implications since it allows the site of the ventricular tachycardia to be compared with that of the underlying cardiac disease. For example, ventricular tachycardia complicating the mitral valve prolapse syndrome has been shown to arise from the mitral papillary muscles.¹⁸ More recently the relation between sites of ischaemia (localised on an electrocardiogram) and of ventricular arrhythmias has been studied clinically.^{19–22} Our atlas may be used to local-

ise ventricular arrhythmias to specific coronary artery territories in those with ischaemia.²³

Another application of the electrocardiographic atlas is to the preoperative localisation of a refractory ventricular tachycardia requiring ablation either surgically or by high energy impulses.^{24 25} The feasibility of the procedure in terms of accessibility of the tachycardia can be considered and the duration of the peroperative mapping procedure reduced by a prior awareness of the area of interest. By means of a standard simple investigation the 12 lead electrocardiographic ventricular arrhythmias can now be more accurately localised.

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