COPYRIGHT NOTICE

A PROSPECTIVE STUDY OF THE VALUE OF THE OESOPHAGEAL ELECTROCARDIOGRAM IN THE DIFFERENTIATION OF WIDE COMPLEX TACHYCARDIAS

by

RAJENDRAN MOODLEY

Submitted in partial fulfilment
of the requirements for the degree of

MASTER OF MEDICINE

in the
Department of Medicine
University of Natal
Durban

1990

ABSTRACT

The accurate differentiation of a ventricular from a supraventricular origin of a wide QRS tachycardia (QRS > 120 milliseconds) is an important clinical problem. Misdiagnosis of this arrhythmia can lead to institution of inappropriate drug therapy acutely with potentially catastrophic consequences. Various diagnostic aids have been used to obtain electrocardiographic potentials to aid in the differentiation. This report assesses the clinical usefulness of oesophageal electrocardiography in the differentiation of wide complex tachycardias and describes a simple, safe technique to obtain oesophageal electrocardiograms. Eighteen consecutive patients between the ages of 27 and 71 years who were haemodynamically stable were selected for this study. The technique was performed in the following manner: A temporary pacing catheter was lubricated and passed nasally and advanced with the patient being instructed to swallow. Adjustments in catheter depth were made as necessary to obtain an optimal recording on a standard electrocardiograph recorder. Satisfactory placement with minimal patient discomfort was achieved within 6.5 minutes (average 4.5 minutes) in all cases. High quality tracings were obtained in every instance. In the 18 patients with tachyarrhythmia, AV dissociation consistent with ventricular tachycardia was demonstrated in 11 instances; in the remainder the diagnosis was supraventricular tachycardia.

Of the 11 patients diagnosed as ventricular tachycardia, 9 were initially misdiagnosed as supraventricular tachycardia, whilst only 1 of 7 patients with supraventricular tachycardia was misdiagnosed.

This study has demonstrated that oesophageal electrocardiography is useful in the differentiation of wide complex tachycardias. The technique outlined in this report is simple

and offers the following advantages: the temporary pacing catheter is associated with minimal discomfort; the catheter allows easy manoeuverability within the oesophagus which allows proper depth to be easily obtained; the equipment used is routinely available. Therefore the technique offers a rapid, safe and simple method of obtaining an oesophageal electrocardiogram which is invaluable in the electrocardiographic differentiation of a wide complex tachycardia.

PREFACE

This study represents original work by the author and has not been submitted in any form to another University. Where use was made of the work of others it has been duly acknowledged in the text.

The research described in this dissertation was carried out in the Department of Medicine, University of Natal, under the supervision of Dr. Mohan Sewdarsen (Head of the Coronary Care Unit, R.K. Khan Hospital, Durban) and Dr. S. Vythilingum (Senior Consultant Cardiologist, Wentworth Hospital, Durban).

ACKNOWLEDGEMENTS

This dissertation was inspired by Professor Y.K. Seedat, Head of the Department of Medicine at the University of Natal, whose commitment to academic excellence is legion. His vision and encouragement is greatly appreciated.

The author wishes to express his sincere gratitude to the following individuals :

Dr. M. Sewdarsen for his perseverance and for executing the onerous task of supervising this thesis admirably.

Dr. S. Vythilingum for his valuable and expert guidance.

Professor A.S. Mitha, whose constructive criticism provided a stimulus for progressive thought.

The Superintendents, King Edward VIII and R.K. Khan Hospitals, Durban, for permission to conduct the study.

My wife Glynnis and daughter Pramodhini for patiently acquiescing to spend long hours alone.

This study was supported by a generous grant from the Research Fund of the Faculty of Medicine, University of Natal.

LIST OF CONTENTS

			Page
ABSTRACT			i
PREFACE			iii
ACKNOWLEDGEMENTS			iv
LIST OF CONTENTS			v
LIST OF FIGURES			vii
LIST OF TABLES			viii
LIST OF PLATES			ix
CHAPTER 1	**INTRODUCTION**		1
	1.1	Motivation for enquiry	1
	1.2	Objective and scope of enquiry	3
CHAPTER 2	**REVIEW OF THE LITERATURE**		5
	2.1	Mechanism of arrhythmias	5
	2.2	Criteria for differentiation	7
	2.2.1	The ventricular rate of the tachyarrhythmia	7
	2.2.2	The regularity	7
	2.2.3	Atrioventricular dissociation	7
	2.2.4	The nature of the QRS complexes	8
	2.2.5	The QRS axis	8
	2.2.6	The QRS configuration	8
	2.2.6.1	Concordance	8
	2.2.6.2	The right bundle branch block shaped QRS complexes	8
	2.2.6.3	The left bundle branch block shaped QRS complex	9
	2.2.7	Variation in QRS complex shapes	9
	2.2.8	Clinical criteria	9
	2.3	Consequences of misdiagnosis	9
	2.4	Attempts at improving diagnosis	11

CHAPTER 3	**PATIENTS AND METHODS**		13
	3.1	PATIENT SELECTION	13
	3.2	CONSENT	13
		3.2.1 *Information given to subject (prior to consent)*	13
	3.3	EQUIPMENT	13
		3.3.1 Oesophageal electrode	13
		3.3.2 Electrocardiographic recorder	16
	3.4	PROCEDURE	16
CHAPTER 4	**RESULTS**		17
	4.1	RECORDING DISTANCES	17
	4.2	TOLERANCE TO PROCEDURE	21
	4.3	ARTEFACTS	22
	4.4	TYPICAL P WAVE MORPHOLGY	23
	4.5	REVISION OF DIAGNOSIS	24
		4.5.1 Appraisal of patients with a final diagnosis of ventricular tachycardia	24
		4.5.2 Appraisal of patients with a final diagnosis of supraventricular tachycardia	24
	4.6	PROGRESS AFTER REVISED DIAGNOSIS	24
	4.7	ILLUSTRATIVE EXAMPLES	27
CHAPTER 5	**DISCUSSION OF RESULTS**		35
CHAPTER 6	**CONCLUSION AND RECOMMENDATIONS**		39
	6.1	CONCLUSION	39
	6.2	RECOMMENDATIONS	40
REFERENCES			41
APPENDIX 1	**CONSENT FORM**		44

LIST OF FIGURES

		Page
FIGURES 1-6	DIAGRAMMATIC REPRESENTATION OF THE SIX MECHANISMS OF ARRHYTHMIAS.	6
FIGURE 7	THE QRS MORPHOLOGY IN V_6 FAVOURING A SUPRAVENTRICULAR ORIGIN.	10
FIGURE 8	THE QRS MORPHOLOGY IN V_6 FAVOURING A VENTRICULAR ORIGIN.	10
FIGURE 9	THE QRS MORPHOLOGY IN V_1 FAVOURING A VENTRICULAR ORIGIN.	10
FIGURE 10	FREQUENCY HISTOGRAM OF OPTIMAL RECORDING DISTANCES	20
FIGURE 11	TYPICAL MORPHOLOGY OF WAVEFORMS RECORDED FROM OESOPHAGEAL ELECTRODE ABOVE THE ATRIAL LEVEL.	23
FIGURE 12	TYPICAL MORPHOLOGY OF WAVEFORMS RECORDED FROM OESOPHAGEAL ELECTRODE AT THE ATRIAL LEVEL.	23
FIGURE 13	TYPICAL MORPHOLOGY OF WAVEFORMS RECORDED FROM OESOPHAGEAL ELECTRODE BELOW THE ATRIAL LEVEL.	23
FIGURE 14	APPRAISAL OF PATIENTS WITH A FINAL DIAGNOSIS OF VENTRICULAR TACHYCARDIA.	25
FIGURE 15	SUPRAVENTRICULAR TACHYCARDIA (QRS < 120 msec).	28
FIGURE 16	WIDE QRS TACHYCARDIA WITH A RIGHT BUNDLE BRANCH BLOCK PATTERN.	29
FIGURE 17	OESOPHAGEAL RECORDING (SAME PATIENT AS IN FIGURE 16) DENOTING P:QRS DISSOCIATION. THE P WAVES ARE DENOTED ON THE OESOPHAGEAL RECORDING; THE SURFACE RECORDING APPEARS BELOW (V_2).	30
FIGURE 18	WIDE QRS COMPLEX TACHYCARDIA DISPLAYING A CONCORDANT QRS AXIS ACROSS V_1 TO V_6, FAVOURING A VENTRICULAR TACHYCARDIA.	31
FIGURE 19	OESOPHAGEAL RECORDING OF SAME PATIENT AS IN FIGURE 18. NOTE THE P:QRS DISSOCIATION. THE OESOPHAGEAL LEAD WAS RECORDED FROM V_1.	32
FIGURE 20	SURFACE ELECTROCARDIOGRAM (ABOVE) SHOWING A LEFT BUNDLE BRANCH BLOCK PATTERN ACROSS V_1 TO V_6. THE OESOPHAGEAL RECORDING BELOW DEMONSTRATES A SPIKY P WAVE FOR EACH QRS COMPLEX, DENOTING A SUPRAVENTRICULAR ORIGIN.	34

LIST OF TABLES

		Page
TABLE I	RECORDING DISTANCES	18
TABLE II	DETAILS OF CATEGORY: VENTRICULAR TACHYCARDIA (FINAL DIAGNOSIS)	19
TABLE III	DETAILS OF CATEGORY: SUPRAVENTRICULAR TACHYCARDIA (FINAL DIAGNOSIS)	26

LIST OF PLATES

		Page
PLATE I	THE MODIFIED OESOPHAGEAL LEAD IS CLIPPED ONTO THE V_1 TERMINAL OF THE RECORDER (SECTION 3.3.1).	15
PLATE II	COMPUTERIZED SCAN OF CROSS SECTION OF THORAX AT ATRIAL LEVEL. NOTE THE PROXIMITY TO THE OESOPHAGUS.	22

CHAPTER 1

INTRODUCTION

1.1 MOTIVATION FOR ENQUIRY

Tachyarrhythmias may occur in ischaemic heart disease and cardiomyopathy. These conditions are common in the South African Asian and Black population respectively. The arrhythmia may on occasions present as a wide complex tachyarrhythmia. This is defined on the standard electrocardiogram as an arrhythmia in which the heart rate is greater than 100 beats/minute and the QRS duration greater than 120 milliseconds (Stewart *et al.*, 1986). The tachyarrhythmia may arise in one of three places - the atrium, the ventricle, or the conducting tissues. In ventricular tachycardia the QRS complexes are wide (>120 milliseconds), whereas in supraventricular tachycardia the QRS complexes are usually narrow (<120 milliseconds) because the impulse is normally conducted via the left and right bundles. However, during a supraventricular tachyarrhythmia the right and left His bundles may not respond on a 1:1 basis. This results in an aberration of conduction. Alternatively, a totally abnormal conducting pathway may exist between the atria and ventricles. These circumstances favour the development of wide QRS complexes, and consequently the arrhythmia will resemble a ventricular tachyarrhythmia.

The differentiation of a wide complex tachyarrhythmia into either a supraventricular tachycardia or a ventricular tachycardia is important because of acute and long-term therapy as well as long-term prognosis in these two groups are different (Miles *et al.*, 1984; Stewart *et al.*, 1986; Wellens & Brugada, 1987). The difficulty of interpretation renders misdiagnosis common (Dancy *et al.*, 1985; Wellens *et al.*, 1984). Normally the P wave is clearly seen in a standard electrocardiogram, but the definition may be lost in the presence of a tachycardia (Enselberg, 1951; Hammil & Pritchett, 1981; Wellens & Brugada, 1987). In wide complex tachyarrhythmias,

the P wave is notoriously difficult to detect on the surface twelve lead electrocardiogram (Enselberg, 1951; Wu *et al.*, 1975; Jenkins *et al.*, 1979; Wellens & Brugada, 1987). Normal P waves are of small amplitude and therefore difficult to separate from baseline shifts and noise (Jenkins *et al.*, 1979). The P waves are often coincident with the QRS complexes or T waves and cannot be separated even by experienced electrocardiographers (Jenkins *et al.*, 1979; Dancy *et al.*, 1985). An especially important indicator of the origin of the tachyarrhythmia is the presence or absence of atrioventricular dissociation (Prystowsky *et al.*, 1980; Dancy *et al.*, 1985; Stewart *et al.*, 1986; Wellens & Brugada, 1987).

Presently, most physicians the world over routinely analyse the twelve lead surface electrocardiogram in order to correctly diagnose the tachyarrhythmia. Criteria to assist clinicians in this regard have been established (Wellens & Brugada, 1987). A lack of scrutiny of the electrocardiogram or unfamiliarity with diagnostic criteria may be the cause of misdiagnosis (Stewart *et al.*, 1986; Dancy *et al.*, 1985). There exists a considerable bias towards the diagnosis of supraventricular tachycardia amongst interns, registrars and consultant physicians (Wellens *et al.*, 1984; Dancy *et al.*, 1985). Consultant cardiologists may also frequently misinterpret the twelve lead electrocardiogram (Wellens *et al.*, 1984; Dancy *et al.*, 1985). Inappropriate therapy may frequently cause shock, hypotension, cardiac arrest or cardiac failure since most antiarrhythmic drugs have negative inotropic effects (Dancy *et al.*, 1985; Stewart *et al.*, 1986). The local experience, with the difficulty in eliciting a correct diagnosis from the twelve lead surface electrocardiogram, as well as the adverse sequelae of inappropriate therapy, concurs with the experience of other workers in other centres. Hence, the prime motivating factor for this dissertation is a desire to stem these adverse, often fatal, consequences of misdiagnosis.

1.2 OBJECTIVE AND SCOPE OF ENQUIRY

The primary objective is to assess the clinical usefulness of oesophageal electrocardiography in the differentiation of wide complex tachycardias. The second objective was to develop and evaluate a simple, safe technique that lends itself to routine clinical use. The demands upon the time and energy of the primary care doctor (often junior) need to be minimized.

Previously, many researchers experienced difficulty in the positioning of the electrode because large bore tubes were used, an example being the Ryles nasogastric tube which was filled with normal saline. Patient tolerance was poor because of extreme discomfort. Diagnostic yield was unrewarding because of distorted electrical waveforms. These factors may have been instrumental in the total abandonment of attempts to record the oesophageal electrocardiogram. If a complex procedure is used, the demands placed upon the physicians time and energy may preclude the technique from being used routinely. If expensive apparatus is used to evaluate the arrhythmia, severe strain upon the budget of a metropolitan hospital or clinic will also limit or preclude the routine use of a diagnostic technique. Hence the third objective was to utilize equipment that is simple and presently routinely available to local hospitals.

The fourth objective was to ensure that the entire technique is done at the bedside, in an emergency room or general ward, and not only in a specialized coronary care unit. The fifth objective was to limit the complications of the procedure to the barest minimum, in view of the adverse sequelae experienced by other workers previously.

The patients were enrolled prospectively. Eighteen patients were studied over a period of twenty months. Only patients who fulfilled the criteria outlined under Chapter 3 (Patients and Methods) were enrolled. The study is designed to promote rapid diagnosis at the bedside soon after initial presentation to an emergency room. The initial twelve lead surface

electrocardiogram, as well as the diagnosis of the emergency doctor, were obtained and later analysed in comparison to the final diagnosis following the study of the oesophageal electrocardiogram. Comparisons are made with respect to the frequency of misdiagnosis in both categories of patients, those with a final diagnosis of supraventricular tachycardia with aberrant conduction and those with a diagnosis of ventricular tachycardia. A review of the hospital records enables a documentation of complications prior to the use of oesophageal electrocardiography to be made.

CHAPTER 2

REVIEW OF THE LITERATURE

2.1 MECHANISMS OF ARRHYTHMIAS

For anatomical and physiological purposes, there are six possibilities if one encounters a wide complex tachyarrhythmia (Wellens & Brugada, 1987). These are :

i. A supraventricular tachycardia with a pre-existent or tachycardia related bundle branch block (Figure 1).

ii. A 'circus' movement tachycardia with atrioventricular conduction over the A-V node and ventriculo-atrial conduction over an accessory pathway in the presence of a bundle branch block (Figure 2).

iii. A supraventricular tachycardia with atrioventricular conduction over an accessory pathway (Figure 3).

iv. A 'circus' movement tachycardia with atrioventricular conduction over an accessory atrioventricular pathway and ventriculo-atrial conduction (ie. retrograde conduction) over the atrioventricular node (Figure 4).

v. A supraventricular tachycardia with atrioventricular conduction over a nodo-ventricular fibre (Figure 5).

vi. A ventricular tachycardia (Figure 6).

Most authors are in agreement that there are two important types generally in terms of management - a supraventricular tachycardia with aberrant conduction (Figures 1 to 5), and a ventricular tachycardia (Figure 6). From the outset the differentiation of wide complex tachyarrhythmias into either component type has intrigued physicians. Hence, various criteria have been devised to evaluate the twelve lead electrocardiogram in order to differentiate the

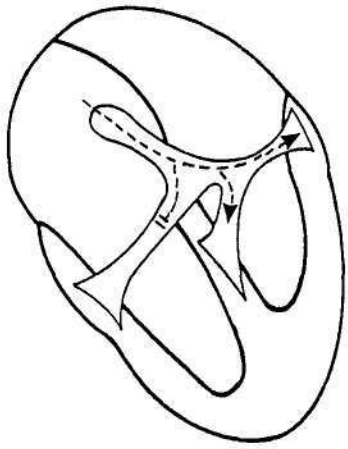


Figure 1

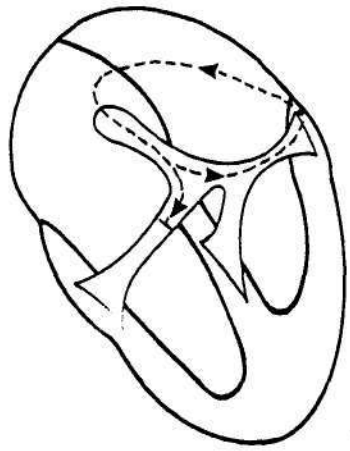


Figure 2

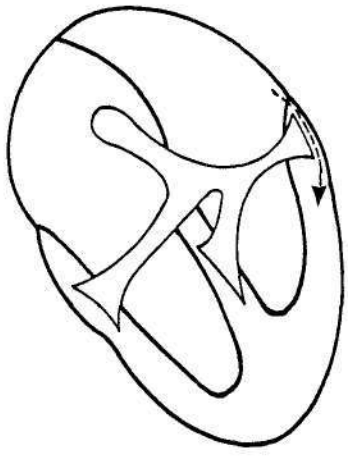


Figure 3

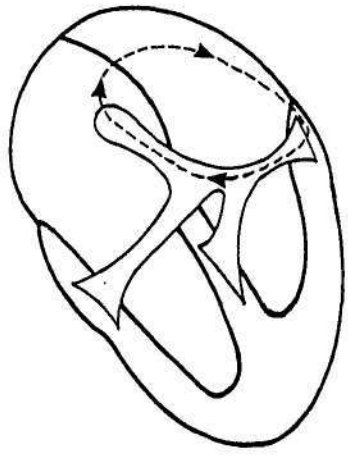


Figure 4

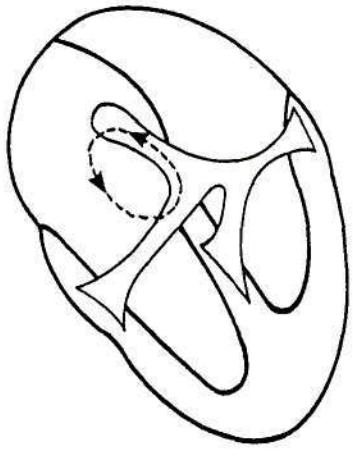


Figure 5

Figure 6

FIGURES 1-6. Diagrammatic representation of the six mechanisms of arrhythmias.

origin of a wide complex tachyarrhythmia. These criteria are discussed next.

2.2 CRITERIA FOR DIFFERENTIATION

2.2.1 The ventricular rate of the tachyarrhythmia

It is generally accepted that a supraventricular tachycardia with aberrant conduction tends to be faster (>200 beats/minute) than a ventricular tachycardia (Graboys, 1987). This has proved to be too crude and unreliable a criterion for differentiation (Wellens *et al.*, 1978, Wellens & Brugada, 1987).

2.2.2 The regularity

Atrial fibrillation in the presence of a bundle branch block or abnormal atrioventricular conduction pathways consistently produces completely irregular QRS patterns. This is a useful clue to the origin of the tachyarrhythmia. Wellens & Brugada (1987) state that all other supraventricular tachycardias (in the presence of either pre-existing or tachycardia related bundle branch blocks), as well as ventricular tachycardias, usually display a regular set of QRS complexes; if irregularities occur they are occasional.

2.2.3 Atrioventricular dissociation

The hallmark of differentiation between a ventricular tachycardia and a supraventricular tachycardia with aberrant conduction is the presence or absence, respectively, of atrio-ventricular dissociation (Robinson & Herrman, 1921; Massumi *et al.*, 1967; Stewart *et al.*, 1986; Wellens *et al.*, 1978; Dancy *et al.*, 1985; Wu *et al.*, 1975; Jenkins *et al.*, 1979). Wellens & Brugada (1987) found that the all important recognition of atrial activity on the surface electrocardiogram was difficult and often impossible. The manifestation of atrial activity on the surface electrocardiogram is dependent in part on the proximity of the recording electrode to the atria (Wu *et al.*, 1979). Normal P waves are slow and of small amplitude and they may coincide with QRS complexes or T waves in a tachycardia. These features render interpretation of the surface

electrocardiogram difficult (Jenkins *et al.*, 1979). Of practical importance is that atrioventricular dissociation is never a feature of an atrial arrhythmia with a rapid ventricular response; rather, it strongly suggests a ventricular tachycardia (Dancy *et al.*, 1985; Langendorf, 1959).

2.2.4 The nature of the QRS complexes

Wellens *et al.* (1981) found that a QRS duration of greater than 140 milliseconds is suggestive of a ventricular tachycardia. Coumel *et al.* (1984) found that if the patient with ventricular tachycardia had ischaemic heart disease, the QRS complex was usually very wide ($>170 \pm 10$ milliseconds), whereas an idiopathic ventricular tachycardia displayed QRS complexes 135 ± 11 milliseconds long. Greenspan (1986), however comments that the presence of an antiarrhythmic drug renders this criterion unhelpful.

2.2.5 The QRS axis

The axis of the QRS in the frontal plane is not a reliable indicator of the origin of the tachyarrhythmia. In ischaemic heart disease patients with ventricular tachycardia, the QRS axis usually points superiorly; in idiopathic cases it usually follows a normal axis; in supraventricular tachycardia with aberrant conduction the axis is highly variable depending on the location of the pathway of conduction. Kindwall (1988) showed that a left axis alone was of no value in distinguishing a ventricular tachycardia from a supraventricular tachycardia with aberrant conduction.

2.2.6 The QRS configuration

2.2.6.1 Concordance. concordance of the QRS complexes across leads V_1 to V_6 implies uniformity in direction of the complexes. Although this is suggestive of ventricular tachycardia, it may occur in a supraventricular tachycardia conducted via an accessory pathway which inserts into the posterobasal left ventricle (Wellens & Brugada, 1987).

2.2.6.2 The right bundle branch block shaped QRS complex. Marriot (1970) studied the morphology of the QRS complexes in wide complex tachyarrhythmias and concluded that a

triphasic complex in lead V_1, as opposed to a biphasic or monophasic complex, was highly suggestive of supraventricular tachycardia with aberrant conduction. This was confirmed by Dancy *et al.* (1985). Wellens *et al.* (1981) described a new phenomenon by studying the V_6 lead. In a supraventricular tachycardia with aberrant conduction the QRS in V_6 began typically with a Q wave followed by an R wave which was taller than the ensuing S wave (Figure 7). In contrast, a ventricular tachycardia was found to often display a QS complex in lead V_6 (Figure 8).

2.2.6.3 The left bundle branch block shaped QRS complex. A slurred S wave on the downstroke in lead V_1, coupled with an initial positive deflection in V_1 suggests a tachycardia of ventricular origin (Kindwall *et al.*, 1987). This is shown in Figure 9. However, these workers subsequently reported that there was a low sensitivity (30 to 44%) in using these criteria and therefore its application in practice is limited (Kindwall *et al.*, 1987).

2.2.7 Variation in QRS complex shapes

When several shapes of the QRS complexes are seen during a tachycardia in the same patient on different occasions, this is suggestive of a ventricular tachycardia (Dancy *et al.*, 1985).

2.2.8 Clinical criteria

At the bedside the following features may suggest a diagnosis of ventricular tachycardia:

i. History of myocardial infarction.

ii. Wide splitting of the first heart sound due to ventricular asynchrony.

iii. Cannon 'a' waves in the jugular venous pulse and beat-to-beat variation in the intensity of the first heart sound, supporting AV dissociation.

2.3 CONSEQUENCES OF MISDIAGNOSIS

Detailed analysis of the QRS morphology is extremely difficult to apply in an emergency as complex details are often forgotten (Dancy *et al.*, 1985). In addition, the criteria described may be unreliable, and recent reports of the outcome of patients with wide complex tachycardias

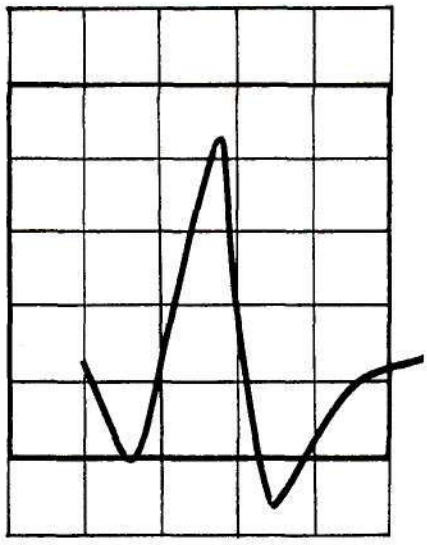


Figure 7

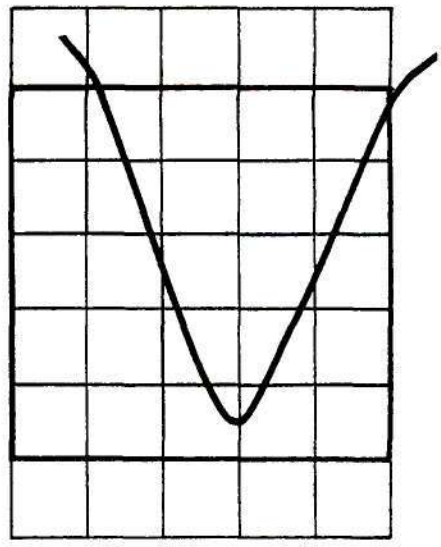


Figure 8

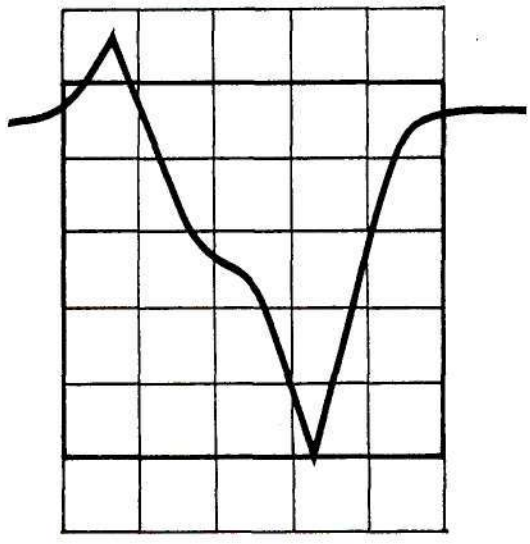


Figure 9

FIGURE 7. The QRS morphology in V_6 favouring a supraventricular origin.

FIGURE 8. The QRS morphology in V_6 favouring a ventricular origin.

FIGURE 9. The QRS morphology in V_1 favouring a ventricular origin.

suggest that errors in diagnosis are still common (Stewart *et al.*, 1985; Dancy *et al.*, 1985). Inappropriate therapy follows, and the consequences are disastrous. If verapamil is used, hypotension, cardiac arrest and cardiac failure may result commonly (Stewart *et al.*, 1986; Dancy *et al.*, 1985; Wellens & Brugada, 1987). Dancy *et al.* (1985) noted in a retrospective study of 163 episodes of wide complex tachycardia in 24 patients that there was a considerable bias towards the diagnosis of supraventricular tachycardia. The clinicians included interns, registrars, consultant physicians and cardiologists. All 24 of their patients had repeatedly been misdiagnosed in the emergency department.

2.4 ATTEMPTS AT IMPROVING DIAGNOSIS

Early attempts aimed at improving P wave detection included novel positioning of surface electrodes. A specific electrode at the right parasternal border was used by Lewis (1925) in order to record right atrial activity. Occasionally paired leads from the right sternal border were used to record atrial recordings (Butterworth & Poindexter, 1946). These methods were crude and did not facilitate good recordings to assist in the diagnosis of the origin of the tachyarrhythmias (Enselberg, 1951). Moreover, factors such as obesity, heavy intercostal and pectoral muscle bulk, emphysema, abnormal position of the heart due to lung fibrosis, pleural effusions and pericardial effusions may conceivably hamper surface recordings. Recently, sophisticated and complex techniques have been utilised in order to assist in the diagnosis of these arrhythmias. Della Bella *et al.* (1987) evaluated a transcardiac electrode and established that in conjunction with algorithms applied to simultaneous surface recordings, accurate diagnosis of the arrhythmias was possible. However, this technique is invasive as intravenous cannulation to the level of the right atrium is required. Slocum *et al.* (1985) devised an algorithm following computer analysis of surface electrocardiograms but concluded that the sensitivity of such a method for the detection of atrioventricular dissociation was only 65% despite the 100% specificity. Schnittger *et al.* (1986) used a bipolar capsular electrode

positioned in the oesophagus but an amplifier and triple channel electrocardiograph recorder were needed. All 14 of their patients with difficult undiagnosed arrhythmias displayed excellent tracings to enable a correct definitive diagnosis in all patients. However, such equipment is expensive and is not available to local hospitals. Jenkins *et al.* (1979) utilized a bipolar rod suspended from electrical wires as an oesophageal electrode. The oesophageal recordings were amplified two to four times and band limited from 5 to 100 Hz to eliminate low frequency artefacts; the signal was differentiated and then fed to a threshold detector, and finally to a computer which displayed the signal for analysis of the arrhythmias. The method proved extremely reliable in the detection of atrial fibrillation, premature atrial beats, premature ventricular beats and all degrees of heart block, as well as in the differentiation of wide complex tachycardias. Such equipment used in their study is not routinely available; and it requires a high level of expertise.

CHAPTER 3

PATIENTS AND METHODS

3.1 PATIENT SELECTION

The study was a prospective one. The patients were selected sequentially following presentation to the emergency department with a wide complex tachyarrhythmia. The following criteria were prerequisites prior to enrolment :

i. The demonstration of a wide complex tachyarrhythmia as defined on a standard twelve lead surface electrocardiogram.

ii. Patients with hypotension (BP <90/60 mmHg), hypoxic confusion from cerebral hypoperfusion, or cardiac failure were excluded. In such circumstances the treatment of choice is cardioversion.

iii. The acquisition of informed consent, (as detailed in Appendix 1) was a prerequisite to oesophageal electrocardiography.

The interpretation of the twelve lead surface electrocardiogram by the emergency doctor and medical registrar was noted.

3.2 CONSENT

Informed consent was obtained by means of a standard, prepared consent form. Specific details regarding the procedure was verbally explained to each patient personally. The study, and consent form, was approved by the Ethics Committee of the University of Natal.

3.2.1 Information given to subject (prior to consent)

The following text is written with reference to the subject.

"You have an abnormal fast heart rhythm. This is visible on the surface electrocardiogram. The origin of the fast rhythm is of great importance to your doctor, because correct treatment depends upon this information. I intend to obtain a clear trace of your heart recording using an electrode positioned in your oesophagus (foregut). This requires the passage of a flexible electrode through your nasal passage. You need to swallow this electrode. To ease the passage of the electrode it has been lubricated. In the event of extreme nasal or throat irritation, you may indicate your discomfort and refusal to proceed. Following a correct diagnosis, appropriate therapy will be administered to abolish your fast heart rhythm".

3.3 EQUIPMENT

3.3.1 Oesophageal electrode

We devised a simple oesophageal electrode by adapting a transvenous pacing lead which is normally used routinely locally as an adjunct to a temporary external pulse generator ('pacemaker'). It is a flexible lead (Plate I) with platinum electrodes at one end. This provides excellent conductivity. At the other end are two pins which are normally connected directly to the temporary pulse generator. One of these pins is modified so that it may be connected to the V_1 terminal of the electrocardiograph recorder. This modification entails soldering the end to a routinely available crocodile clip (Plate 1). To enable simultaneous surface recordings to occur it is sufficient to connect either the V_2 or V_3 terminal to the usual surface position. The flexible lead is covered in durable vinyl by manufacturing design. It was marked into 5 cm intervals along its length in visible, non-erasable, non-toxic, non-allergenic paint normally used by toy manufacturers.

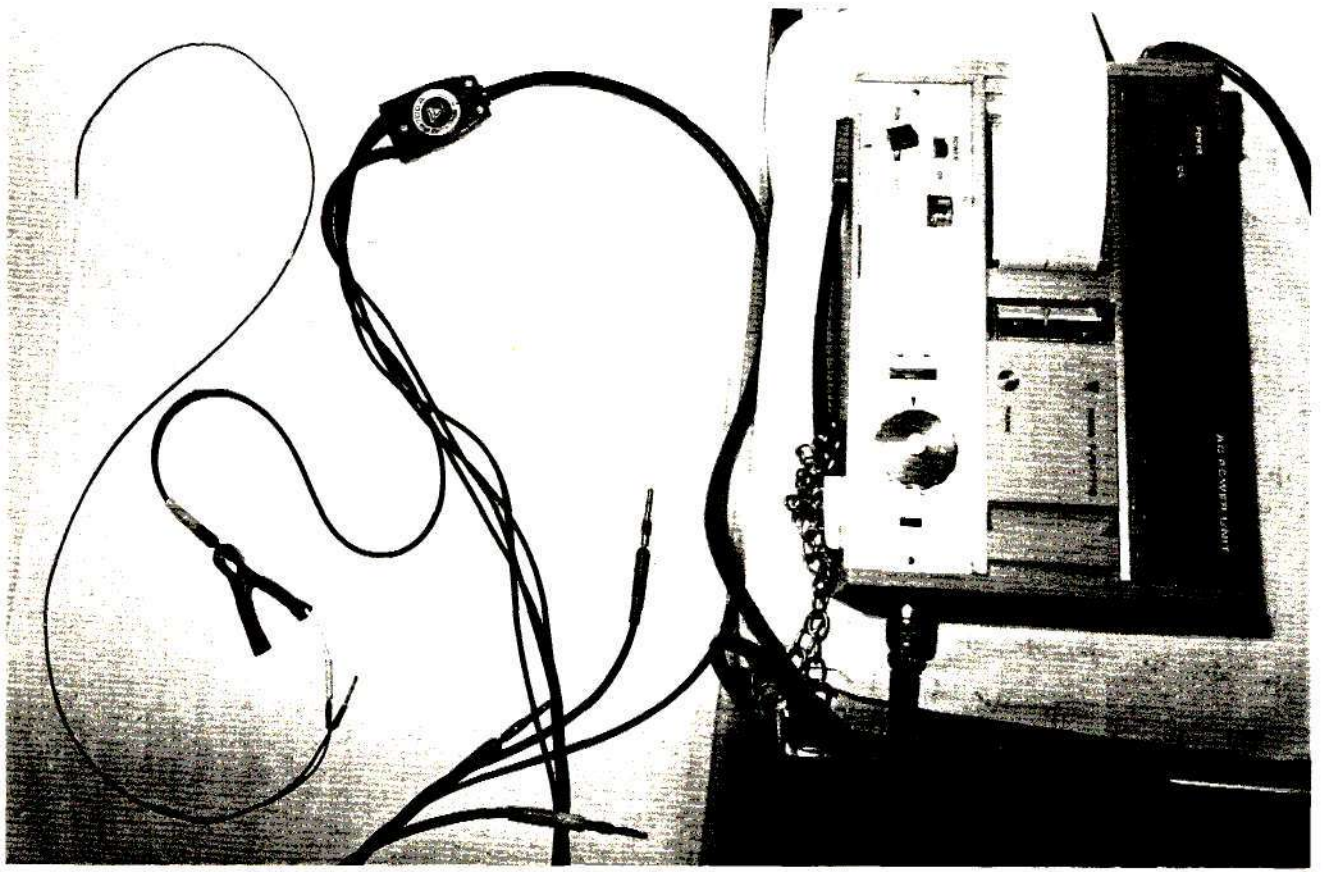


PLATE I. The modified oesophageal lead is clipped onto the V_1 terminal of the recorder (Section 3.3.1).

3.3.2 Electrocardiographic recorder

The recordings were done at a velocity of 25 mm second, and a voltage specification of 1 millivolt per centimetre. The machines, denoted below, are routinely available to local hospitals.

FUKUDA "MODEL FJC-7110"
HEWLETT-PACKARD "4700A CARDIOGRAPH".

All recordings done on a single channel machine (illustrated) were repeated on the 3 channel machine

3.4 PROCEDURE

The "oesophageal" electrode was lubricated. The lead was introduced into the naries; the distal end remained free for easy manoeuverability. The patient was given a glass of water to encourage swallowing. The lead was passed to a depth of 50 cm. The distal end, attached to a crocodile clip, was clipped onto either the V_1 or V_2 terminal of an electrocardiograph machine. The patients were asked to expire and hold their breath. The recording was then commenced, and the lead was withdrawn in half to one centimetre intervals during a continuous recording. The position of the highest and lowest atrial activity was noted, as well as the level of the best obtainable recording.

CHAPTER 4

RESULTS

Arrhythmias in 18 patients were studied over a period of 20 months. In two instances the patients were referred after having had only vagal stimulation procedures done, eg. eyeball pressure and carotid massage. The rest of the patients were treated with antiarrhythmic drugs prior to their arrival. Five patients were females, and thirteen patients were males. The average age of the female patients was 48.2 years; the average age of the males was 48.5 years.

The frequency of symptoms is listed :

Syncope	10 (55%)
Palpitations	6 (33%)
Chest pain	8 (44%)
Dyspnoea	6 (33%)
"Chest uneasiness"	9 (50%)

Two patients who complained of chest pain sustained a myocardial infarction (Table II). The diagnosis was made on estimation of cardiac enzymes in the serum. The rest of the patients displayed normal cardiac enzymes.

4.1 RECORDING DISTANCES

The distances from the naries to the optimal recording varied in different patients. Table I shows the highest atrial recording levels, the lowest levels, as well as the best levels.

The frequency histogram for the optimal recording distances appears in Figure 10.

TABLE I. Recording distances.

HIGHEST ATRIAL RECORDING LEVEL (cm)	LOWEST ATRIAL RECORDING LEVEL (cm)	OPTIMAL LEVEL (cm)
29.5	37.5	34.0
31.0	37.0	35.5
36.0	41.0	39.0
28.0	35.0	30.0
35.0	44.5	36.5
32.0	39.0	35.0
35.5	43.0	37.0
30.0	38.0	32.5
31.5	40.0	33.0
32.0	43.0	36.0
31.0	34.5	34.0
35.5	44.0	37.0
34.0	42.5	38.5
35.0	42.0	39.0
33.0	41.0	35.5
36.0	45.0	37.0
34.0	41.5	38.0
34.5	40.5	37.5
Median and standard deviation		
33.5 ± 2.4	41 ± 3.1	36.25 ± 2.4

TABLE II. Details of category: Ventricular tachycardia (final diagnosis).

UNDERLYING MEDICAL HISTORY	HEART RATE	EPISODES OF ARRHYTHMIA*	COMPLICATIONS**	THERAPY***
IHD	180/m	5	Hypotension Cardiac failure Cardiac arrest Recurrence	Verapamil IV Lignocaine IV
Clear	220/m	8	Cardiac arrest Hypotension Recurrence	Verapamil IV Disopyramide IV
HT§ DM	188/m	-	Nil	Lignocaine IV
HT DM	210/m	2	Cardiac arrest	Verapamil IV
"Influenza like illness"§	196/m	-	Nil	Disopyramide
IHD, AMI Peripheral vasculopathy	204/m	-	Nil	Verapamil IV
"influenza like illness"§	176/m	-	Persistence	Verapamil IV
CCMO	184/m	1	Cardiac failure Hypotension	Lignocaine IV
IHD	206/m	-	NII	Cardiac massage Eyeball pressure
DM, AMI IHD	180/m	2	Hypotension	Verapamil IV
CCMO	200/m	1	Hypotension	Digoxin

KEY:
* Total number of episodes of arrhythmia prior to entry into study
** Complications prior to entry into study
*** Therapy prior to entry into study
§ Correct initial diagnosis

ABBREVIATIONS
IHD = ischaemic heart disease
CCMO = congestive cardiomyopathy
AMI = acute myocardial infarction
HT = hypertension
DM = diabetes mellitus

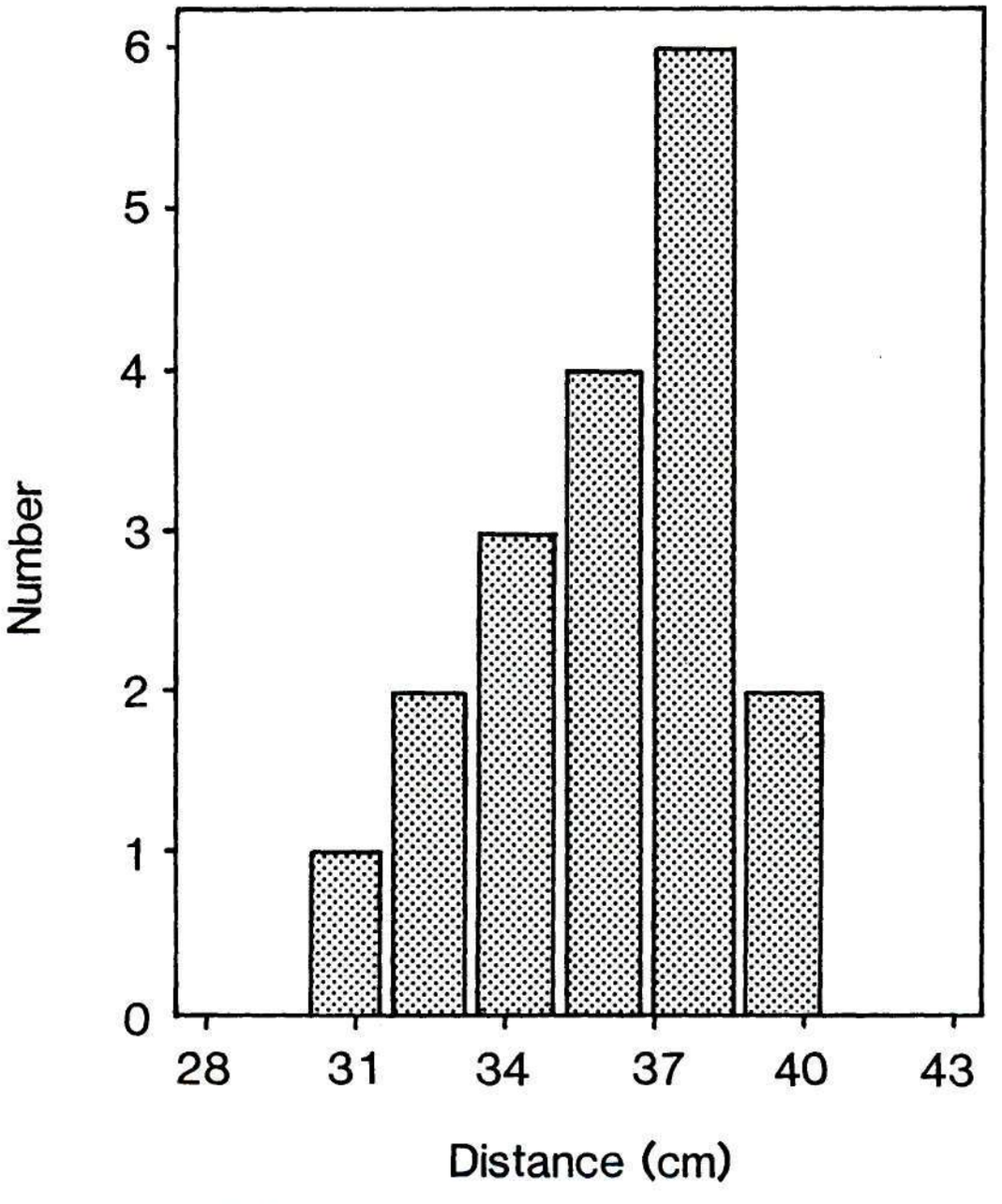


FIGURE 10. Frequency histogram of optimal recording distances.

4.2 TOLERANCE TO PROCEDURE

There were no major complications. Minor irritation occurred during nasal introduction in five patients and in two of these five the lead was reinserted after reassurance, with no further complications. The average time taken for a complete study of different levels was 4.5 minutes (range 3 minutes to 6.5 minutes).

4.3 ARTEFACTS

Respiratory excursions accounted for many episodes of wandering baselines on the recordings. To overcome this, the recordings were taken at the end of expiration, during breathholding. Four patients were anxious, and swallowing movements added to baseline excursions, but in no patient did this preclude a decipherable recording.

4.4 TYPICAL P WAVE MORPHOLOGY

The morphology of the P wave was dependent on the position of the recording oesophageal electrode relative to that of the atrium. For clarity, a computerized scan of a horizontal cross section of the thorax between the seventh and eighth thoracic vertebra is included (Plate II). Note the proximity of the oesophagus to the atria. The typical morphology of the oesophageal electrode recording at a level above (Figure 11), at (Figure 12) and below (Figure 13) the atria is depicted. The P wave was found to be typically biphasic at the atrial level (Figure 12), dominantly negative at higher levels (Figure 11), and dominantly positive below the atrial level. As the electrode was passed further below, ventricular activity was recorded (synchronous with the surface lead QRS complexes) and the P waves were lost.

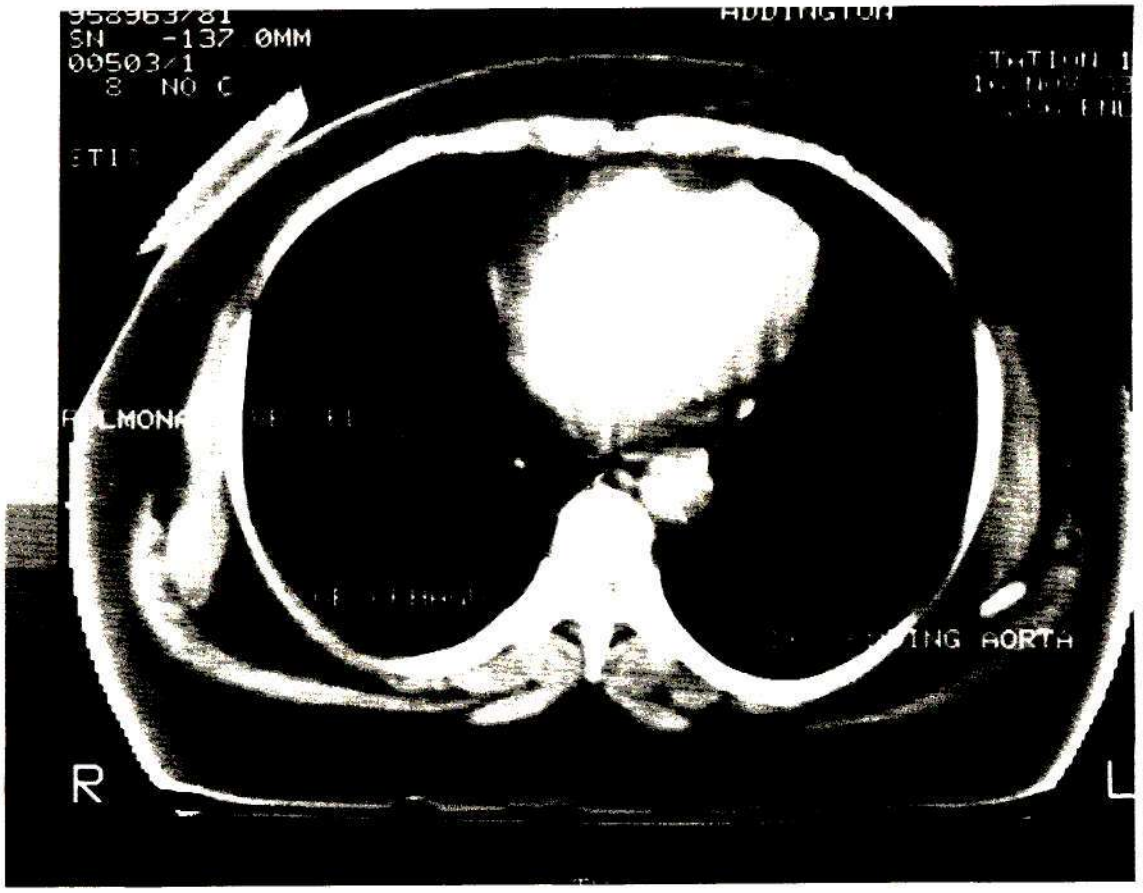


PLATE II. Computerised scan of cross section of thorax at atrial level. Note the proximity to the oesophagus.

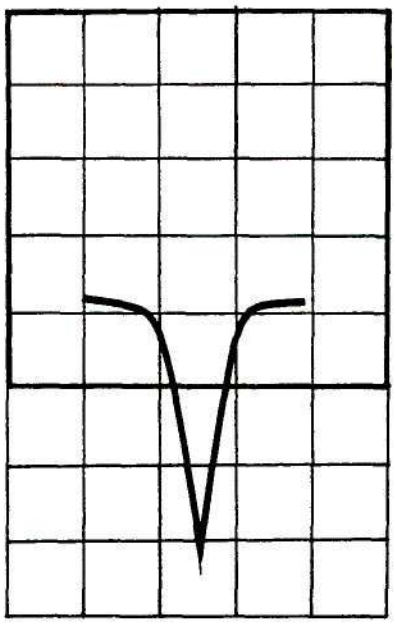


Figure 11

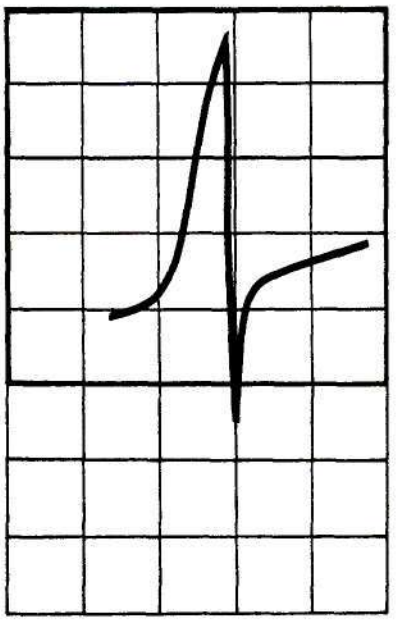


Figure 12

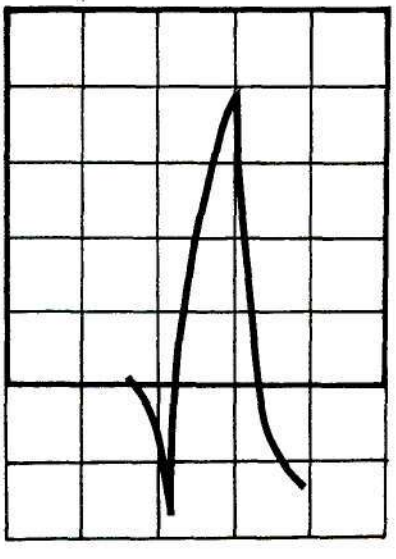


Figure 13

Typical morphology of waveforms recorded from oesophageal electrode at varying levels.

FIGURE 11. Above the atrial level.

FIGURE 12. At the atrial level.

FIGURE 13. Below the atrial level.

4.5 REVISION OF DIAGNOSIS

Results before and after the oesophageal electrocardiogram are depicted in Figure 14. Fifteen patients were initially diagnosed as supraventricular tachycardia and three patients as ventricular tachycardia. These initial diagnoses are those of the emergency doctor.

The revised diagnosis was ventricular tachycardia in eleven patients and supraventricular tachycardia in seven patients. Nine of the eleven patients with ventricular tachycardia were initially diagnosed as supraventricular tachycardia, whilst only one of seven patients with supraventricular tachycardia was misdiagnosed. The most notable feature on the flow diagram, however, is a bias towards an initial diagnosis of supraventricular tachycardia.

4.5.1 Appraisal of patients with a final diagnosis of ventricular tachycardia

Table II summarises the important data in this category.

In Table II it can be seen that there are six patients presenting with more than one episode of arrhythmia prior to reaching a final diagnosis on the oesophageal electrocardiogram. These six patients were given verapamil and complications ensued in five of these patients.

4.5.2 Appraisal of patients with a final diagnosis of supraventricular tachycardia

Six of the seven patients with a final diagnosis of supraventricular tachycardia had no adverse effects following treatment (Table III). The one patient who developed cardiac failure also had anaemia and chronic renal failure, hence the complication may not have been associated with treatment.

4.6 PROGRESS AFTER REVISED DIAGNOSIS

Two episodes of recurrent ventricular tachycardia was noted in one of two patients who sustained myocardial infarctions. These were controlled with amiodarone. No recurrence of arrhythmias was noted at follow-up in other patients (follow-up period range 1-18 months; average 7 months).

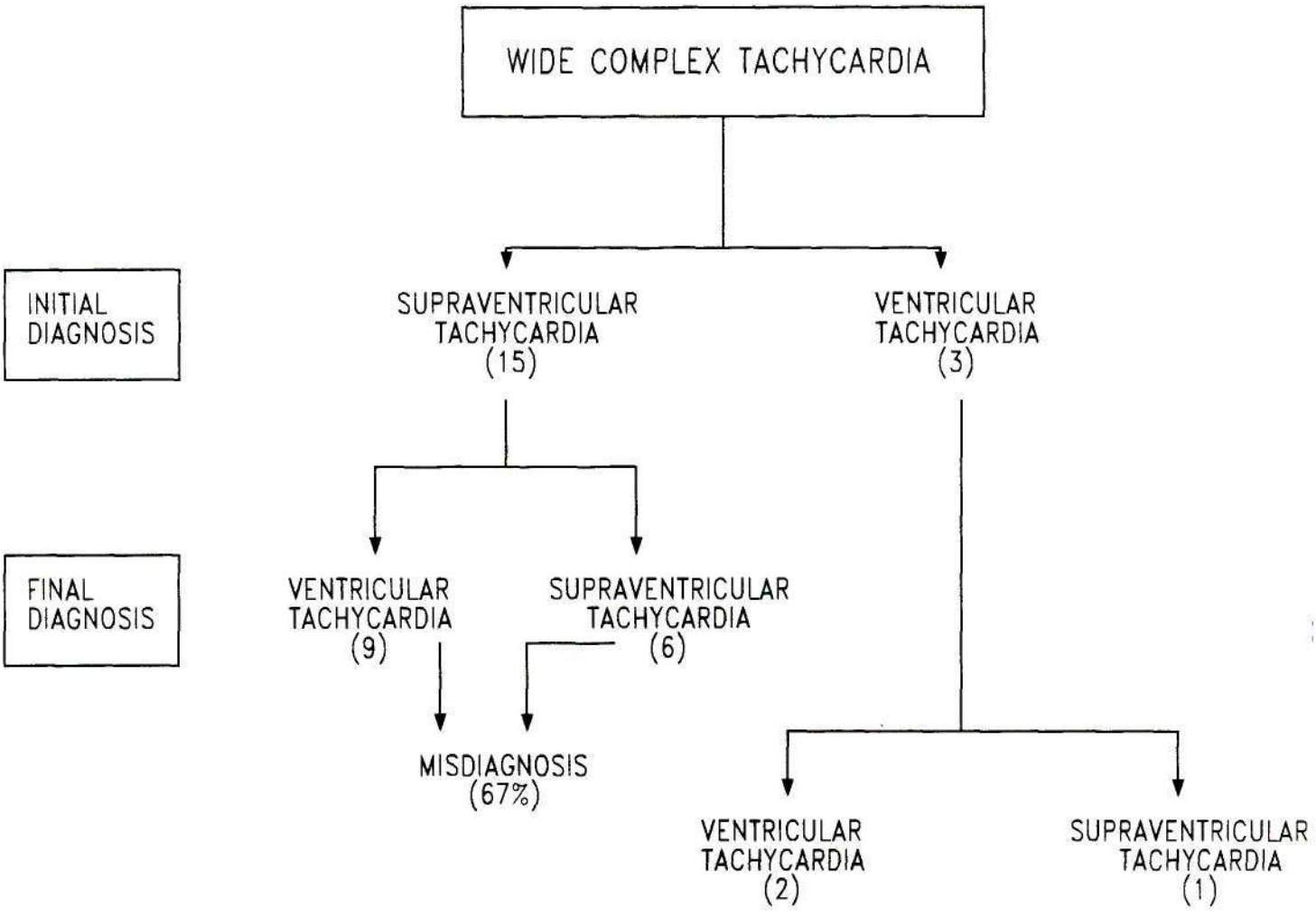


FIGURE 14. APPRAISAL OF PATIENTS WITH A FINAL DIAGNOSES OF VENTRICULAR TACHYCARDIA.

TABLE III. Details of category: supraventricular tachycardia.

UNDERLYING MEDICAL HISTORY	HEART RATE	EPISODES OF ARRHYTHMIA	COMPLICATIONS	THERAPY
DM anaemia Renal failure	188/m	3	Cardiac failure	Verapamil IV
HT Cardiac failure on treatment	210/m	-	Nil	Digoxin PO
IHD Gout	194/m	-	Nil	Diltiazem PO
IHD**	196/m	-	Nil	Disopyramide IV
Clear	188/m	2	Nil	Verapamil IV
CCMO	204/m	-	Nil	Carotid massage
"influenza like illness"	208/m	-	Nil	Verapamil PO

Key as for Table II.

4.7 ILLUSTRATIVE EXAMPLES

The impact of oesophageal electrocardiography in this study is best appreciated when comparison with conventional surface electrocardiograms is made. The series of electrocardiograms that follow are laser print reproductions of the original recordings.

A classical supraventricular tachycardia is illustrated in the conventional twelve lead electrocardiogram in Figure 15 (reduced) to demonstrate narrow QRS complexes of less than 120 milliseconds (less than three small blocks wide).

The tachycardia in Figure 16 demonstrates wide complexes. The initial diagnosis of the emergency doctor was a supraventricular tachycardia, because of the right bundle branch block pattern and discordant QRS complexes across leads V_1 to V_6.. In Figure 17 (enlarged), the oesophageal recording of the same patient was recorded simultaneously with V_2. There is P:QRS dissociation and the revised diagnosis was hence a ventricular tachycardia. Note the wandering baseline (referred to in Section 4.3) due to respiratory excursions. This oesophageal recording was performed at 36 cm and it highlights the spiky biphasic nature of the P wave at this "atrial level".

The wide complex tachycardia depicted in Figure 18 was diagnosed initially as a ventricular tachycardia (note the concordant QRS complexes across leads (V_1 to V_6). The oesophageal electrocardiogram (enlarged) is shown in Figure 19 and clearly demonstrates P:QRS dissociation and confirms the diagnosis as ventricular tachycardia. The P wave in this recording is dominantly negative because the recording was taken at a distance of 30 cm from the naries ("above atrial level").

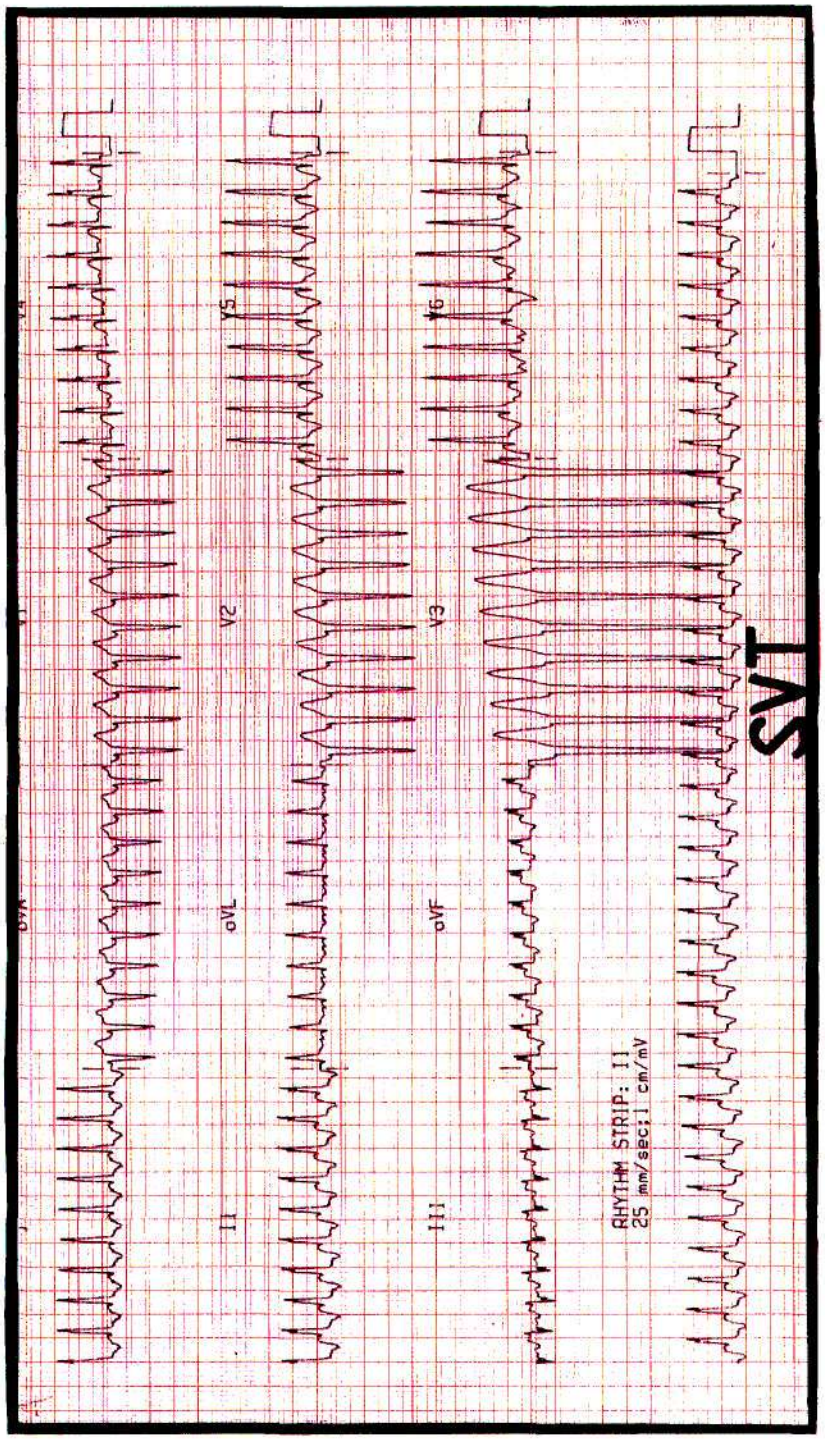


FIGURE 15. Supraventricular tachycardia (QRS <120 msec).

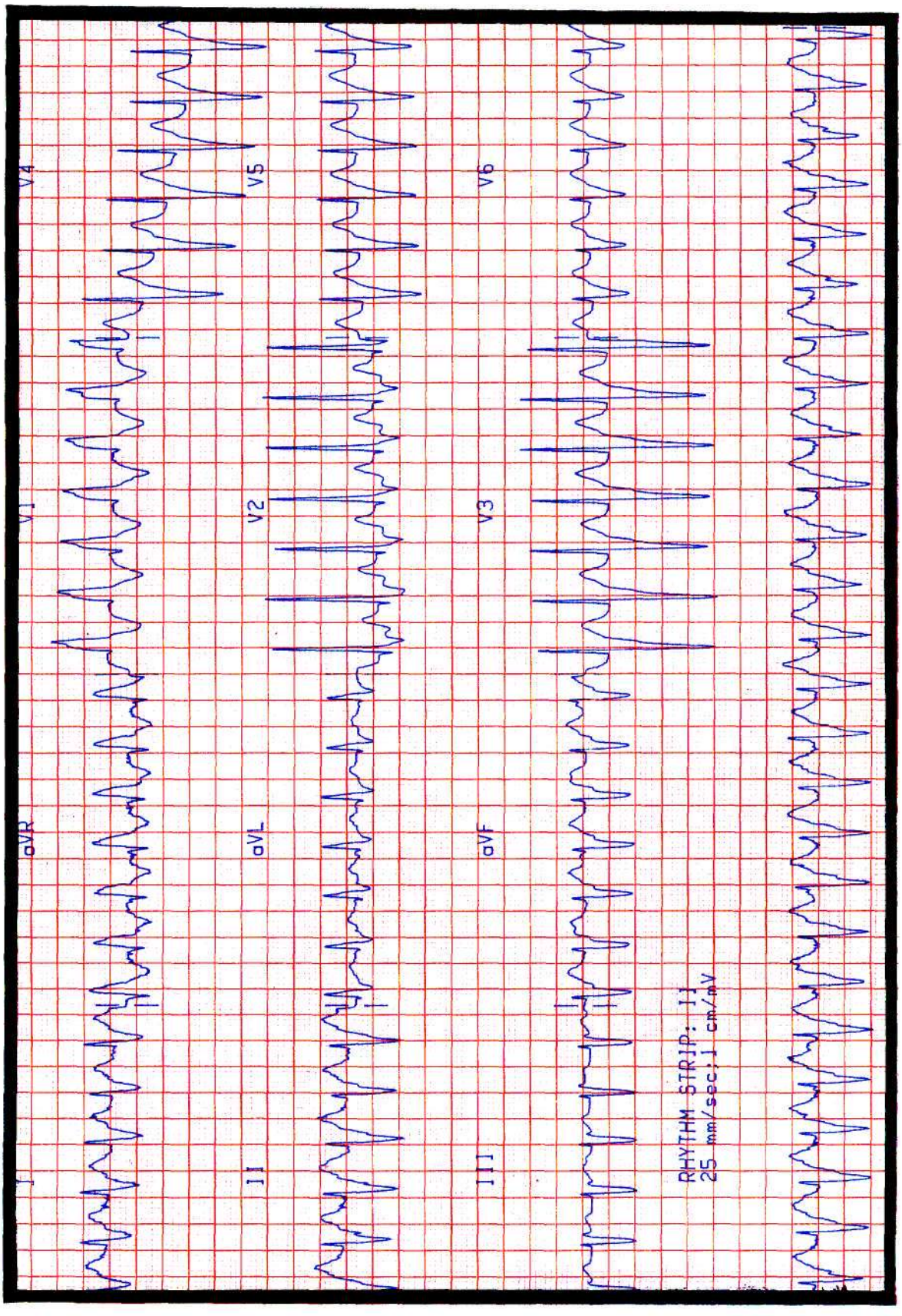


FIGURE 16. Wide QRS tachycardia with a right bundle branch block pattern.

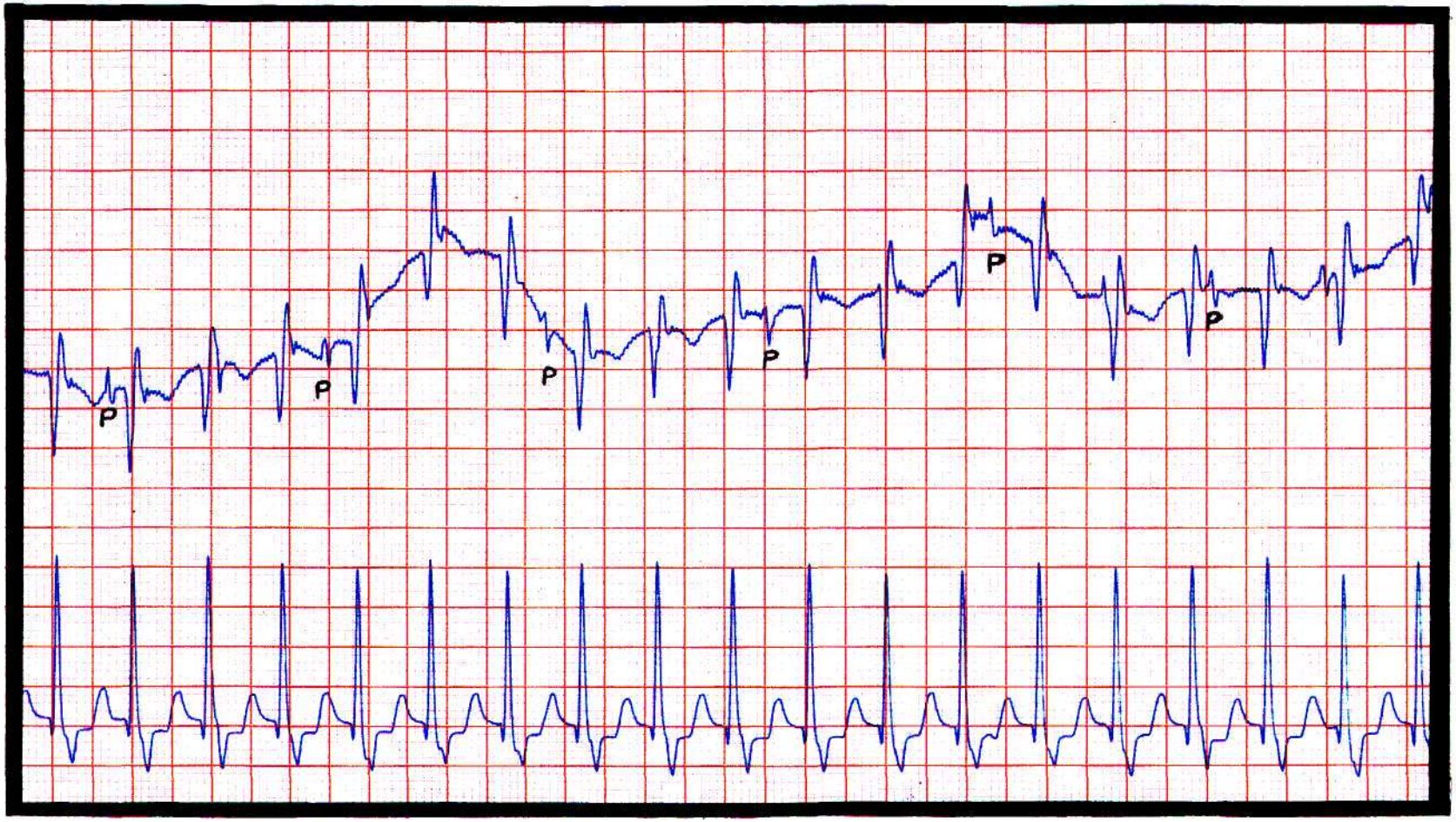


FIGURE 17. Oesophageal recording (same patient as in Figure 16) denoting P:QRS dissociation. The P waves are denoted on the oesophageal recording; the surface recording appears below (V_2).

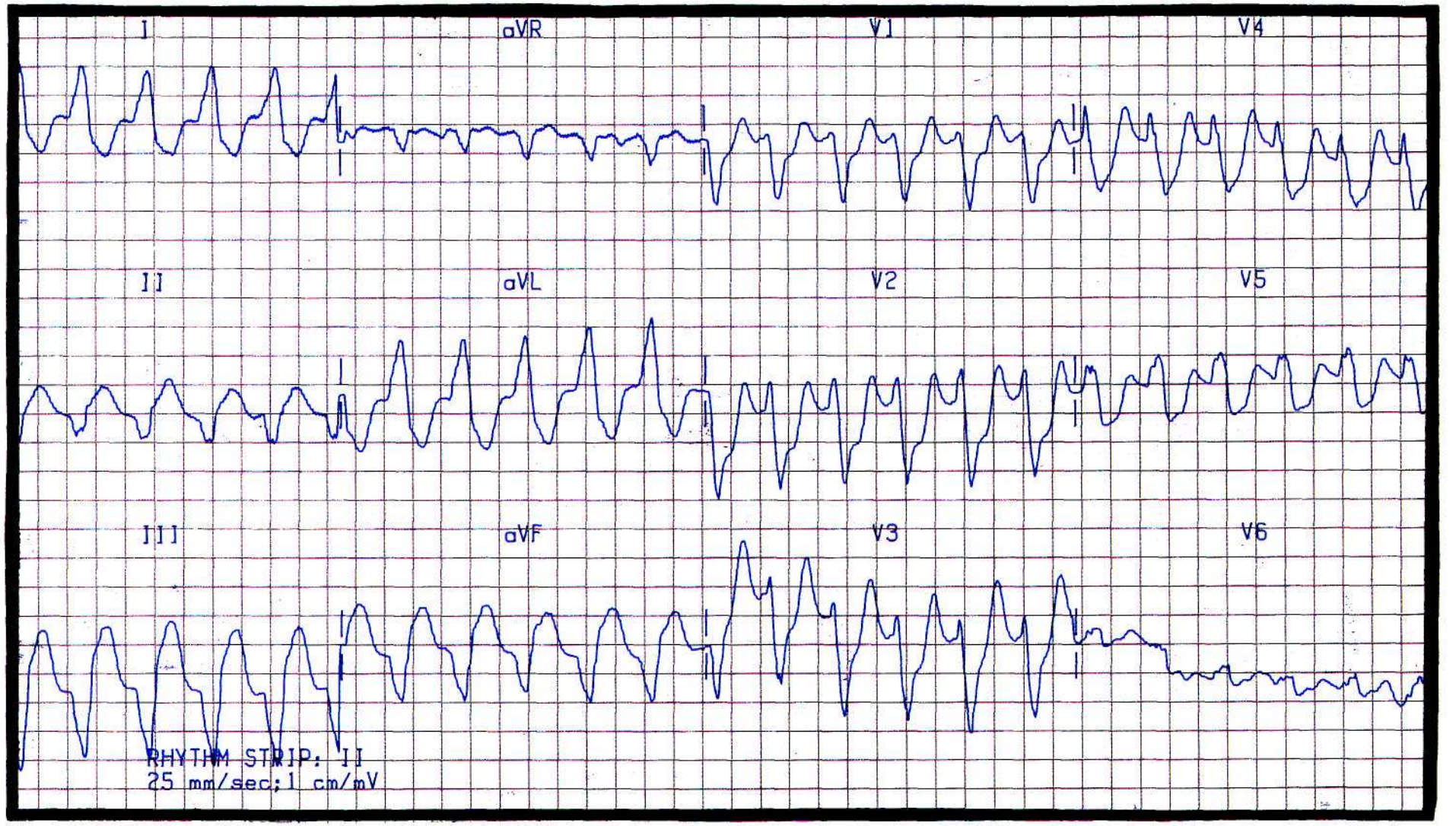


FIGURE 18. Wide QRS complex tachycardia displaying a concordant QRS axis across V_1 to V_6, favouring a ventricular tachycardia.

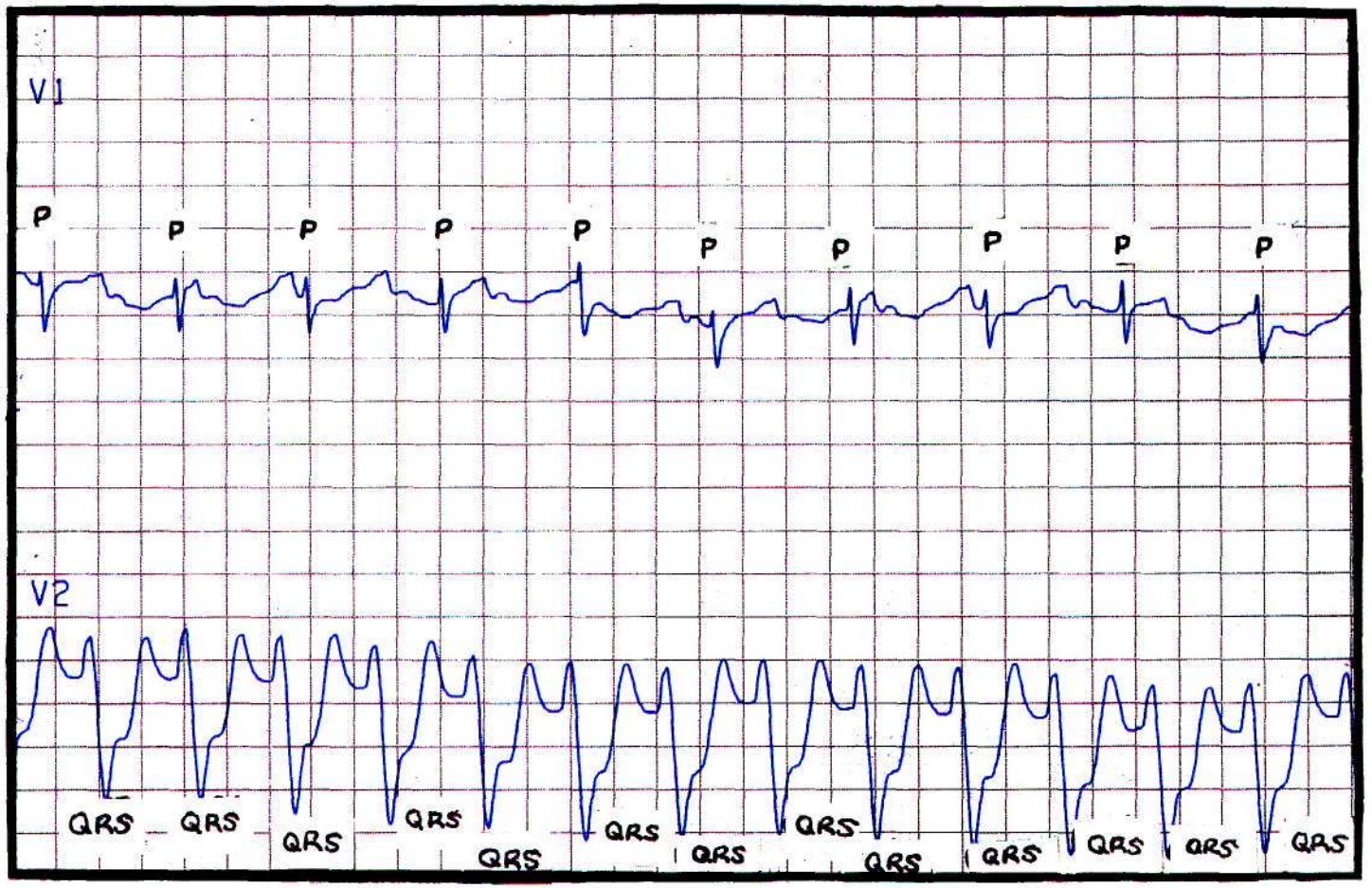


FIGURE 19. Oesophageal recording of same patient as in Figure 18. Note the P:QRS dissociation. The oesophageal lead was recorded from V_1.

In Figure 20 (enlarged), both the surface chest lead recordings and the oesophageal recording are reproduced. The initial diagnosis was a ventricular tachycardia. The left bundle branch block pattern was consistent with this diagnosis. However, the oesophageal recording, at a distance of 39 cm from the naries ("below atrial level") refutes this diagnosis because of the 1:1 P to QRS complex relationship, and also because the P waves follow on immediately (<70 msec) from the QRS complexes. These features suggest a tachycardia of supraventricular rather than ventricular origin. The P wave is dominantly positive at this "below atrial" level.

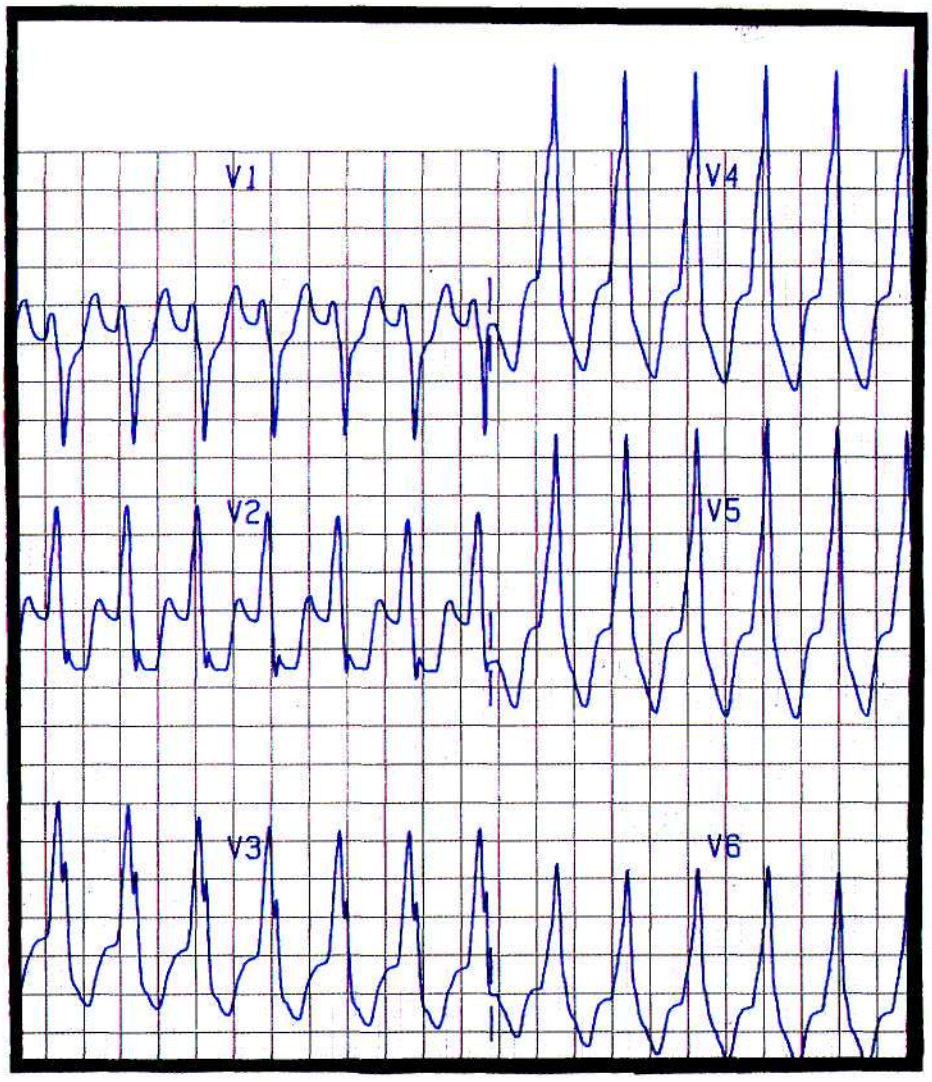
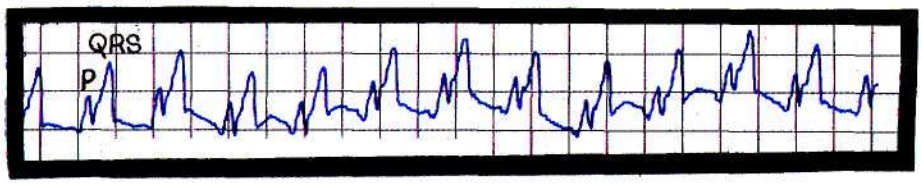


FIGURE 20. Surface electrocardiogram (above) showing a left bundle branch block pattern across V_1 to V_6. The oesophageal recording below demonstrates a spiky P wave for each QRS complex, denoting a supra-ventricular origin.



CHAPTER 5

DISCUSSION OF RESULTS

The present study has demonstrated a simple technique of oesophageal electrocardiography in the diagnosis of wide complex tachycardias in haemodynamically stable patients. This technique entails the use of a modified transvenous pacing electrode and an electrocardiograph machine, both of which are routinely available in local hospitals. Notably, the average duration of the procedure was 4.5 minutes and was accomplished with minimal discomfort to the patients. In 16 patients the electrode was swallowed quickly and easily; two patients needed reassurance and reintroduction after minimal discomfort. The recordings obtained were decipherable and enabled a revision or confirmation of the initial diagnosis in all patients. It must be noted that although the electrode imposes little discomfort, respiratory excursions and occasionally swallowing movements may cause wandering baselines. The experience in this study is that recordings done during breathholding at the end of expiration may curb these excursions. Other investigators using different equipment and techniques have not furnished information regarding the time taken for such procedures (Schnittger *et al.*, 1986; Copeland *et al.*, 1959; Kistin *et al.*, 1950; Hammill *et al.*, 1981). Furthermore, the equipment used by other investigators varied from sophisticated computers to specialized pill electrodes which are not usually available to local hospitals. Brevity aside, the simplicity of the equipment and technique outlined in this study suggests that it has potential for routine use amongst junior medical staff in medical wards and emergency departments.

Application of the technique described has also enabled an estimate of the optimal level for oesophageal recordings to be made. In this study, optimal recordings were obtained between 33,8 cm and 38,7 cm. In this respect, the data confirm the findings of Kistin *et al.* (1950). It must be noted that the P wave morphology on oesophageal electrocardiography will vary

depending upon the position of the recording electrode. It is apt to be biphasic at the optimal (atrial) level (Figure 12); Predominantly negative at a higher level (Figure 11), and predominantly positive at levels lower than the atrium (Figure 13).

Haemodynamic stability during wide QRS complex tachycardia in conscious adults is all too frequently interpreted as evidence for a supraventricular mechanism. Scott Millar (1989) suggests that teachers have given undue prominence to supraventricular tachycardia complicated by abnormal ventricular conduction. This study also confirms the existence of the bias (83% Initial diagnosis) towards the diagnosis of supraventricular tachycardia with aberrant conduction. Many physicians instinctively associate ventricular tachycardia with haemodynamic instability. It is very significant, therefore, that ventricular tachycardia was found to be the cause of 61% of regular wide QRS complex tachycardias in conscious, normotensive adults in this study. It is also evident from Table II that patients with ventricular tachycardia, despite a rapid rate and in some instances the presence of underlying heart disease such as congestive cardiomyopathy or ischaemic heart disease, were very tolerant of the tachycardia, as all patients were haemodynamically stable. The spectrum of arrhythmias described in these patients is not an artefact of a tertiary referral centre as these patients presented themselves to local general hospitals. Similar patterns were recently demonstrated in British and American general hospitals (Dancy *et al.*, 1985; Stewart *et al.*, 1986).

Graboys (1987) believes that the conscious adult with a heart rate exceeding 200 beats/minute has a supraventricular tachycardia, the converse (< 200 beats/minute) being true for ventricular tachycardia. In this study (Tables II and III), five of eleven patients with ventricular tachycardia had a rate exceeding 200 beats/minute, and four of seven patients with supraventricular tachycardia with aberrant conduction had a rate lesser than 200 beats/minute. Hence the data in this study refute this notion and confirms the findings of Wellens (1978). Apart from a clinical bias towards a diagnosis of supraventricular tachycardia with aberrant conduction, the

differentiation of wide complex tachycardias by twelve lead surface electrocardiograms is extremely difficult. The finding of atrioventricular dissociation for practical purposes should be interpreted as evidence for a ventricular tachycardia. However in some instances of ventricular tachycardia, ventriculoatrial conduction may occur in which case retrograde P waves will be visible after the QRS complexes on a 1:1 basis. These patients should be treated as for ventricular tachycardia (Wellens, 1987).

There remains concern that the practical diagnostic principles needed for differentiating mechanisms of wide complex tachycardia on a surface electrocardiogram may be difficult to apply at the level of the emergency department. Reasons that have been advanced for a high rate of misdiagnosis are :

i. an unfamiliarity with the diagnostic criteria (Dancy *et al.*, 1985).

ii. difficulty in discerning the different waveforms on a surface electrocardiogram (Wu *et al.*, 1975; Jenkins *et al.*, 1979; Hammill & Pritchett, 1981).

iii. the widely held notion that ventricular tachycardia must of necessity lead to haemodynamic instability (Morady *et al.*, 1985).

iv. A considerable bias towards the diagnosis of supraventricular tachycardia with aberrant ventricular conduction despite the fact that it is lesser common than ventricular tachycardia (Scott Millar, 1989).

A misdiagnosis rate of 39% was evident in a retrospective analysis of twelve lead surface electrocardiograms undertaken by Stewart *et al.* (1986). Dancy *et al.* (1985) showed that an alarmingly high misdiagnosis rate existed amongst interns, registrars, consultant physicians and consultant cardiologists. The potential for misdiagnosis is clearly confirmed in this prospective study, in which a total of 56% of the patients were misdiagnosed.

Adverse reactions following misdiagnosis in this study included hypotension in five patients, cardiac arrest in three patients and cardiac failure in two patients. In all instances, verapamil was administered intravenously in the erroneous belief that the mechanism was a supraventricular one with abnormal conduction. In a retrospective analysis of fifteen patients with misdiagnosed ventricular tachycardia, Stewart *et al.* (1986) showed that verapamil was given to thirteen patients, and similarly, all thirteen patients showed haemodynamic deterioration. Several adverse reactions to verapamil have been reported. These include asystole (Kounis, 1980); hypotension (Heng *et al.*, 1975; Lipman *et al.*, 1982; Tucker & Serote, 1984) and ventricular fibrillation (Buxton *et al.*, 1984). It is conceivable that it may cause peripheral vasodilatation (Singh & Roche, 1977; Angus *et al.*, 1976) and depress sinoatrial node activity (Carrasco *et al.*, 1978) accounting for the hypotension. Verapamil is also known to produce an increased heart rate by a baroreflex response to peripheral vasodilation (Angus *et al.*, 1976; Carrasco *et al.*, 1978), as well as exerting a depressant effect on the myocardium (Chew *et al.*, 1981; Lewis *et al.*, 1975). These mechanisms are the likely explanation for the haemodynamic deterioration in our patients.

Misdiagnosis may have other long-term sequelae, such as multiple hospitalizations with the attendant expenses and psychological trauma. In addition, misdiagnoses can conceivably produce long-term misdirected prophylactic drug therapy with unwanted side effects, recurrent episodes of recalcitrant arrhythmia and unnecessary costs. Adequate suppression of ventricular tachycardia is necessary to protect a patient from future major arrhythmic events. Careful prognostication can only follow upon the correct diagnosis of the mechanism of the arrhythmia.

CHAPTER 6

CONCLUSION AND RECOMMENDATIONS

6.1 CONCLUSION

i. The results confirm that misdiagnosis of wide complex tachycardia is common. This study also establishes that there is a considerable bias towards the diagnosis of a supraventricular tachycardia with aberrant conduction as a mechanism for the wide QRS complex tachycardia in patients who are haemodynamically stable.

ii. a. The study refutes the notion that the heart rate may be a useful differentiating feature for diagnosis.

b. The interpretation by the medical staff of the twelve lead electrocardiogram as a means to correctly diagnose wide complex tachycardias was commonly found not to be helpful as an aid to diagnosis.

iii. Serious adverse reactions following inappropriate therapy consequent upon misdiagnosis, are common.

iv. This study has demonstrated a simple technique of oesophageal electrocardiography that can be easily learnt by junior house staff and employed in the emergency room or general ward. This technique will assist in the bedside diagnosis of wide complex tachycardias. On average, optimal recordings of atrial activity in an adult are likely to be obtained between 33.8 and 38.7 centimetres.

6.2 RECOMMENDATIONS

i. In haemodynamically stable patients presenting with wide complex tachycardias, oesophageal electrocardiography as outlined in this study should be routinely performed prior to commencement of therapy.

ii. The use of verapamil should be strongly discouraged in any patient with wide complex tachycardia unless clinical, ECG and oesophageal lead criteria are all consistent with a diagnosis of supraventricular tachycardia. This conclusion is strongly supported by the authors experience of cardiac arrest following inappropriate prior use of verapamil in some patients with ventricular tachycardia in this series, as well as that reported elsewhere.

REFERENCES

A. JOURNALS

Angus JA, Richmond DR, Dhumma-Upakorn P, Cobbin LB, Goodman AH. Cardiovascular action of verapamil in the dog with particular reference to myocardial contractility and atrioventricular conduction. Cardiovasc Res 1976; 10: 623-632.

Butterworth S, Poindexter AC. The esophageal electrocardiogram in arrhythmias and tachycardias. Am Heart J 1957; 32: 681-688.

Buxton AE, Waxman HL, Marchlinski FE, Josephson ME. Electropharmacology of nonsustained ventricular tachycardia: Effects of class I antiarrhythmic agents, verapamil and propranolol. Am J cardiol 1984; 53: 738-744.

Carrasco HA, Fuenmayor A, Barboza JS, Gonzalez G. Effects of verapamil on normal sinoatrial node function and on the sick sinus syndrome. Am Heart J 1978; 96: 760-771.

Chew CYC, Hecht HS, Collett JT, McAllister RG, Singh BN. Influence of severity of ventricular dysfunction on hemodynamic responses to intravenously administered verapamil in ischaemic heart disease. Am J Cardiol 1981; 47: 917-922.

Copeland GD, Tullis IF, Brody DA. Clinical evaluation of a new esophageal electrode, with particular reference to the bipolar esophageal electrocardiogram. Part II: Observations in cardiac arrhythmias. Am Heart J 1959; 57(6): 874-885.

Coumel P, Leclercq JF, Attuel P *et al.* The QRS morphology in post myocardial infarction ventricular tachycardia. A study of 100 tracings compared with 70 cases of idiopathic ventricular tachycardia. Eur Heart J 1984; 5: 792-799.

Dancy M, Camm AJ, Ward D. Misdiagnosis of chronic recurrent ventricular tachycardia. Lancet 1985; ii: 320-323.

Della Bella P, Brugada P, Lemery R *et al.* A transcardiac lead system for identification and termination of supraventricular and ventricular tachycardia. Am J Cardiol 1987; 60(13): 1043-1050.

Enselberg CD. The oesophageal electrocardiogram in the study of atrial activity and cardiac arrhythmias. Am Heart J 1951; 41: 382-409.

Graboys TB. A practical approach to supraventricular tachycardia. Choices Cardiol 1987; 1: 114-116.

Greenspan AM. Ventricular tachycardia. Geriatrics 1986; 41(4): 67-73.

Hammill SC, Pritchett ELC. Simplified esophageal electrocardiography using bipolar recording leads. Ann Intern Med 1981; 95: 14-18.

Heng MK, Singh BN, Roche AGH, Norris RM, Mercer CJ. Effects of intravenous verapamil on cardiac arrhythmias and on the electrocardiogram. Am Heart J 1975; 90: 487-498.

Jenkins JM, Wu D, Arzbaecher RC. Computer diagnosis of supraventricular and ventricular arrhythmias. Circulation 1979; 60(5): 977-985.

Kindwall KE, Brown JP, Josephson ME. Electrocardiographic criteria for ventricular and supraventricular tachycardia in wide complex tachycardias with left bundle branch block morphology. J Am Coll Cardiol 1987; 9: 206.

Kindwall KE, Brown JP, Josephson ME. Electrocardiographic criteria for ventricular tachycadia in wide complex left bundle branch block morphology tachycardias. Am J Cardiol 1988; 61(5): 1279-1283.

Kistin AD, Brill WD, Robb GP. Normal esophageal and gastric electrocardiograms. Descriptions, statistical analyisis and bearing on theories of "electrocardiographic position". Circulation 1950; 2: 578-595.

Kounis NG. Asystole after veropamil and digoxin. Br J Clin Pract 1980; 34: 57-58.

Langendorf R. Differential diagnosis of ventricular tachycardia. Exp Med Surg 1959; 8: 228-239.

Lewis BS, Mitha AS, Gotsman MS. Immediate haemodynamic effects of verapamil in man. Cardiology 1975; 60: 336-376.

Lipman J, Jardine I, Roos C, Dreosti L. Intravenous calcium chloride as an antidote to verapamil-induced hypotension. Intensive Care Med 1982; 8: 55-57.

Marriot HJL. Differential diagnosis of supraventricular and ventricular tachycardia. Geriatrics 1970; 25: 91-102.

Massum RA, Tawakoll AA, Kistin AD. Reevaluation of electrocardiographic and bedside criteria for diagnosis of ventricular tachycardia. Circulation 1967; 36: 628-636.

Miles WM, Prystowsky EN, Heger JJ, Zipes DP. Evaluation of the patient with wide QRS tachycardia. Med Clin North Am 1984; 68(5): 1015-1038.

Morady F, Baerman JM, DiCarlo LA *et al*. A prevalent misconception regarding wide complex tachycardias. J Am Med Assoc 1985; 254: 2790-2792.

Prystowsky EN, Pritchett ELC, Gallagher JJ. Origin of the atrial electrocardiogram recorded from the oesophagus. Circulation 1980; 61(5): 1017-1023.

Robinson GC, Herrmann GR. Paroxysmal tachycardia of ventricular origin and its relation to coronary occlusion. Heart 1921; 8: 59-81.

Schnittger I, Rodriquez IM, Winkle RA. Esophageal electrocardiography: A new technology revives an old technique. Am J Cardiol 1986; 57: 604-607.

Scott Millar RN. "It looks like SVT" - the misuse of intravenous verapamil in broad complex tachycardia. S Afr Med J 1989; 76: 296-297.

Singh BN, Roche AHG. Effects of intravenous verapamil on hemodynamics in patients with heart disease. Am Heart J 1977; 94: 593-599.

Slocum J, Byrom E, McCarthy L, Sahakian A, Swiryn S. Computer detection of atrioventricular dissociation from surface electrocardiograms during wide QRS tachycardia. Circulation 1985; 72(5): 1028-1036.

Stewart RB, Bardy GH, Greene HL. Hemodynamic deterioration following inappropriate use of verapamil for wide complex tachycardia of unknown etiology. J Am coll Cardiol 1985; 5: 422.

Stewart RB, Bardy GH, Green HL. Wide complex tachycardia: misdiagnosis and outcome after emergent therapy. Ann Intern Med 1986; 104: 766-771.

Tucker RA, Serote WF. Parenteral-verapamil-induced sustained hypotension. Drug Intell Clin Pharm 1984; 18: 239-241.

Wellens HJJ, Bär FW, Lie KI. The value of the electrocardiogram in the differential diagnosis of a tachycardia with a widened QRS comples. Am J Med 1978; 64: 27-33.

Wellens HJJ, Brugada P, Heddle WF. The value of the twelve lead electrocardiogram in diagnosing type and mechanism of a tachycardia: a survey among 22 cardiologists. J Am Coll Cardiol 1984; 4(1): 176-179.

Wellens HJJ, Brugada P. Diagnosis of ventricular tachycardia from the 12-lead electrocardiogram Card Clinics 1987; 5(3): 511-525.

Wu D, Denes P, Leon FA, Chhablani RC, Rosen KM. Limitations of the surface electrocardiogram in the diagnosis of atrial arrhythmias. Circulation 1979; 60(5): 977-985.

B. BOOKS

Lewis T. The mechanism and graphic registration of the heart beat. 3rd Ed. London: Shaw & Sons, Ltd. 1925.

Wellens HJJ, Bär FW, Vanagt EJ *et al*: The differentiation between ventricular tachycardia and supraventricular tachycardia with aberrant conduction. The value of the 12-lead electrocardiogram. In: Wellens HJJ, Kulbertus HE, Eds. Whats new in electrocardiography? The Hague: Martinus Nijhoff, 1981: 184-199.

APPENDIX 1

CONSENT FORM

1. I NAME:

hereby consent to the following procedure and/or treatment being conducted on the person indicated in (4) below.

2. I acknowledge that I have been informed by:

concerning the possible advantages and possible adverse effects which may result from the abovementioned procedure and/or treatment and of the ways in which it is different from the conventional procedure and/or treatment. The information which I was given and which I acknowledge I understand is shown on the reverse side of this form.

3. I agree that the above procedure and/or treatment will be carried out and/or supervised by:

4. I acknowledge that I understand the contents of this form, including the information provided on its reverse and as the

SUBJECT	PARENT	GUARDIAN	OTHER (SPECIFY)

freely consent to the above procedure and/or treatment being conducted on:

NAME:

5. I am aware that I may withdraw my consent at any time without prejudice to further care.

SIGNED	SUBJECT:	DATE				
SIGNED	WITNESS:	DATE				
SIGNED	INFORMANT:	DATE				
SIGNED	RESEARCHER:	DATE				

* With the exception of the names and signatures in paragraphs 1, 4 and 5, please provide the above information.

12. INFORMATION GIVEN TO SUBJECTS

Please indicate what will be told to subjects in simple language. The procedure or treatment which will be applied should be described and reference should be made to possible side effects, discomfort, complications and/or benefits. It must be made clear to the patient that he/she is free at any time to withdraw without suffering any disadvantage.

NOTE: IF NO ANIMAL SUBJECTS ARE TO BE USED, PLEASE PROCEED TO PART D.