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Value of MCL_1, MCL_6, and Selected Leads in the Diagnosis of Wide QRS Complex Tachycardia

by

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VALUE OF MCL_1, MCL_6, AND SELECTED ECG LEADS IN THE DIAGNOSIS OF WIDE QRS COMPLEX TACHYCARDIA

PhD DISSERTATION

by

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ABSTRACT

VALUE OF MCL_1, MCL_6, AND SELECTED ECG LEADS IN THE DIAGNOSIS OF WIDE QRS COMPLEX TACHYCARDIA

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Use of the conventional precordial leads V_1 and V_6 has been suggested for distinguishing supraventricular tachycardia with aberrant ventricular conduction from ventricular tachycardia. For two decades, MCL_1 MCL_1 and MCL_6 have been widely used as V_1 and V_6 substitutes for bedside monitoring, but their use has never been validated. To determine the value of these bedside leads compared to their conventional lead counterparts, 121 wide QRS complex tachycardias were recorded from 92 patients during cardiac electrophysiology study with simultaneous MCL_1, V_1, MCL_6, V_6, and His bundle electrogram recordings. In addition, a full 12-lead electrocardiogram was recorded to evaluate selected leads hypothesized to be of value in making the diagnosis.

As determined from the His bundle recording, 31 of the tachycardias were supraventricular with aberration; 86 were ventricular. A majority of both normal and wide QRS complexes had identical patterns in MCL_6 and V_6. Although QRS morphology clearly differed between MCL_1 and V_1 in about one third of wide QRS complex tachycardias, no difference in diagnostic accuracy resulted.

A dissociated P wave during tachycardia, which was diagnostic of VT, was more likely to be observed in V_1 or MCL_1. A width of greater than 160 ms discriminated best between aberrant supraventricular and ventricular tachycardia. Axis deviation was a more useful criteria to diagnose ventricular tachycardias with a right, rather than a left bundle branch block contour.

A new criterion, measurement of QRS onset to the predominant peak or nadir of the complex, proved to be valuable in diagnosing wide QRS complex tachycardia in MCL_6 and V_6. An interval of

50 ms or less was suggestive of aberrant supraventricular tachycardia; an interval of 70 ms or more was virtually diagnostic of ventricular tachycardia.

Recommendations for clinical practice included the following. For single-channel monitors, leads MCL_1 and MCL_6 had the highest diagnostic accuracy; for dual-channel monitors, combined MCL_1/MCL_6 were superior to other two-lead sets. With the current inability to display two precordial leads simultaneously, there was no advantage in monitoring more than two leads.

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CHAPTER ONE: STUDY PROBLEM AND ITS SIGNIFICANCE

Introduction

Accurate differentiation between supraventricular and ventricular tachycardia (VT) is of obvious diagnostic and therapeutic importance. Several investigators have shown that the conventional 12-lead electrocardiogram (ECG) is valuable in distinguishing supraventricular tachycardia (SVT) with aberrant ventricular conduction from VT (Kindwall, Brown & Josephson, 1988; Wellens, Bär & Lie, 1978; Wellens, Bär, Vanagt, Brugada & Farré, 1981). For example, Wellens and associates (1981) demonstrated that if a wide QRS complex tachycardia were documented in all twelve leads, the arrhythmia could be correctly diagnosed (using the His bundle electrogram as the "gold" standard) about 92 percent of the time. Unfortunately, a limited number of electrocardiographic leads are available for continuous bedside monitoring of patients hospitalized in critical care units. Wide complex tachycardias in these patients are rarely captured on all twelve leads of a conventional ECG, particularly non-sustained tachycardias which terminate before an ECG can be acquired. Thus, the diagnosis and initial treatment of wide QRS complex tachycardia typically rests upon analysis of criteria from a single bipolar bedside monitoring lead.

The unipolar precordial leads V_1 and V_6 from a conventional 12-lead ECG have proven especially helpful in the diagnosis of wide QRS complex tachycardia (Kindwall et al., 1988; Sandler & Marriott, 1965; Swanick, LaCamera & Marriott, 1972; Wellens et al., 1978). Specifically, the unique contour of the prolonged QRS complex in these two leads often provides strong evidence of the impulse's supraventricular versus ventricular site of origin. During the past two decades, bipolar leads MCL_1 and MCL_6 have been routinely used as V_1 and V_6 substitutes for

continuous bedside monitoring, but the usefulness of these "modified" leads has never been critically validated.

The purpose of the present study was to determine the value of bedside leads MCL_1 and MCL_6 compared to their conventional ECG counterparts, V_1 and V_6, in diagnosing wide QRS complex tachycardia. An additional purpose was to evaluate selected bipolar and unipolar leads and lead sets hypothesized as valuable in making the diagnosis from the bedside. Specifically, the goal was to determine the lead with the greatest diagnostic accuracy for monitoring patients when only single-lead monitoring is available, and to determine whether multiple leads are necessary (and, if so, which ones), to make an accurate diagnosis.

An additional goal of the study was to test whether a new criterion, measurement of QRS onset to the predominant peak (or nadir) of the complex, would discriminate aberrant SVT from VT. It was hypothesized that bundle branch block aberration in patients without severe intramyocardial conduction disease should produce a wide complex with an initial sharp, rapid deflection resulting in a short interval from QRS onset to predominant peak/nadir. In contrast, initial activation of the ventricles from an ectopic ventricular focus should have a more slurred initial deflection.

Definition of Supraventricular and Ventricular Tachycardia

Cardiac impulses that arise from a site above the bifurcation of the bundle of His into the right and left bundle branches are designated as supraventricular in origin. Supraventricular foci include: (a) the heart's normal pacemaker, the sinoatrial (SA) node, (b) the atrial myocardium, and, (c) the atrioventricular (AV) node and the specialized conduction system above the bifurcation of the His Bundle. The supraventricular impulse divides at the bifurcation of the His bundle and

travels simultaneously to the right and left ventricles resulting in synchronous biventricular activation giving rise to a narrow (< 120 milliseconds (ms), QRS complex (Josephson, Waxman, Marchlinski, Horowitz & Spielman, 1981).

A normal, physiologic conduction delay and refractoriness built into the AV node allows time for atrial contraction to contribute to ventricular filling. Moreover, the delay acts to filter out excessively rapid supraventricular impulses before they reach the ventricles. For example, atrial fibrillation does not normally result in ventricular fibrillation; rather, only about 120-180 of the 400-600 impulses bombarding the AV node per minute are allowed passage to the ventricles. Consequently, supraventricular tachyarrhythmias are not generally life-threatening and are treated in a non-emergency fashion with drugs aimed at increasing the block at the AV node (to slow the ventricular rate), or terminating the arrhythmia.

In contrast to supraventricular rhythms, ventricular arrhythmias arise from a site distal to the bifurcation of the bundle of His. The ectopic impulse can arise from the specialized conduction system in the ventricles (i.e., the bundle branches, fascicles of the left bundle branch, or the Purkinje network), or the ventricular myocardium (Prystowsky & Zipes, 1984). Activation of the ventricles during ventricular arrhythmias proceeds in an unorthodox manner, spreading out from the site of impulse formation in one ventricle and subsequently depolarizing the second ventricle. Recent evidence suggests that in abnormal hearts where there are myocardial delays caused by scars, hypertrophy, and intramyocardial disease, the ectopic ventricular impulse often travels a circuitous route to the epicardial surface, with the point of epicardial breakthrough as far as six centimeters or more from the site of impulse origin (Josephson et al., 1981). Such activation of the ventricles produces a wide (> 120 ms) distorted QRS complex.

The ventricles are unprotected from excessively rapid rates when the impulse arises from below the AV node. Thus, VT can be extremely rapid and may accelerate into ventricular fibrillation causing sudden cardiac death if not immediately terminated. The majority of sudden arrhythmic deaths in cardiac patients occur from ventricular fibrillation. Recent documentation of the electrical events preceding sudden cardiac death which occurred during routine ambulatory monitoring revealed that ventricular fibrillation was initiated by VT in all fifteen cases (Pratt et al., 1983).

Misdiagnosis of Wide QRS Complex Tachycardia in Critical Care Units

The hallmark of ventricular rhythms is that they produce QRS complexes which are wide and distorted compared to the narrow QRS complexes seen during sinus rhythm or in arrhythmias arising from a supraventricular site. The diagnosis of VT would be an easy task except that occasionally SVT also has a wide, distorted QRS complex which closely mimics VT (Cohen, Lau, Stein, Young & Damato, 1968; Dressler & Roesler, 1952; Sandler & Marriott, 1965; Langendorf, 1951; Schrire & Vogelpoel, 1955). Although numerous research articles have been published over the past three decades delineating the electrocardiographic criteria for wide QRS complex tachycardia, recent reports indicate that misdiagnosis remains a common problem in clinical practice (Buxton, Marchlinski, Doherty, Flores & Josephson, 1987; Cooper & Marriott, 1989; Dancy, Camm & Ward, 1985; Morady et al., 1985; Stewart, Bardy & Greene, 1986).

The differential diagnosis of wide QRS complex tachycardia is more than an exercise in electrocardiography. It is a decision with important therapeutic and prognostic consequences. The most disastrous error is misdiagnosing VT as SVT. Failure to recognize and promptly treat VT may result in the patient's sudden death, negating the whole purpose of monitoring the patient in

a critical care unit. Although the patient may be successfully resuscitated, prolonged hypoxemia and hypotension during the resuscitation period may result in cerebral and myocardial ischemia and infarction as well as a myriad of other complications of cardiac arrest.

A population especially vulnerable to the misdiagnosis dilemma are patients with recurrent sustained VT. These patients are frequently evaluated in emergency rooms or other critical care units during tachycardia and they may be conscious and hemodynamically stable. Although VT may result in hemodynamic deterioration and loss of consciousness, it also may be associated with little change in blood pressure and minimal or no neurological symptoms (Morady et al., 1985). Moreover, the recurrent VT patient may be treated with antiarrhythmic agents which slow the rate of the tachycardia so that it does not result in hemodynamic deterioration.

Absence of dramatic clinical signs and symptoms during VT may lead to the erroneous conclusion that the tachycardia is supraventricular in origin. Morady and colleagues (1985) asked physicians to diagnose an ECG which contained all the classic features of VT in a case study questionnaire. Fifty three percent of the 196 physicians misdiagnosed VT as SVT with bundle branch block. Nearly 60 percent stated that they were persuaded by the patient's near-normal blood pressure and clinical status when attempting to differentiate VT from SVT. Morady concluded that more emphasis should be placed on electrocardiographic findings than on the patient's blood pressure or clinical status. Cooper and Marriott (1989) replicated Morady's study to test critical care nurses' ability to distinguish between VT and SVT. Of the 2,521 responders, 1,962 (78%) misdiagnosed VT as SVT with aberrant ventricular conduction.

The danger in misdiagnosing VT as SVT lies in the administration of drugs which may make the patient much worse. Verapamil, the drug of choice for converting many SVTs to sinus rhythm,

is not successful in treating most VTs (Belhassen & Horowitz, 1984; Sung, Shapiro, Shen, Morady & Davis, 1983). Verapamil is a myocardial depressant and such negative inotropic action on the heart may destabilize an already tenuous situation, leading to hemodynamic collapse (Stewart et al., 1986). Stewart and co-workers (1986) reviewed the diagnosis and management of 46 patients with wide QRS complex tachycardia presenting emergently whose tachycardia mechanisms were subsequently established by intracardiac electrograms. Eight of the tachycardias were SVT with aberration and 38 were VT. Fifteen of the 38 VTs (39%) were misdiagnosed as SVT at the time initial therapy was instituted. Verapamil was administered to patients in 13 of the 15 episodes of misdiagnosed VT, and hemodynamic deterioration resulted in all 13. The outcomes included hypotension requiring vasopressors, acceleration of the tachycardia, degeneration of VT into ventricular fibrillation requiring defibrillation, and asystole following cardioversion.

Buxton and colleagues (1987) reviewed the records of 129 patients referred to their emergency room for the evaluation of sustained VT during a three-year period. Twenty-seven of these patients received intravenous verapamil based on the interpretation that the tachycardia was SVT with aberrant ventricular conduction. Forty-four percent of the patients who received verapamil developed acute severe hypotension or loss of consciousness necessitating immediate cardioversion. These authors concluded that the use of verapamil to differentiate SVT with aberrant conduction from VT was hazardous.

Despite these poor outcomes, verapamil still continues to be recommended by some as a diagnostic tool to differentiate wide QRS complex tachycardia (Morgan & Brennan, 1985). Dancy and colleagues (1986, p. 897) argued against using verapamil in this manner stating: "In the case described by Morgan and Brennan (1985), it should have been possible to diagnose VT from the

(ECG) recording illustrated (in their article) using currently accepted criteria. . . . We believe that established criteria for distinguishing between SVT and VT should have been applied before choosing a potentially dangerous treatment."

The second error is misdiagnosing SVT as VT. This error also can lead to poor outcomes, although they are generally less serious than those resulting from failure to recognize VT. First, it delays initiation of appropriate therapy for SVT aimed at slowing and ultimately converting the tachycardia. Patients with poor left ventricular function may not tolerate atrial fibrillation, atrial flutter, or other SVTs with rapid ventricular rates. Second, it conveys to the patient and family that a life-threatening situation is at hand. In addition to being emotionally traumatic for the patient, the unwarranted anxiety can stimulate a sympathetic nervous system response resulting in elevated plasma levels of catecholamines which may increase the rate of the SVT. Third, lidocaine, (the standard treatment for VT in critical care units), given to patients in certain SVTs may cause sudden acceleration of the ventricular rate with resultant hemodynamic deterioration (Adamson & Spracklen, 1968; Marriott & Bieza, 1972). Marriott and Bieza (1972) reported a case in which a patient with atrial flutter and variable AV conduction with an average ventricular rate of 165 received a single bolus of lidocaine. The ventricular rate promptly accelerated to 265 with the development of 1:1 conduction across the AV node. Lidocaine may cause acceleration of the ventricular rate in atrial fibrillation or flutter by slowing of the atrial rate enabling the AV node to conduct all the ectopic atrial impulses (Hayes, Ettinger, Wanat & Killip, 1967; Rosen, Lau, Weiss & Damato, 1970).

The misdiagnosis dilemma in critical care units occurs for several reasons. First, although some electrocardiographic criteria for making the differential diagnosis have been well established,

other criteria are still in the process of being investigated with new criteria being continually reported. Moreover, the latest research findings are not always in agreement. For example, disagreement exists regarding the importance of identifying left axis deviation in diagnosing VT (Caceres et al., 1987; Kindwall et al., 1988; Wellens et al., 1978). Second, critical care practitioners, who make the initial diagnosis and institute treatment, may be unaware of the well-established electrocardiographic criteria as well as the more recent research findings. Third, as Morady and co-workers (1985) point out, the critical care practitioner may be fooled by the patient's near-normal blood pressure and clinical status during VT, ignoring important electrocardiographic clues. Fourth, there may be inadequate electrocardiographic information available to make the correct diagnosis. For example, the diagnosis often requires comparison of tachycardia and baseline QRS complexes, and previous ECGs taken during sinus rhythm may be unavailable. Moreover, the tachycardia may be recorded by a limited number of leads, or by leads which are unhelpful in making the diagnosis. For example, the diagnosis requires close scrutiny of the wide QRS complexes in the unipolar precordial leads V_1 and V_6, as well as determination of QRS axis by the examination of several limb leads (Wellens et al., 1978).

Limitations of Continuous Bedside Electrocardiographic Monitoring

A conventional 12-lead ECG consists of three bipolar leads (leads I, II, and III), and nine unipolar leads (aVR, aVL, AVF, and the six precordial or V leads). Six leads are designated limb leads because they have electrodes placed on the limbs (leads I, II, III, AVR, AVL, and AVF). The bipolar limb leads, the first leads to be introduced, have two drawbacks (Marriott, 1983). First, each is derived from two points distant from the heart. Second, the three electrodes are all in a single (frontal) plane of the body. In the 1930s, it was discovered empirically that if all three

limb electrodes were connected through resistances of 5000 ohms each to form a common central terminal, while the "exploring" electrode was placed closer to the heart and moved around the bend of the thorax, unique views of the heart could be obtained (Marriott, 1983). The advantage of these six unipolar precordial leads (V_1-V_6) was that they were close to the heart and that the "indifferent" electrode which was hooked up to the common central terminal had virtually no influence on the tracing.

Current practice generally relies on a single bipolar ECG lead for continuous monitoring and rhythm analysis which is often inadequate for making the diagnosis of wide QRS complex tachycardia (Mirvis et al., 1989). With the introduction of bedside monitoring in the 1960s, no attempt was made to use a particular lead. The common practice was to move two electrodes around the chest until a sizeable P wave and QRS complex were obtained. This practice resulted in impressive quantities of arrhythmic information being lost. In 1970, Marriott and Fogg recommended a simple bipolar hook-up requiring only three electrodes placed on the chest which produced a tracing similar to the patient's V_1 tracing from a conventional ECG (Figure 1). They called the lead MCL_1 for the following reasons: (a) M stands for "modified". The lead is not a "true" unipolar V_1 because it is bipolar with the negative electrode on the left infraclavicular space influencing the tracing as well as the positive electrode. (b) CL stands for chest and left arm positions. (c) The subscript 1 indicates the position of the chest electrode in the V_1 position. Although Marriott's simple hook-up does not allow for displaying the two precordial leads simultaneously, a modified V_6 lead (MCL_6) can be obtained by exchanging two electrodes.

Although it has become increasingly standard practice in critical care units in the United States to monitor patients on MCL_1 or MCL_6, a significant number of units continue to monitor

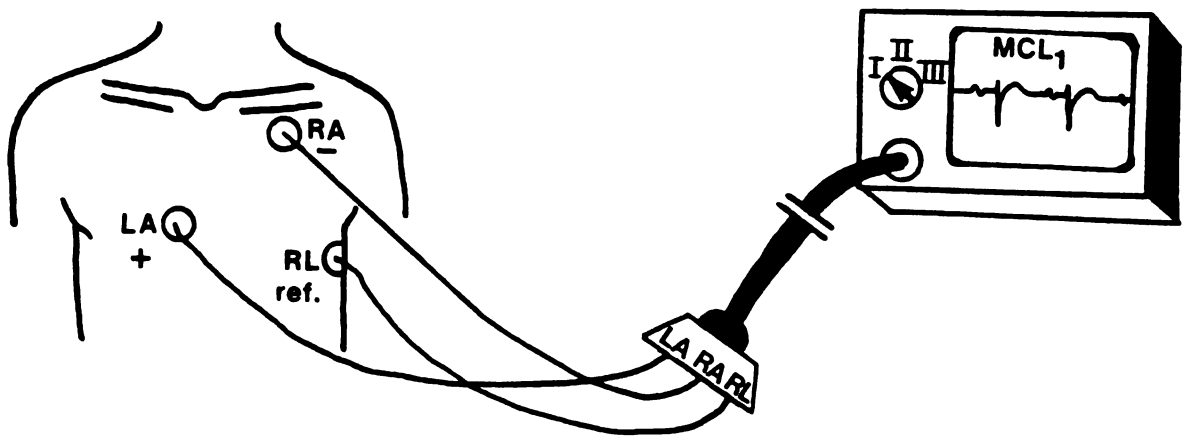


Figure 1. Electrode placement for continuous bedside monitoring with MCL_1. The selector dial on the bedside monitor is placed on lead I. The positive left arm (LA) electrode is placed in the fourth intercostal space to the right of the sternum, the negative right arm (RA) electrode is placed in the left infraclavicular space, and the ground right leg (RL) electrode is placed in the V_6 position. To obtain MCL_6, the selector dial is turned to lead II.

patients with a single bipolar lead (typically lead II) (Marriott & Gozensky, 1982). In units that do monitor patients with the modified precordial leads (i.e., MCL_1 and MCL_6), an assumption is made that the recordings are identical to the patient's conventional precordial leads (i.e., V_1 and V_6) in terms of QRS morphology. The modified leads were conceived in theory and confirmed in practice by noting a close similarity between a patient's V_1 and V_6 recordings from a conventional ECG machine and their MCL_1 and MCL_6 recordings from the bedside monitor. Marriott recalls (personal communication, 1986), that about 45 men out of 50 had MCL_1 recordings identical to V_1; however this was not quantified nor systematically documented in a study.

Therefore, it is unclear whether the characteristic QRS patterns suggestive of aberrant SVT or VT observed in V_1 and V_6 are also present in MCL_1 and MCL_6.

Since about 1980, bedside monitoring manufacturers have provided multichannel bedside monitors with the capability of displaying two, three, and four leads simultaneously. Such multichannel systems have the capability of displaying one unipolar precordial lead and one or more limb lead simultaneously. Unfortunately, no bedside monitoring systems provide for the simultaneous display of two unipolar precordial leads such as V_1 and V_6. For example, in order to monitor V_1 and V_6 simultaneously at the bedside with current multichannel monitors, two five-electrode cables would have to be attached to the patient. One cable's chest electrode would be placed in the V_1 position; the second cable's chest electrode would be placed in the V_6 position. A total of ten electrodes would be required (two electrodes on each extremity and two on the chest). Moreover, each bedside would have to be equipped with two separate ECG monitors. This hook-up is impractical for numerous reasons: (a) the patient's mobility is severely curtailed, (b) the problems of muscle artifact are multiplied especially when the patient moves extremities, e.g., brushing teeth, eating, getting up to the commode or bedside chair, (c) the ten electrodes are difficult to maintain and expensive to replace, (d) the multiple electrodes and their cables may get in the way during resuscitation and other procedures, and, (e) the additional ECG monitors and leads required at each bedside are costly. Although current technology is unavailable for monitoring simultaneous unipolar precordial leads (i.e., V_1 and V_6), it *is* possible to monitor simultaneous bipolar precordial leads (i.e., MCL_1 and MCL_6) (Figure 2).

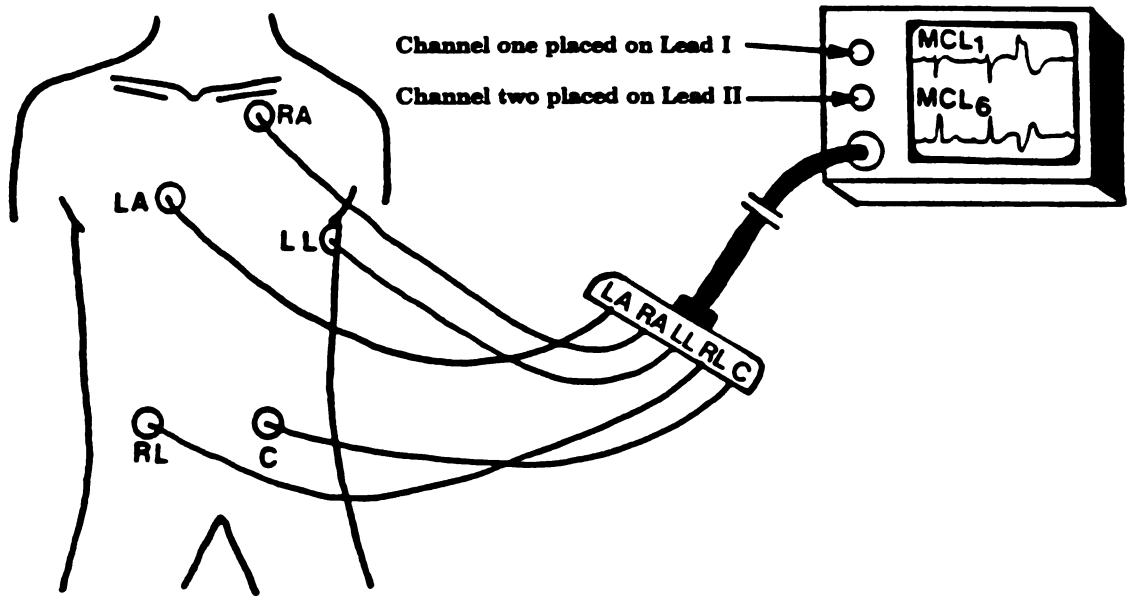


Figure 2. Electrode placement for simultaneous monitoring of MCL_1 and MCL_6 leads in dual-channel bedside monitors. With the selector dial on channel one placed on lead I, the left arm (LA) is the positive electrode, the right arm (RA) is the negative electrode, and the remaining three are reference (ground) electrodes. The resultant lead on channel one is the bipolar precordial lead, MCL_1. With the selector dial on the second channel placed on lead II, the left leg (LL) is the positive electrode, the RA remains the negative electrode, and the remaining three are reference electrodes. The resultant lead on channel two is the bipolar precordial lead, MCL_6. Note that the right leg (RL) and chest (C) electrodes can be placed anywhere on the body since they are reference electrodes in this system.

A recent random sample survey conducted by this author of 300 staff nurses employed in critical care units in the United States indicated that 52 percent of the units had single-channel bipolar monitors, 39 percent had dual-channel monitors, and, nine percent had both. A single lead II was the first choice (70%) for single-channel monitoring, and lead II plus V_1 was the lead set of choice (92%) for dual-channel monitoring. None of the 300 respondents were monitoring with dual MCL_1/MCL_6 leads.

In summary, electrocardiographic information necessary to make a definitive diagnosis of wide QRS complex tachycardias from the bedside may be unavailable in critical care units for the following reasons: (a) The arrhythmia may not be captured on a conventional 12-lead ECG so that QRS morphology in V_1 and V_6 and QRS axis from several limb leads can be determined. Although the nurse commonly attempts to obtain a 12-lead ECG when a patient develops a wide QRS complex tachycardia, the arrhythmia may no longer be present by the time the ECG machine is attached. (b) Previous ECGs taken during sinus rhythm may not be available for comparison. (c) Leads selected for continuous monitoring may be unhelpful in distinguishing aberrant SVT from VT (e.g., a single lead II), (d) If the tachycardia is recorded by the traditional bipolar system monitoring a single modified precordial lead, typically only one precordial lead is recorded (either MCL_1 or MCL_6), unless the tachycardia is sustained long enough for the nurse to change leads. Moreover, it is unclear whether the diagnostic criteria established from the unipolar precordial leads can be generalized to the modified precordial leads. In addition, no information from limb leads is available for determination of QRS axis. (e) If the arrhythmia is documented by multichannel monitors that record one unipolar precordial lead and one or more limb leads, information is still missing from the second precordial lead. Thus, the critical care practitioner may be forced to make a diagnosis and initiate treatment with inadequate information.

CHAPTER TWO: CONCEPTUAL FRAMEWORK AND LITERATURE REVIEW

Conceptual Framework

Cardiac rhythm refers to the electrical activation of the myocardium which produces rhythmic muscular contraction of the heart. The electrical impulse is generated by specialized pacemaker cells which do not require stimulation from an external source. Hence, these cells are said to be automatic or to exhibit "automaticity." The electrical current is caused by movement of charged particles; namely, sodium, potassium, and calcium ions, back and forth across the cell membrane. This ionic fluctuation creates an electrical field that is distributed throughout the body, producing electrical potentials radiating out to the body surface. When two electrodes are placed on the body surface and connected through an appropriate amplifier, a graphic recording or ECG of the intracardiac events can be obtained. Thus, the phenomenon of cardiac rhythm is closely linked to the phenomenon of electrocardiographic pattern. Cardiac rhythms are the human responses themselves, whereas electrocardiographic patterns are the visual representations of these responses.

The advent of the ECG in the 1930s brought about a revolution in the analysis of cardiac rhythm. Prior to the ECG, cardiac rhythm was assessed indirectly by checking the pulse, blood pressure, skin color and temperature, urine output, sensorium, and all the physiological indexes of hemodynamic functioning. Without electrical activity of the heart there can be no mechanical activity; however, a wide variety of cardiac rhythms occur with no measurable change in hemodynamics and which can be detected only by the ECG.

Links to Central Concepts in Nursing

How does the phenomenon of cardiac rhythm fit within the context of nursing? Nursing is defined as the diagnosis and treatment of human responses to actual or potential health problems

(ANA, 1980). The responses of individuals which are of interest to nursing are as numerous as the human organism is complex; they include physiological, psychological, and socio-cultural responses. Nurses believe that man is greater than the sum of his parts; thus, they emphasize a holistic approach in caring for or studying man. Nurses are also interested in man's interaction with his environment. Health restoration and promotion is a goal shared with a number of health professionals; however, it has been a central goal in nursing from the writings of Florence Nightingale in the mid-1800s (Meleis, 1985). Nurses believe health is more than the absence of disease; however, the concept of health is illusive, often value-laden, and there is currently no consensus in the discipline as to its definition (Hall & Allan, 1986).

Nurse theorists and meta-theorists generally agree on the central concepts within the domain of nursing (Table 1). Yura and Torres (1975) reported a National League for Nursing survey of baccalaureate nursing programs which identified four major concepts: man, society, health, and nursing. Fawcett (1978) agreed that the four concepts were central to nursing but substituted the word person for man to avoid sexism and environment for society. Flaskerud and Halloran (1980) state that there is a consensus among nurses on the importance of the following domain concepts: "Person as an entirety is addressed holistically or as having parts broadly identifiable as biologic, psychologic, and socio-cultural. Person as a patient or candidate for nursing actions is defined as ill, healthy, or both healthy and ill. Nursing actions are defined as what nurses do for and with the person as patient. Environment, interacting with the person or coextensive with the person, is defined as a source of or an influence on the health or illness of the person" (p. 3). Newman (1983) asserts that the "domain of nursing has always included the nurse, the patient, the situation in which they find themselves, and the purpose of their being together, or the health of the patient"

Table 1
Areas of Agreement Regarding Central Concepts in the Nursing Domain

	INDIVIDUAL	ENVIRONMENT	HEALTH	NURSING	TRANSITIONS
Yura & Torres (1975)	man	society	health	nursing	-----
Fawcet (1978)	person	environment	health	nursing	-----
Flaskerud Halloran (1980)	person/ patient	environment	health/ illness	nursing action	-----
Newman (1983)	patient/ client	environment	health	nursing action	-----
Chinn & Jacobs (1983)	client	environment	-----	nursing action	-----
Kim (1983)	client	environment	-----	nursing action	-----
Stevens (1984)	person/ patient	-----	health	nursing acts	-----
Meleis (1985)	client	environment	health	nursing process therapeutics intera-ction	transitions

(p. 388). As is evident in Table 1, all theorists agree on the central concepts of individual and nursing; however, some omit health (Chinn & Jacobs, 1983; Kim, 1983; Stevens, 1984) and Stevens (1984) omits environment although she talks about interrelationships between nursing acts, the patient, and health. Meleis (1985) introduces a "candidate" central concept, transitions.

The individual and cardiac rhythm. There are four major areas of focus in the study of individuals: (a) physiological factors, (b) psychodynamic factors, (c) personal traits, and, (d) role performance, i.e., functional status (Weiss 1986). Analysis of cardiac rhythm yields considerable information about the physiological functioning of the individual. Fisch (1980) says of the ECG: "It is of recognized value, serves as a standard of excellence, is traditional, enduring, . . .and, thus fits the criteria to be called a classic" (p. III-1). He emphasizes that the ECG serves as an independent marker of heart disease, reflects anatomic, metabolic, and hemodynamic alterations, allows the electrocardiographer to deduce complex electrophysiologic concepts though deductive reasoning, is a stimulus for laboratory confirmation of postulated mechanisms and concepts, is vital for proper diagnosis and therapy, and, is without peer for the diagnosis of arrhythmias.

In addition to abnormalities seen with primary cardiac disorders, abnormal cardiac rhythms occur in response to numerous metabolic imbalances or other systemic causes (Greenbaum, 1977). These "secondary" arrhythmias can be due to such things as: (a) electrolyte imbalances, (b) acid-base abnormalities, (c) hypoxemia, (d) elevated intracranial pressure, (e) vasovagal stimulation as in nausea and vomiting, suctioning, endotracheal intubation, bronchoscopy, or rectal manipulation, (f) anemia, (g) dehydration, hypovolemia, (h) body positional changes including orthostatic changes, (i) fever, sepsis, (j) mechanical stimulation from intracardiac catheters such as temporary pacemakers, central venous catheters, and pulmonary artery catheters, and, (k) numerous medications and drug toxicities.

The second area of interest in studying individuals focuses on psychodynamic features. Cardiac rhythm responses are not only influenced by physiological changes; they are profoundly influenced by emotions such as anxiety, fear, pain, worry, and rage. Moreover, such reactions can

be evoked simply by recalling unpleasant experiences or thoughts. Poets of all ages have regarded the heart as the seat of the emotions; "stout-hearted" and "faint-hearted" are part of our modern language. That emotion has a profound effect on the action of the heart is a concept that, no doubt, precedes recorded history. Primitive man, hiding from a lion in the jungle experienced the same pounding in his chest that modern man experiences when undergoing an important interview. Emotional stressors, perceived by the brain, arouse the sympathetic nervous system resulting in surges of potent stimulatory adrenal hormones causing increased heart rate and susceptibility to arrhythmias (Bishop & Reichert, 1970). The psychodynamic field is closely linked with the construct of environment because it is often perceived environmental stressors which trigger the sympathetic-adrenal response.

The third focus of person research described by Weiss (1986) is the trait field. It remains to be seen whether study of cardiac rhythm will shed any light on personality traits. Friedman and Rosenman (1959) popularized the term "type A" to describe behavior connoting time urgency, impatience, hostility, competitive drive; the sense of an effort-oriented person caught up in a joyless struggle. These researchers reported that type A behavior was associated with an increased risk for developing atherosclerotic heart disease and have proposed several possible mechanisms.

Type A behavior is often attended by the aforementioned cardiac excitatory responses with excesses in sympathetic-adrenal activity; thus, it is likely that study of coronary prone behavior will include analysis of cardiac rhythm (Eliot & Buell, 1982). Studies of circumstances preceding the onset of life-threatening arrhythmias suggest that intense emotional states, often involving anger, are associated with arrhythmia onset in approximately 20 percent of the cases (Reich, 1985). The

role of psychodynamic factors in the pathogenesis of sudden cardiac death is an exciting area for future research.

The fourth focus of person research is role performance or functional status (Weiss, 1986). Cardiac rhythm responses to exercise are used to assess a person's functional capacity, prescribe safe pulse rate limits for exercise, and to evaluate recovering myocardial infarction patients' readiness for hospital discharge. Heart rate is the most easily measured of all indexes of myocardial oxygen consumption (Kirk & Jennings, 1982). The product of heart rate times systolic blood pressure, termed "double product," correlates even closer with myocardial oxygen consumption and is used to interpret exercise ECG stress tests (Froelicher, 1982). The concept of functional status is closely linked with the construct of health. According to Smith (1981), many peoples' notion of health is based on the common sense criteria of whether the person has the physical capacity to do their job.

The environment and cardiac rhythm. Environment is a more illusive construct than individual, and the two are inextricably interwoven, making separate study of either impossible (Rogers, 1970). Physiologists commonly conceive of environment as the physicochemical conditions surrounding living cells (Guyton, 1976). The famous 19th century French physiologist, Claude Bernard, popularized the notion of the *milieu interieur* or internal environment which consists of the extracellular fluid bathing cells (Bernard, 1974). The extracellular fluid provides a stable physicochemical environment supplying oxygen and nutrients and eliminating metabolic end products. The semi-permeable cell membrane allows for continuous interaction between the cell and its environment. Abnormalities in the internal environment surrounding myocardial cells

such as hypoxia, electrolyte and acid-base disorders, lack of nutrients, and presence of toxic substances can trigger cardiac arrhythmias.

To the social scientist, environment encompasses all that surrounds the individual; for example, social systems, society, culture, family, the patient's room, the nurse, etc. Moos (1979) reduced the infinite number of relevant environmental variables influencing health outcomes into four groups: (a) the physical setting, (b) the organizational factors, such as hospital size, (c) the characteristics of the inhabitants of a particular environment, such as the numerous health care personnel involved in a critical care unit patient's welfare, and, (d) the social climate which includes relationships, personal growth and goals, and the system's stability and flexibility to change. Precise definition of a person's environment is complicated by the fact that what matters is the environment as it is *perceived* rather than as it exists in objective reality and that environment extends far beyond the immediate situation or the people with whom the person interacts on a face-to-face basis (Bronfenbrenner, 1979).

A large number of animal experiments have implicated psychological stress in the precipitation of arrhythmias and sudden death (Eliot & Buell, 1982). For example, Gelhorn (1967) demonstrated that the combination of arousal with enforced helplessness and extreme conflicting stimulation resulted in simultaneous activation of the sympathetic and parasympathetic nervous systems constituting a neurophysiological basis for the development of cardiac arrhythmias. Moreover, several reports have documented the effects of psychological stress in lowering the threshold for ventricular fibrillation and sudden death both in animals and in humans (Engel, 1978; Lown, DeSilva & Lenson, 1978; Lynch, Paskewitz, Gimbel & Thomas 1977). Although the link

between emotions and cardiac arrhythmia remains controversial, the ability of the human mind to control physiological functions is acknowledged.

Measurement of cardiac rhythm responses provides a unique opportunity to look at the complex interplay of physiological, emotional, and environmental factors at a particular point in time. Cardiac rhythm responses are dynamic, ever-changing, with almost no lag time between perceived stimulus and heart rhythm response. The rationale for the lie detector test is based on the premise that cardiac rate is not under voluntary control; thus, the criminal may deny the crime, but his cardiac rhythm responses will expose him. The advantage of using cardiac rhythm responses as a dependent variable in nursing research is that the individual is unlikely to withhold responses under involuntary control, whereas the subject may withhold responses under voluntary control, (e.g., in an interview or questionnaire).

Nursing therapeutics and cardiac rhythm. Of the four groups of relevant environmental variables described by Moos (1979), one closely linked to the phenomenon under discussion is social climate which includes nursing therapeutics. The most frequent use of electrocardiographic data by nurse researchers, for example, is to evaluate the safety or efficacy of various nursing interventions and routines (Drew, 1989b). Similarly, in practice, critical care nurses use excessive increases in heart rate or development of ectopic beats and rhythms to judge whether the patient sitting up in the chair is becoming fatigued and should return to bed. Nurses decide between bedpans, bedside commodes, or bathroom privileges based in part upon cardiac rhythm responses. Physicians frequently defer such patient care decisions to the nurse because the nurse becomes familiar with the patient's continuous electrocardiographic patterns and trends. In addition, nurses use the ECG to assess the degree of importance the patient attaches to the situation in which he

finds himself. For example, the nurse may perceive, based upon observation of changes in cardiac rate and rhythm, that a young, high-strung, male executive with an acute myocardial infarction is enraged over his lack of control and that it would be safer to allow him to control his visiting hours and telephone calls.

An important aspect of nursing therapeutics closely linked to cardiac rhythm is monitoring or surveillance. The term "monitor" comes from the Latin *monere*, meaning one who reminds or warns; an overseer; to watch, observe, or check, especially for a special purpose; to keep track of, regulate, or control (Gove, 1986). The term "surveillance" derives from the Latin word *vigilare* and means a close watch kept over a person; to post a vigilance (Gove, 1986). Monitoring of critically-ill patients requires a depth of knowledge, experience, and cognitive ability unlike any other services nurses provide. What separates the novice from the expert practitioner is that the former is more task-oriented and the latter is more surveillance-oriented. Changes in cardiac rhythm are often the first clue that the patient's condition has changed.

The American Nurses' Association's definition of nursing addresses *potential* and *actual* health problems. Nursing care for the patient with a potential cardiac rhythm disorder includes constant monitoring/surveillance, assessment of arrhythmogenic risk, and removal of arrhythmogenic factors, (e.g., treatment of pain and anxiety, regulation of activity, promotion of adequate oxygenation, nutrition, hydration, acid-base and electrolyte balance, etc.) Nursing care for the patient with an actual cardiac rhythm problem consists of two phases. The first decision-making phase includes documentation, analysis, and physical assessment. The second phase consists of actions such as resuscitation, administration of antiarrhythmic agents, manipulation of settings on devices such as temporary pacemakers, notification of the physician, and evaluation of treatment.

The establishment of coronary care units in the 1960s brought about a revolution in nursing as the responsibility for diagnosis and treatment of cardiac arrhythmias shifted to nurses. Karliner (1981) states: "the nurse remains the cornerstone of care in the prevention and treatment of dysrhythmias. Even when 'round-the-clock medical coverage is present (and this is often not the case in intermediate-sized and smaller institutions), the coronary care unit nurse must be skilled in cardiac resuscitation and recognition of potentially dangerous dysrhythmias. . . .It is often the routine procedure for nurses to institute resuscitative measures and make critical decisions regarding the nature and treatment of dysrhythmias for prolonged periods, often on the basis of optional standing orders which are implemented at their discretion" (p. 85).

Society places a high value on the surveillance role of nurses. Critical care patients and their families frequently express their relief at having highly-specialized nurses monitoring the situation around the clock. Newly-admitted acute myocardial infarction patients often are afraid to go to sleep fearing they will die in their sleep. These patients feel immensely reassured when they are told that a nurse will be watching their heartbeat on a minute-to-minute basis at the nurses' station. In her keynote address at the 15th Annual Western Council on Higher Education for Nursing Research, Barnard (1982) poses: "I question whether the assessment/diagnostic activity that nursing engages in is one of the more important nursing acts. We are dealing many times with patients who are in transition, who need to be watched, where changes are the basis of their response to the health problem. I propose that in the future we need to clarify whether the dominant behavior in nursing is monitoring and diagnosis, and, if so, I think this gives impetus to the focus of nursing research" (p. 11).

Health and cardiac rhythm. There is currently no consensus in nursing as to a definition of health. Although nurses reject the biomedical tendency to focus on elimination of signs and symptoms of disease, the ANA definition focuses on health *problems* rather than on health per se. Contrary to their own definition, however, the writers of the policy statement define health as "a dynamic state of being in which the developmental and behavioral potential of an individual is realized to the fullest extent possible" (Hall & Allan, 1986, p. 318). Smith (1981) defines health as having four aspects: (a) the clinical aspect considers health to be an absence of disease, (b) the role performance aspect interprets health as the capacity to perform social roles, (c) the adaptive aspect considers health as flexibility in interacting with the environment, and, (d) the eudaemonistic aspect defines health as exuberant well-being. Cardiac rhythm responses to exercise are used to evaluate functional capacity. For example, it is strongly recommended that persons whose job entails the safety of the public such as commercial airline pilots or fire-fighters undergo a maximal exercise ECG test to insure their capability to perform under physical and emotional stress. The same test is used to predict the presence of coronary artery disease which relates to Smith's clinical aspect of health. The phenomenon does not appear to be closely linked with either adaptive or eudaemonistic health.

Transitions and cardiac rhythm. In addition to the four central nursing concepts of individual, environment, health, and nursing, Meleis (1985) proposes "transitions" be considered an important domain concept. Meleis expands Bronfenbrenner's (1979) "ecological transitions," which refers to shifts in role or setting occurring throughout the life span, to include situational and health/illness events as well.

Changes in cardiac rhythm are markers of transition. For example, premature ventricular contractions and spontaneous ventricular fibrillation during myocardial infarction mark the physicochemical instability in the internal environment occurring with an interruption of blood flow to the myocardium (Kirk & Jennings, 1982). Similarly, a marker for the opening up of a previously-occluded coronary artery by thrombolytic therapy is the sudden appearance of "reperfusion arrhythmias" (Keith, Fox, Bergmann & Sobel, 1985).

Changes in cardiac rhythm herald transitions into acute illness (e.g., sinus tachycardia seen in an acute asthmatic attack), transitions from rest to exercise, transitional changes during sleep (e.g., increased heart rate and premature ectopic beats during REM sleep), transition from emotional calm to stress, anxiety, fear, rage, or other emotions. Cardiac rhythm responses to exercise are used as a marker of stages of recovery in the myocardial infarction or cardiac surgical patient. Cardiac rhythm abnormalities signal imminent cardiac arrest 15 seconds or more before the patient has symptoms. This early warning of impending death has resulted in early treatment and saving of lives in critical care units.

Conceptual Model

A model depicting the relationships between cardiac rhythm and the central domain concepts of individual, environment, health, transitions, and nursing therapeutics is shown in Figure 3 (Drew, 1989a). The benefits to nursing research that will result from the study of cardiac rhythm are numerous. First, measurement of cardiac rhythm provides an objective, quantifiable, noninvasive, inexpensive, readily accessible, reliable, and valid tool for measuring human responses. Second, it provides information on physiological and psychological responses and the mind-body interaction. Third, it can be used to study humans across the life span including prenatal life. Fourth, it can

be used to assess the importance of various environmental stressors and influences on the person. Fifth, it can be used to evaluate the recovery process in cardiac surgical or myocardial infarction patients. Last, it can be used to evaluate the safety or efficacy of various nursing interventions and routines.

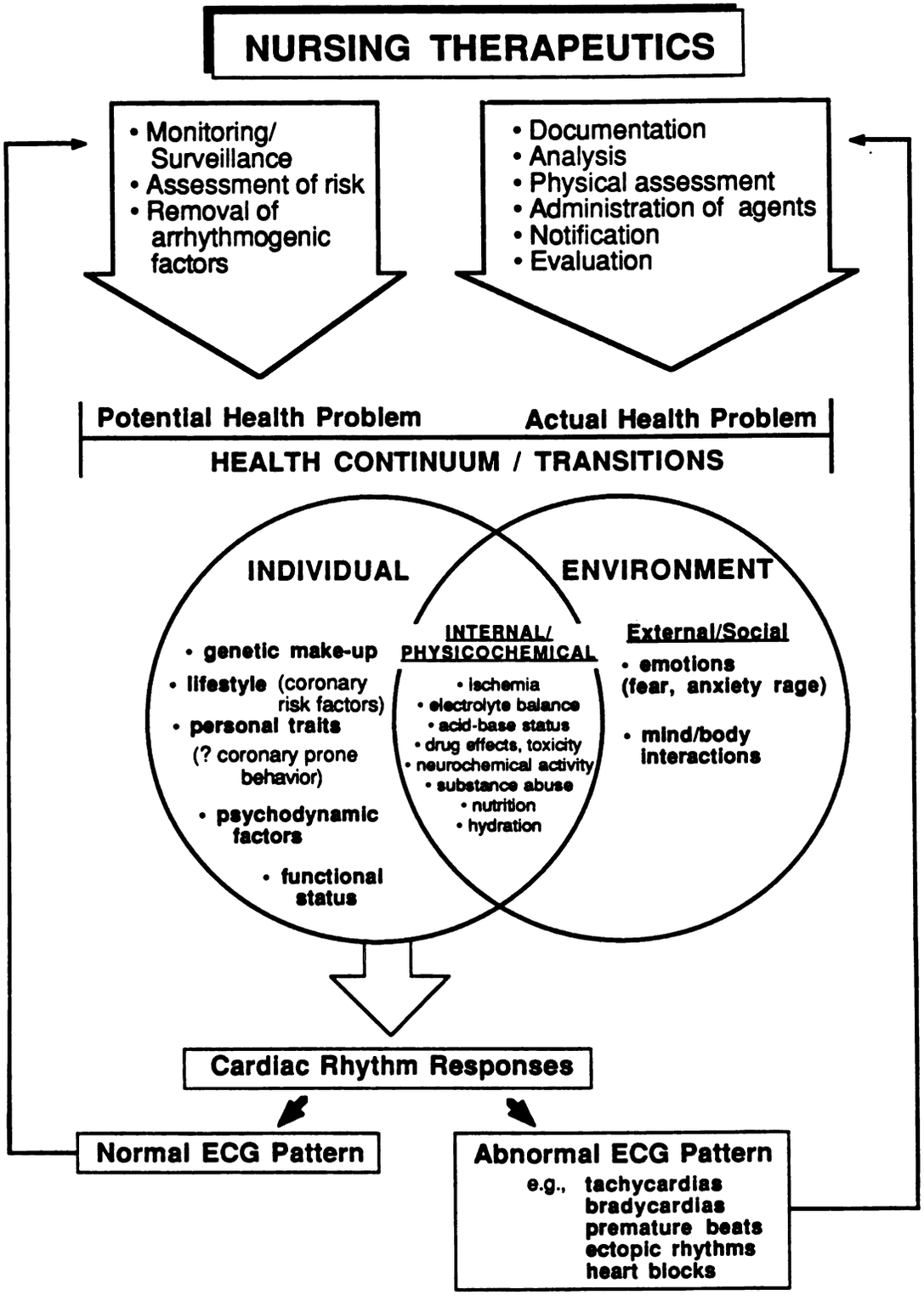


Figure 3. Relationships between cardiac rhythm and central concepts in nursing.

Review of Literature

Tachycardias with a Wide QRS Complex

The differential diagnosis of wide QRS complex tachycardia includes: (a) SVT with preexistent bundle branch block, (b) SVT with aberrant ventricular conduction, (c) SVT with antegrade conduction over an anomalous accessory AV pathway or nodoventricular pathway, and, (d) VT (Wellens, Bär, Vanagt, Brugada & Farré, 1981) (Figure 4).

SVT with Preexistent Bundle Branch Block

The bundle branches are part of the specialized His-Purkinje conduction system of the heart located in the intraventricular septum. If one of these branches of the bundle of His is blocked by disease, the impulse travels down the healthy branch to the contralateral ventricle first. Subsequently, the impulse gets "derailed" from the conduction system and spreads through myocardium to activate the ipsilateral ventricle. Depolarization of first one ventricle and then the other takes longer than simultaneous biventricular activation. Moreover, more time is required for spread of the impulse through myocardium rather than the specialized conduction system; hence, the prolonged QRS complex.

The characteristic triphasic QRS morphology of right bundle branch block is best observed from a right precordial lead (MCL_1 or V_1) because the exploring electrode in the fourth right intercostal space is located near the right ventricular free wall. Consequently, delayed right ventricular activation which occurs in right bundle branch block produces a primarily positive wide QRS complex with a large R′ wave in these leads (Figure 5A). The characteristic monophasic QRS morphology of left bundle branch block can be observed in a right or left precordial lead (Figure 5B). Delayed left ventricular activation in left bundle branch block produces a negative,

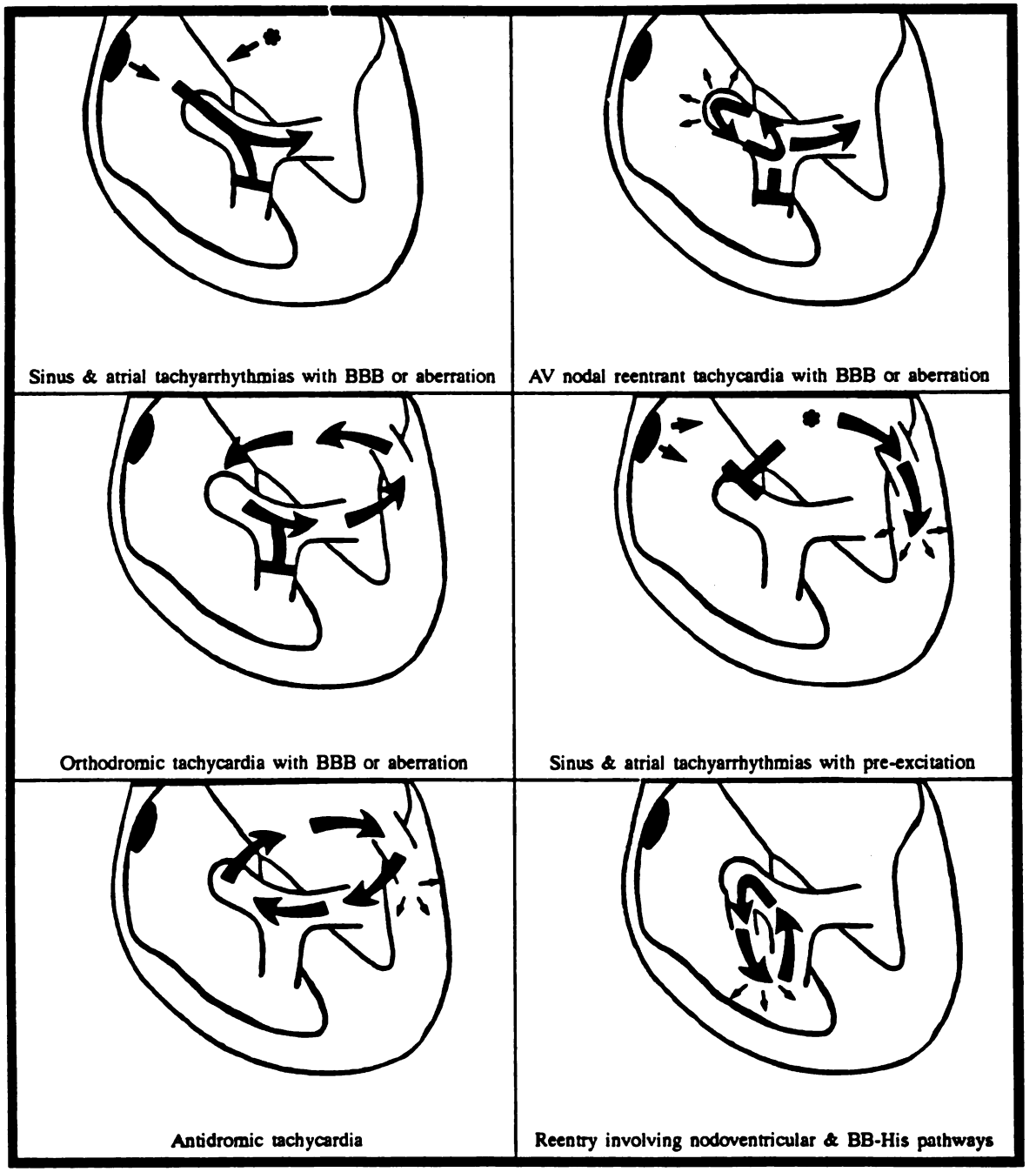


Figure 4. Mechanisms of various supraventricular tachyarrhythmias with a wide QRS complex. BBB = bundle branch block; BB-His = bundle branch-His bundle; AV = atrioventricular

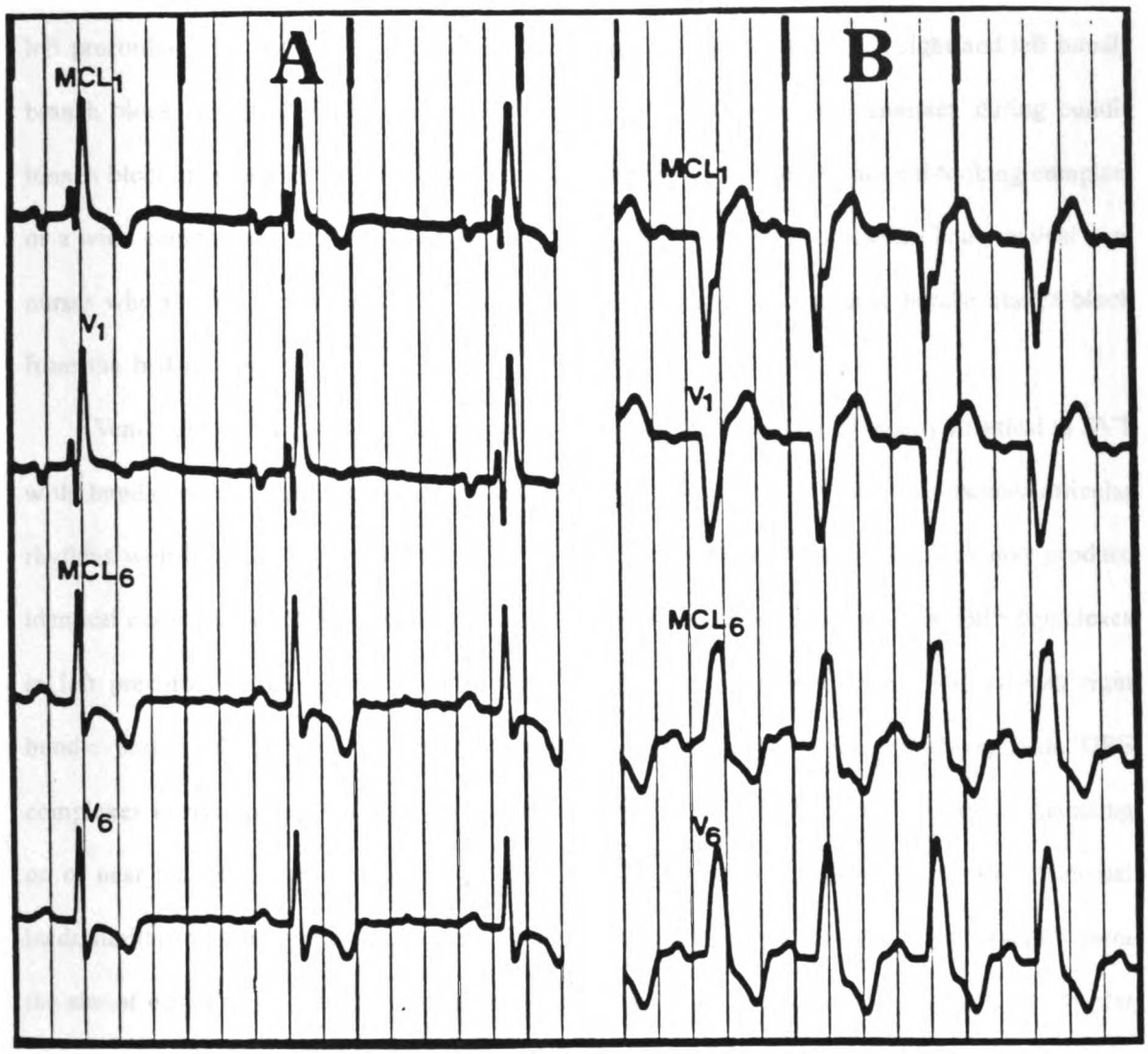


Figure 5. Characteristics of right and left bundle branch block in the conventional and modified precordial leads. Vertical time lines of 0.20 second are indicated on this and subsequent tracings. The primarily positive, wide triphasic rsR′ pattern of right bundle branch block (tracing A) is evident in the right precordial leads (MCL_1 and V_1). The QRS complex in the left precordial leads (MCL_6 and V_6), however, is deceptively narrow with the delayed right ventricular activation represented by a small amplitude S wave. Thus, right bundle branch block is easy to overlook when monitoring with MCL_6 or V_6. The QRS pattern of left bundle branch block (tracing B), is easy to detect in either right or left precordial leads. It produces a negative, wide QS or Rs complex in MCL_1 or V_1 and a positive, wide monophasic R wave in MCL_6 or V_6.

wide QRS complex in right precordial leads (MCL_1, or V_1), and a positive, wide QRS complex in left precordial leads (MCL_6, V_6, or lead I). It is important to emphasize that right and left bundle branch block cannot be diagnosed from lead II. Part of the wide QRS complex during bundle branch block may be isoelectric in lead II, which may result in a narrow, normal-looking complex, or a wide complex which is not predictive of which bundle branch is blocked. Thus, critical care nurses who routinely monitor their patients in lead II are unable to diagnose bundle branch block from the bedside monitor.

Ventricular tachycardia can produce wide QRS complexes which look nearly identical to SVT with bundle branch block. For example, both right ventricular rhythms and supraventricular rhythms with left bundle branch block have delayed left ventricular activation which may produce identical negative, wide QRS complexes in right precordial leads and positive, wide QRS complexes in left precordial leads. Both left ventricular rhythms and supraventricular rhythms with right bundle branch block have delayed right ventricular activation producing positive, wide QRS complexes in right precordial leads (Figure 6). In some cases, left ventricular rhythms originating on or near the intraventricular septum produce negative, wide QRS complexes in right precordial leads similar to right ventricular rhythms. In these cases, the impulse travels a circuitous path from the site of origin on or near the septum to an epicardial breakthrough point on the right ventricular wall, activating the right ventricle prior to the left ventricle (Josephson et al., 1981). Thus, right ventricular beats mimic left bundle branch block, and left ventricular beats can mimic right or left bundle branch block.

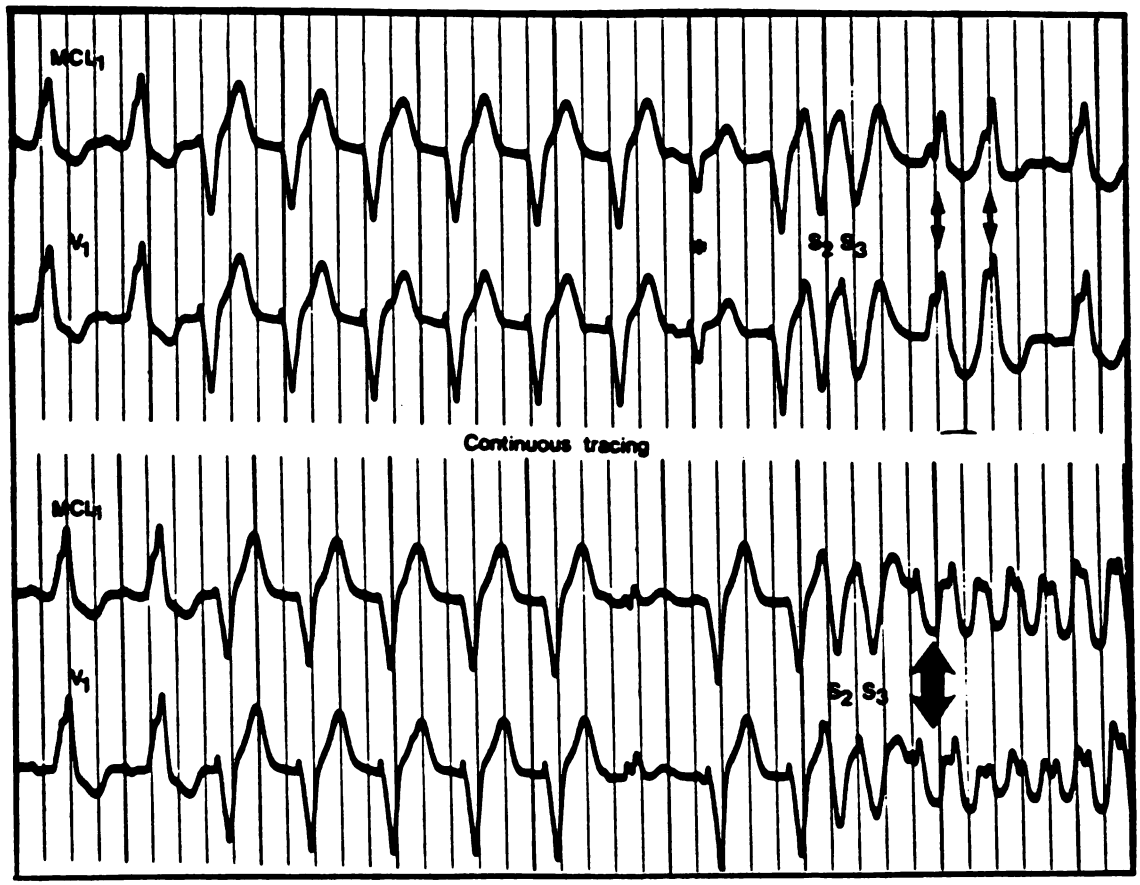


Figure 6. Comparison of QRS morphology in supraventricular rhythms with bundle branch block and ventricular rhythms. The first two beats are sinus beats with right bundle branch block. The QRS complex in these beats shows a wide, monophasic R wave with a taller right peak which is a variant of the characteristic triphasic contour during right bundle branch block. The next ten beats originate from the right ventricle and are paced beats. The first eight beats are paced at the basic drive rate of 100 beats per minute with the last two beats paced prematurely (S_2 and S_3). The seventh paced beat (*) is a fusion beat which simulates normal intraventricular conduction. Such normalization of the QRS complex occurs when there is fusion between a supraventricular impulse with bundle branch block and an impulse from the ipsilateral ventricle (i.e., right bundle branch block and right ventricle). Notice that the right ventricular beats have an identical negative, wide QS contour to that of supraventricular rhythms with left bundle branch block. Two left ventricular premature beats (small arrows) are induced which closely mimic the right bundle branch block contour of sinus rhythm. Following three sinus beats, the drive is repeated and rapid VT with a right bundle branch block contour is initiated (large arrows).

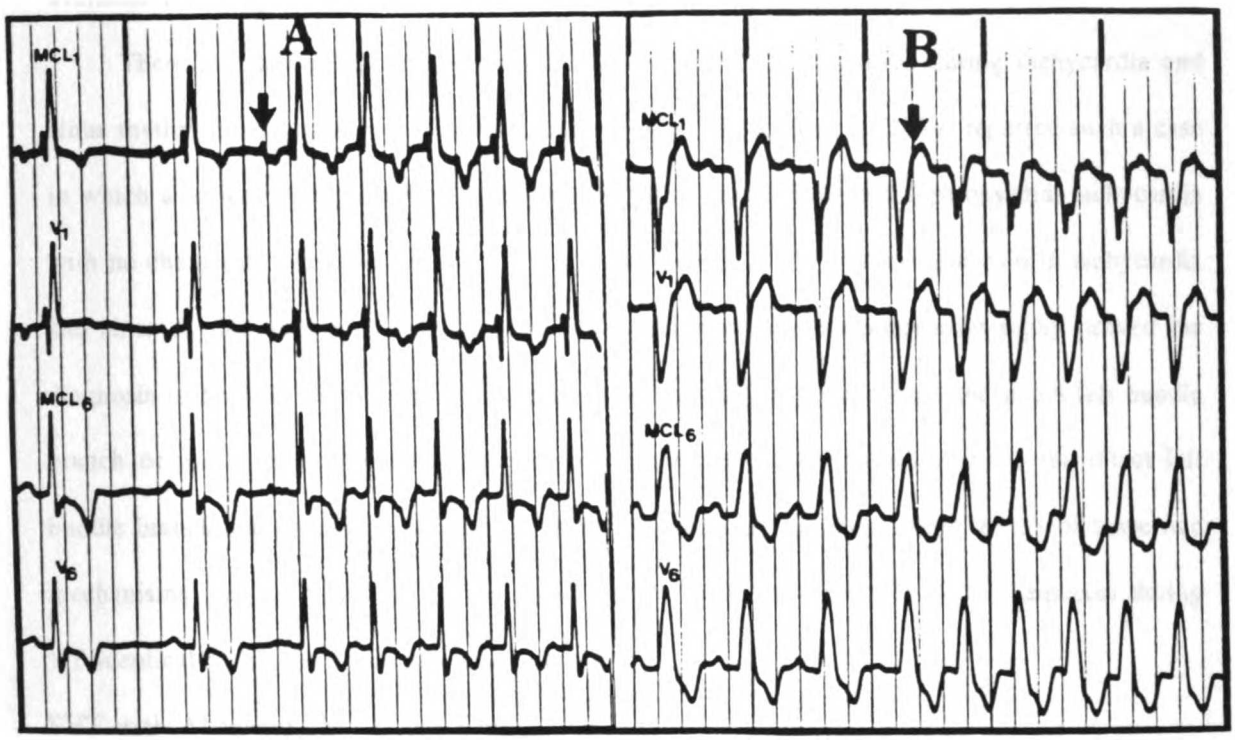


Figure 7. Initiation of atrial overdrive pacing (arrow) during sinus rhythm with right bundle branch block (tracing A) and left bundle branch block (tracing B) illustrates that the correct diagnosis of supraventricular tachycardia can be made by observing QRS complexes during tachycardia identical in morphology to those of sinus rhythm. To be certain that the complexes during baseline and tachycardia are not just coincidentally identical in one or two leads, however, a comparison of QRS morphology should be made in multiple leads.

Supraventricular tachycardia with preexisting bundle branch block is easy to diagnose because an identical wide QRS complex is also present during baseline sinus rhythm (Figure 7). Thus, if the wide QRS complex during tachycardia is clearly different from baseline rhythm with bundle branch block, the tachycardia is VT (Dongas et al., 1985). The diagnosis can be difficult, however,

when a patient presents emergently with a wide QRS complex tachycardia and no prior ECGs are available for comparison.

There are rare exceptions to the rule of identical QRS complexes during tachycardia and sinus rhythm indicating a supraventricular origin. Ross and associates (1979) reported such a case in which a patient with preexistent right bundle branch block developed a paroxysmal tachycardia with no change in QRS morphology. Although the similarity of QRS configuration in tachycardia and sinus rhythm suggested a supraventricular origin, His bundle electrocardiography proved the diagnosis to be VT. These authors speculated that the VT originated from the intact left bundle branch or was a case of bundle branch reentry with antegrade conduction down the intact left bundle branch and retrograde conduction up the blocked right bundle branch. Either of these two mechanisms (i.e., fascicular or bundle branch reentry VT) could produce QRS complexes during VT identical to SVT with bundle branch block.

SVT with Aberrant Ventricular Conduction

A second group of SVTs which may be indistinguishable from VT are those with aberrant ventricular conduction (Marriott, 1970; Marriott & Thorne, 1971; Massumi, Tawakkol & Kistin, 1967; Sandler & Marriott, 1965; Wellens et al., 1978). Aberrant ventricular conduction (also called ventricular aberration, aberrancy, or functional bundle branch block) is a temporary and benign bundle branch block brought on by an abrupt change in the cardiac cycle length, e.g., a premature beat or paroxysmal tachycardia. The bundle branches, like all parts of the electrical conduction system of the heart, have a dormant period following response to an electrical stimulus. This dormant period, called the refractory period, is a time during which the bundle branches do not respond to another stimulus. If the length of the required refractory period is slightly different for

the right and left bundle branches, a short window of time exists when one of the bundle branches is ready to conduct another impulse while the other is unable to respond. Thus, the bundle branch which is taken "by surprise" does not conduct the impulse and the QRS takes the shape of a right or left bundle branch block.

The length of the refractory period of the bundle branches changes dynamically on a beat-to-beat basis and is dependent upon the previous cycle length. The longer the preceding R-R interval, the longer the subsequent refractory period; the shorter the preceding R-R interval, the shorter the subsequent refractory period. Thus, aberrancy usually occurs in QRS complexes which end a short cycle preceded by a long cycle, because this situation makes it likely that one of the bundle branches will be refractory to the descending impulse (Figure 8).

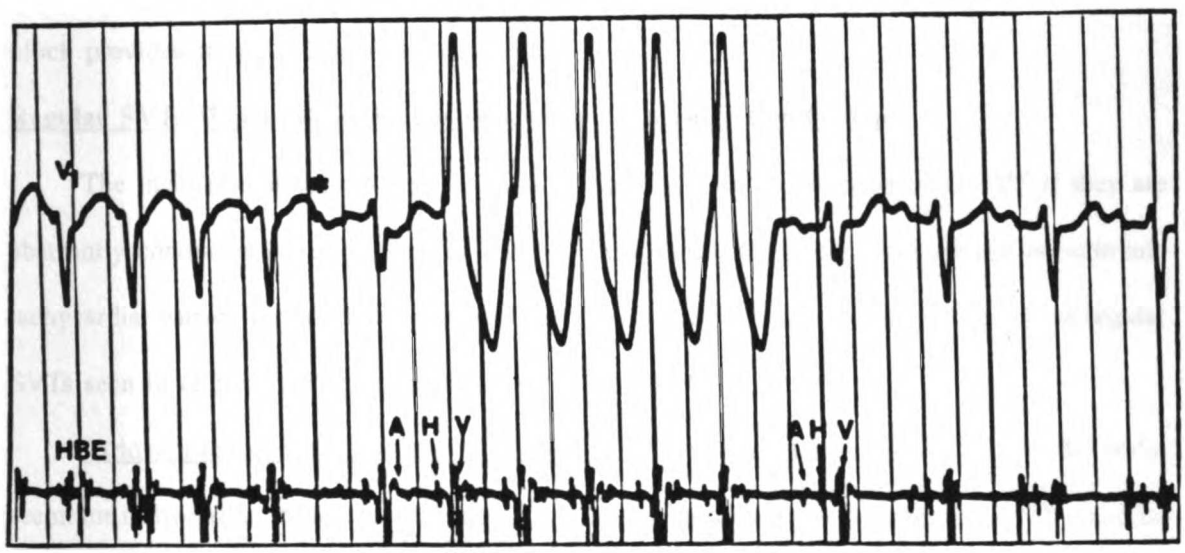


Figure 8. The top tracing is recorded from the surface lead V_1. Atrial overdrive pacing at a rate of 150 beats per minute has 1:1 conduction to the ventricles except for one cycle (*) which has 2:1 conduction across the AV node. This longer cycle produces aberrant conduction when 1:1 conduction resumes simulating ventricular tachycardia. Atrial pacing is terminated and sinus rhythm takes over at a rate of about 100 beats per minute. Notice that the H-V interval during the aberrant beats is equal to that of sinus rhythm proving a supraventricular origin of the anomalous beats. HBE = His bundle electrogram; A = atrial depolarization; H = His bundle depolarization; V = ventricular depolarization.

Right bundle branch block aberration during SVT is much more common than is left bundle branch block aberration. The incidence of aberration which exhibits a right bundle branch block contour reported in various studies ranges from 63 to 94 percent (Cohen et al., 1968; Gulamhusein, Yee, Ko & Klein, 1985; Kulbertus, de Leval-Rutten & Casters, 1976; Marriott & Sandler, 1966; Sandler & Marriott, 1965; Wellens et al., 1981). Normally, a supraventricular impulse descends the left bundle branch ahead of the right, resulting in activation of the left side of the septum before the right. Because the left bundle branch is depolarized first, it is also repolarized first, and ready to conduct a new impulse a few milliseconds before the right bundle branch. Therefore, the bundle branch most likely to be taken "by surprise" by an early impulse is the right bundle branch. Thus, the characteristic triphasic QRS contour (rsR′ in V_1 and Qrs in V_6) of right bundle branch block provides strong evidence for aberration.

Regular SVTs Which May Manifest Aberrant Ventricular Conduction

The incidence of various regular SVTs that are difficult to distinguish from VT if they are aberrantly conducted is summarized in Table 2. Atrioventricular nodal reentrant and orthodromic tachycardia warrant further discussion because together they comprise nearly 90 percent of regular SVTs seen in clinical settings (Josephson & Seides, 1979).

Atrioventricular nodal reentrant tachycardia. The most common regular SVT is AV nodal reentrant tachycardia. Moe and colleagues (1956) were the first to demonstrate that SVT could be produced by longitudinal dissociation of dual AV nodal pathways, setting up a reentrant circuit. One of the dual pathways has fast conduction with a long refractory period; the other pathway has slow conduction with a short refractory period. Typically, SVT is produced when an atrial premature beat blocks in the fast pathway (due to its longer refractory period), while it is conducted

Table 2
Mechanisms of Regular Supraventricular Tachycardia in 150 Patients (Josephson & Seides, 1979)

AV nodal reentrant tachycardia	58%
Orthodromic tachycardia	30%
Atrial tachycardia	8%
SA nodal reentrant tachycardia	4%
Antidromic tachycardia	0%

antegradely through the slow pathway. If slow conduction exceeds the refractory period of the fast pathway, the impulse can reexcite the fast pathway retrogradely, completing the reentrant circuit. This common form of AV nodal reentrant tachycardia produces P waves which are buried in the QRS, or P waves closely following the QRS (Josephson & Seides, 1979; Wu et al., 1978). An uncommon form of AV nodal reentrant tachycardia circulates in the opposite direction, producing P waves preceding the QRS complex. Since the atria are activated in a retrograde fashion during AV nodal reentrant tachycardia, P waves, if visible, are inverted in the inferior leads.

As might be expected, AV nodal reentrant tachycardia is less likely to have aberrant ventricular conduction than other SVTs because it is usually initiated when the impulse "jumps" over to the slow AV pathway. Hence, the long interval the impulse requires to traverse the slow AV pathway practically insures that both bundle branches will be able to conduct the impulse.

Orthodromic tachycardia. The second most common regular SVT is orthodromic tachycardia, which may occur in patients with anomalous AV pathways (e.g., the Wolff-Parkinson-White or WPW syndrome). Orthodromic tachycardia is a macro-reentrant tachycardia in which the impulse travels antegradely down the AV node and retrogradely up the accessory AV pathway. This

produces a normal, narrow QRS complex unless there is preexistent bundle branch block or aberrant ventricular conduction.

In patients with WPW syndrome, the normal sinus impulse may travel through both the AV node and accessory pathway, which causes the ventricular myocardium to be activated before it would have been had the impulse traveled exclusively through the normal AV nodal pathway. This "preexcitation" of the ventricles produces a widened QRS complex; the slurred upstroke of which is known as the "delta" wave. In diagnosing paroxysmal SVT, the observation of preexcited QRS complexes in ECGs taken prior to the tachycardia should lead one to suspect a macro-reentrant mechanism involving the accessory AV pathway. Some AV bypass tracts are capable of retrograde conduction only. This is called a "concealed" bypass tract because the QRS complex during sinus rhythm never exhibits the telltale delta wave. Concealed bypass tracts can also sustain an orthodromic tachycardia because antegrade conduction across the accessory pathway is not required for such a tachycardia.

The presence of AV block, retrograde ventriculo-atrial block, or AV dissociation excludes orthodromic tachycardia because both the atria and ventricles are a requisite part of the reentrant circuit. The ventricles must be activated before the impulse can travel retrogradely through the accessory pathway to activate the atria. Therefore, P waves are inscribed following the QRS complex. Since the atria are depolarized in a retrograde fashion during orthodromic tachycardia, P wave morphology is abnormal. The polarity of P waves in various leads will depend upon the accessory pathway's location. For example, a left free wall pathway produces an inverted P wave in lead I during orthodromic tachycardia.

SVT with Antegrade Conduction Over an Accessory Pathway

A third group of SVTs which are indistinguishable from VT on the surface ECG are those with antegrade conduction over an anomalous accessory pathway. Such a pathway can be located in several places connecting the atrial and ventricular myocardium (AV bypass tract) or connecting the AV node and ventricular myocardium (nodoventricular or Mahaim fibers). Three types of tachycardias may have antegrade conduction over an anomalous accessory pathway: (a) sinus tachycardia and atrial tachyarrhythmias (atrial tachycardia, flutter, fibrillation) with preexcitation; (b) antidromic tachycardia, and, (c) reentrant tachycardia with antegrade conduction over a nodo-ventricular pathway and retrograde conduction over the bundle branch-His system (Wellens & Brugada, 1987).

Atrial tachyarrhythmias with preexcitation. During atrial tachyarrhythmias, the degree of preexcitation may vary depending on the relative contributions of the normal and abnormal pathways. Thus, if the impulse travels exclusively through the AV node, a normal, narrow QRS complex results. In contrast, if the impulse travels exclusively through the anomalous pathway, maximal preexcitation with a wide, distorted QRS complex results. If the impulse travels across both pathways, the QRS complex will exhibit varying degrees of fusion. In contrast to the AV node, the refractory period of the accessory pathway behaves like muscle and shortens with decreasing cycle lengths (Josephson & Seides, 1979). Thus, the degree of preexcitation may become greater at faster heart rates because the AV node becomes refractory while the accessory pathway conducts readily.

During maximal preexcitation, ventricular activation proceeds from the end of the accessory pathway which inserts directly into ventricular myocardium. The resultant wide QRS complex is indistinguishable from VT originating from the same site.

A fairly common cause of wide QRS complex tachycardia in WPW Syndrome is the onset of atrial fibrillation with antegrade conduction over the accessory pathway (Gallagher, Pritchett, Sealy, Kasell & Wallace, 1978). Numerous published case studies of atrial fibrillation in patients with WPW erroneously diagnosed as VT misled researchers to believe for years that VT was irregular (Fleishman, 1952; Levine & Beeson, 1941; Palatucci & Knighton, 1944). Now it is appreciated that extremely rapid (greater than 220) and grossly irregular tachycardias with wide QRS complexes are more likely to be atrial fibrillation with preexcitation than VT (Wellens & Brugada, 1987).

Antidromic tachycardia. A second and extremely rare cause of wide QRS complex tachycardia in WPW syndrome is antidromic tachycardia, which is a macro-reentrant tachycardia with antegrade conduction over the accessory pathway and retrograde conduction through the AV node. In reference to the rarity of this type of tachycardia in clinical settings, Gallagher and colleagues (1978, p. 289) state that "while an antidromic mechanism is theoretically plausible, the differential diagnosis of such a tachycardia should embrace other possibilities." Because the reciprocating impulse travels exclusively down the accessory pathway during antidromic tachycardia, the resultant QRS complex manifests maximal preexcitation, and, thus, cannot be distinguished from VT on the surface ECG.

Reentrant tachycardia involving a nodoventricular pathway. A third and infrequent cause of wide QRS complex tachycardia in WPW syndrome is a reentrant tachycardia with antegrade

conduction over a nodoventricular (Mahaim) pathway and retrograde conduction over the bundle branch-His system. As in antidromic tachycardia, such a tachycardia produces maximal preexcitation and cannot be distinguished from VT from the surface ECG.

Ventricular Tachycardia

From the time that aberrant ventricular conduction was first described (Lewis, 1909), research aimed at deciphering electrocardiographic criteria to make the differential diagnosis of wide QRS complexes has been reported. The problem with the early research, however, was that there was no foolproof method of determining a ventricular versus a supraventricular origin of an arrhythmia (with the exception of those few studies which induced premature beats directly by catheters placed in the atrium or ventricle). By and large, the diagnosis was deduced from analysis of surface electrocardiographic recordings.

Introduction of a technique for recording electrical events from within the heart (Scherlag et at., 1969), revolutionized research aimed at delineating electrocardiographic criteria to differentiate wide QRS complex tachycardias because it provided a foolproof empirical method to diagnose the site of origin of the arrhythmia. Intracardiac electrograms, unlike ECGs recorded from the body surface, are capable of recording the depolarization of the bundle of His. The relationship between the His deflection (H) and ventricular depolarization (V) makes it possible to determine whether the impulse originates in the ventricle or from a supraventricular site. Differentiation between supraventricular and ventricular rhythms by intracardiac recordings is made in the following way (Figure 9): (a) a diagnosis of a supraventricular origin is made when the H-V interval is equal to or longer than the H-V interval during sinus rhythm, and, (b) a diagnosis of ventricular origin is made when the bundle of His is activated during or after the QRS complex, or in the case of His-

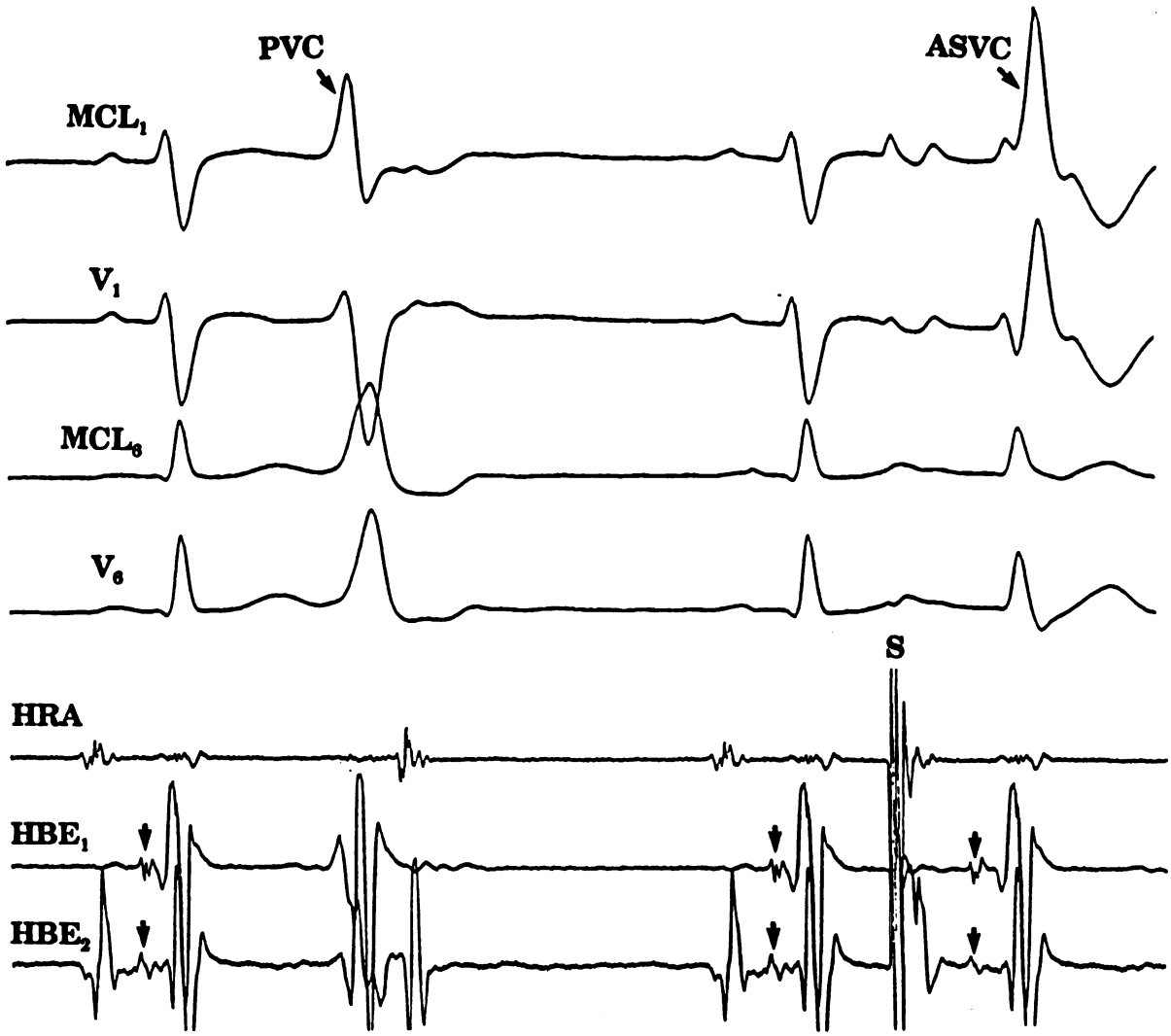


Figure 9. Definitive diagnosis of a supraventricular versus a ventricular site of origin from the His bundle electrogram (HBE). Four surface leads are shown on top followed by three intracardiac leads (high right atrial (HRA), and two HBEs). The recording is made with a paper speed of 100 millimeters per second. The first complex is a sinus beat with a His deflection visible (arrow) between the atrial and ventricular potential on the HBE recording. The subsequent wide QRS complex is ventricular in origin as evidenced by no His deflection prior to the ventricular potential. AV dissociation is present with the atrial potential following the premature ventricular complex (PVC). The last complex is an aberrant supraventricular complex (ASVC) produced by pacing the atrium as evidenced by a H-V interval longer than the H-V interval during sinus rhythm. The atrial pacing stimulus is indicated (S).

bundle activation preceding the QRS, when the H-V interval measures less than the H-V interval during sinus rhythm. A diagnosis of ventricular origin is also made when H-V dissociation is present. (Aranda, Befeler, Castellanos & El Sherif, 1976; Levy, 1984; Prystowsky & Zipes, 1984; Slama Leclercq & Laaban, 1984) Thus, research conducted during the past two decades with the His bundle electrogram used as the "gold standard" to judge a supraventricular versus a ventricular origin of wide QRS complex tachycardias is more reliable than previous research, and is the focus of the present discussion.

Diagnosis of Wide QRS Complex Tachycardias from the Surface ECG

Four characteristics of a wide QRS complex tachycardia are important in distinguishing between a aberrant SVT and VT: (a) the relationship between atrial and ventricular activity, (b) QRS width, (c) frontal plane QRS axis, and, (d) QRS morphology in leads V_1 and V_6 (Wellens et al., 1978). The latter three criteria can be determined by careful scrutiny of the QRS complex alone. There are instances, however, in which some of these criteria are invalid. Therefore, the subsequent discussion will examine the four criteria separately, including situations in which the criteria are not applicable.

Relationship Between the Atria and Ventricles During Wide QRS complex Tachycardia

Dissociation between atrial and ventricular activity during a wide QRS complex tachycardia rules out SVT with one rare exception (Wellens et al., 1978). The exception is AV nodal tachycardias with AV dissociation and a wide QRS complex. Such AV nodal tachycardias, however, typically have retrograde ventriculo-atrial conduction rather than AV dissociation. Furthermore, AV nodal tachycardias generally have normal, narrow QRS complexes unless the patient has a preexistent bundle branch block or aberrant ventricular conduction. Therefore, it is

extremely rare for both of these uncommon conditions to coincide during AV nodal tachycardia (i.e., absence of retrograde conduction allowing for AV dissociation, plus fixed or functional bundle branch block producing a wide QRS complex).

Although the observation of AV dissociation during wide QRS complex tachycardia is virtually diagnostic of a ventricular origin, failure to observe dissociation is unhelpful for two reasons. First, some form of ventriculo-atrial conduction is present in approximately 50 percent of VTs (Wellens et al., 1978) Second, even if AV dissociation is present, P waves are often buried within the wide, distorted QRS complexes and invisible on the surface ECG. The reason that Wellens and co-workers could identify AV dissociation so readily during VT was that they had the benefit of intracardiac recordings. Such recordings are unavailable in clinical settings unless the patient has a temporary pacemaker wire in the atrium. Nonetheless, several strategies can be employed at the bedside to search for dissociated P waves during a wide QRS complex tachycardia. The first method is to look for ventricular capture beats and fusion beats (Dressler & Roesler, 1952). Ventricular capture beats with normal, narrow QRS complexes can interrupt VT, especially when the rate of the tachycardia is not extremely rapid. They occur when the dissociated sinus impulse "slips" through the AV node and captures the ventricles when they are not refractory to the descending impulse. During rapid VT, such non-refractory periods of the ventricles are brief, and capture by a descending sinus impulse is improbable.

Partial ventricular capture beats (i.e., fusion beats) occur when the ventricles are partly activated by the dissociated sinus impulse and partly by the ectopic ventricular impulse. The width and contour of the fusion QRS complex depends on the relative contributions to ventricular depolarization of the sinus and ectopic ventricular foci. The resultant complex will be more

abnormal than the pure sinus-conducted complex, and less abnormal than the pure ventricular ectopic complex. Thus, both ventricular capture and fusion beats provide evidence of independent atrial and ventricular activity during a wide QRS complex tachycardia. One must be certain, however, that fusion beats are the product of independent atrial and ventricular activity rather than fusion between two independent ventricular impulses. Situations in which fusion can occur between two ventricular foci resulting in normalization of the QRS complex include: (a) fusion between the VT impulse and a ventricular premature beat arising in the contralateral ventricle, and, (b) fusion between the VT impulse and a ventricular echo beat which occurs when a retrogradely-conducted impulse travels to the AV node and reenters the ventricle (Wellens & Brugada, 1987). Thus, it is important to identify atrial activity (i.e., a P wave) prior to the fusion complex.

A second strategy for identifying independent atrial activity is to record from a lead located within or close to the atria. Temporary atrial epicardial pacing wires placed routinely during cardiac surgery can be used to record the atrial electrogram when wide QRS complex tachycardia occurs following open-heart surgery (Finkelmeier & Salinger, 1984; Mantle, Strand & Wixson, 1978; Sulzbach, 1985; Waldo, MacLean, Cooper, Kouchoukos & Karp, 1978). Esophageal electrograms can be recorded by inserting an electrode in the same manner as a nasogastric tube, or by having the patient swallow a specially-designed pill electrode (Arzbaecher, 1978; Hammil & Pritchett, 1981).

The QRS Complex During Wide QRS Complex Tachycardia

QRS width. In a landmark study on criteria to distinguish SVT with aberration from VT, Wellens and co-workers (1978) compared the electrocardiographic features of 70 instances of VT with 70 episodes of SVT with aberrant conduction, the origin of all cases being proven by

simultaneous His bundle electrograms. Wellens and associates (1982) later expanded the study to include 100 instances of VT and 100 episodes of SVT with aberration or bundle branch block. These researchers found that all cases of SVT with aberrant conduction had a QRS width less than or equal to 140 ms, whereas 59 percent of cases with VT had a QRS width greater than 140 ms. Thus, these researchers concluded that a QRS width of more than 140 ms strongly suggested VT.

The VTs which have QRS widths of less than 140 ms tend to occur in patients without heart disease (Coumel, Leclercq, Attuel & Maisonblanche, 1984). Coumel and co-workers (1984) compared 100 episodes of VT belonging to patients with acute or previous myocardial infarction with 70 cases of VT belonging to a younger group without evidence of heart disease. The average QRS width was 171 ± 30 ms in the heart disease group, and 135 ± 10 ms in the normal heart group ($P < 0.001$). Ventricular tachycardia in young people without heart disease tends to be well-tolerated, and may be managed outside critical care units. Thus, the VTs which occur in critical care units undoubtedly have an even higher incidence of QRS widths greater than 140 ms.

Although the observation of extremely wide QRS complexes during tachycardia is a very helpful clue in diagnosing VT, there are three situations in which it is invalid: (a) tachycardias occurring in patients receiving antiarrhythmic drugs which prolong the QRS complex, (b) the presence of preexistent bundle branch block, and, (c) the presence of antegrade conduction over an accessory AV pathway. Regarding the first exception, it is important to point out that none of Wellens' SVT patients were receiving antiarrhythmic drugs at the time of study. It is a well-established fact that certain antiarrhythmic drugs (especially the Class IA, IC, and III agents), may prolong the QRS complex. Thus, the diagnostic value of observing a QRS duration greater than 140 ms in a patient receiving such drugs is unknown. Regarding the second exception, while none

of Wellens' (1981) 89 cases of SVT with aberration had QRS complexes of more than 140 ms, five of the eleven cases of SVT with preexistent bundle branch block did. These five QRS widths ranged from 160 to 180 ms and occurred in both preexistent right and left bundle branch block. Patients with permanent bundle branch block often have QRS widths of more than 140 ms during sinus rhythm. Obviously, if these patients develop SVT, their QRS widths are no narrower during the tachycardia than they are during sinus rhythm. It is unclear why Wellens and associates found fixed bundle branch block wider than functional bundle branch block (i.e., aberration).

The third situation in which a QRS duration of greater than 140 ms is unhelpful involves a SVT with antegrade conduction over an accessory AV pathway. This is not surprising because, in such tachycardias, ventricular activation starts outside the specialized ventricular conduction system, similar to during VT. These exceptions to the rule underscore the importance of careful inspection of the ECG during sinus rhythm to detect preexistent bundle branch block or preexcitation.

QRS axis. Wellens and co-workers (1978) demonstrated that the majority of patients with VT had a markedly abnormal QRS axis in the frontal plane. In fact, 91 percent of wide QRS complex tachycardias with left axis deviation (- 30 to - 150 degrees) were VT. A normal or right axis deviation was more likely to be SVT with aberration, but these findings were less helpful. For example, normal axis was SVT 80.5 percent of the time; right axis deviation was SVT 71 percent of the time. In contrast to Wellens' data, Caceres and co-workers (1987) found left axis deviation (- 30 to - 180 degrees) to be less helpful in diagnosing VT, indicating VT 84 percent of the time. A drawback of Caceres' study, however, was his small sample of SVTs with aberration (only 16 cases).

Caceres did find that an extremely abnormal axis deviation (i.e., in the northwest quadrant of - 90 to - 180 degrees) was diagnostic of VT since it did not occur in SVT with aberration. Akhtar and associates (1988) looked at axis orientation in 150 patients with wide QRS tachycardia and confirmed Caceres' observation that a bizarre frontal plane axis was diagnostic of VT. These researchers found that about 20 percent of Vts had an axis in the northwest quadrant, and that none of the aberrant SVTs exhibited an axis in this range.

Coumel and co-workers (1984) reported that left axis deviation was more indicative of VT in patients with heart disease than in patients with normal hearts (Table 3). In addition, Wellens and associates (1982) reported that left axis deviation was a more helpful clue when looking solely at wide QRS complex tachycardias with a right bundle branch block contour (Table 4). Kindwall and co-workers' study (1988) agreed with Wellens' data showing that left axis deviation in left bundle branch block-contour tachycardias is less diagnostic of VT. These researchers reported that left axis deviation in left bundle branch block-contour tachycardias had a sensitivity of 67 percent, specificity of only 41 percent, and predictive value for VT of just 79 percent.

Table 3
A Comparison of Frontal Plane QRS Axis During Ventricular Tachycardia in Patients With and Without Heart Disease

	NORMAL AXIS - 30° to +120°	RIGHT AXIS +120° to +180°	LEFT AXIS - 30° to - 180°
HEART DISEASE	16%	28%	54%
NO HEART DISEASE	75%	7%	18%

Table 4
Incidence of Left Axis Deviation in 200 Episodes of Wide QRS Complex Tachycardia

	ASVT	VT
RBBB CONTOUR	4%	74%
LBBB CONTOUR	13%	57%

ASVT = aberrant supraventricular tachycardia
VT = ventricular tachycardia
RBBB = right bundle branch block
LBBB = left bundle branch block

There are two situations in which the finding of a left axis deviation is unhelpful in the diagnosis of VT. The first is SVT with preexistent bundle branch block. In Wellens' study (1981), 82 percent of patients with SVT and preexistent bundle branch block had left axis deviation. This is not surprising since both left bundle branch block and right bundle branch block with left anterior hemiblock typically have left axis deviation. The researchers do not speculate on whether aberration with right bundle branch block and left anterior hemiblock contour would also have a similar left axis deviation. An axis in the northwest quadrant, however, has been shown to be helpful in diagnosing VT despite the presence of preexistent bundle branch block. For example, Kremers and co-workers (1988) studied the effect of preexistent bundle branch block on the electrocardiographic diagnosis of VT and concluded that an axis in the northwest quadrant strongly favored VT. They observed no sinus ECG with preexistent bundle branch block that had this bizarre axis.

The second situation in which aberrant SVTs can exhibit a markedly abnormal axis are tachycardias with antegrade conduction over an accessory AV pathway. Marked left axis deviation can be found during antegrade conduction over a right-sided or posteroseptal accessory pathway, and marked

right axis deviation can be found during antegrade conduction over a left lateral pathway (Wellens & Brugada, 1987). The latter situation may explain the discrepancy between Wellens' and Caceres' data of the importance of left axis deviation in diagnosing VT. Wellens' 100 episodes of wide QRS complex SVT excluded patients with accessory pathways, whereas 41 percent of Caceres' episodes of SVT represented antegrade conduction over an accessory AV pathway.

In summary, the observation of left axis deviation during right bundle branch block-contour wide QRS complex tachycardia provides strong evidence for VT in cardiac patients without accessory pathways or prior bundle branch block. Moreover, the observation of a bizarre axis in the - 90 to - 180 degree range (irrespective of bundle branch block contour or presence of preexistent bundle branch block) in such patients is essentially diagnostic of VT.

QRS morphology in leads V_1 and V_6. Because right and left bundle branch block can be readily identified in V_1 or V_6, aberrant conduction can also be readily identified in these leads. Moreover, ventricular rhythms have been shown to exhibit characteristic QRS patterns different from the classic aberrant supraventricular patterns. Thus, V_1 and V_6 have proven valuable in distinguishing aberrant SVT from VT (Figure 10) (Kindwall et al., 1988; Sandler & Marriott, 1965; Swanick, LaCamera & Marriott, 1972; Wellens et al., 1978).

QRS morphology in right bundle branch block contour tachycardias. Wellens and co-workers (1978) found that a monophasic or biphasic pattern in V_1 strongly favored a ventricular origin, whereas a triphasic pattern favored a supraventricular origin. A third pattern Wellens found to be diagnostic of VT was the taller left peak pattern originally reported by two coronary care unit nurses (Gozensky & Thorne, 1974). These nurses analyzed ventricular premature beats in 500 consecutive acute myocardial infarction patients noting that many of the right bundle branch block

LEAD V_1	LEAD V_6
VENTRICULAR QRS MORPHOLOGIES	
Monophasic R	Biphasic Rs with R:S ratio < 1.0
Taller left peak	Monophasic Q
Biphasic Rs	Notched QS
Biphasic Qr	Biphasic Qr
One or more of the following: (a) R > 30 ms (b) Slurred or notched S descent (c) QRS onset to S nadir > 60 ms	
ABERRANT SUPRAVENTRICULAR QRS MORPHOLOGIES	
Bimodal rR′ or Triphasic rsR′	Triphasic qRs with R:S ratio > 1.0
All of the following: (a) R ≤ 30 ms or no R (b) Straight S descent (c) QRS onset to S nadir ≤ 60 ms	
UNHELPFUL QRS MORPHOLOGIES	
Slurred or notched taller right peak	Monophasic R
	Taller left or right peak
	Biphasic RS with R:S ratio > 1.0

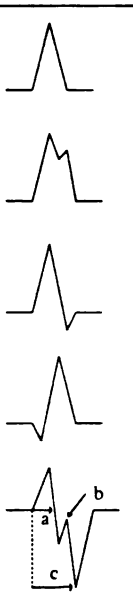


Figure 10. QRS patterns reported to be of value in distinguishing aberrant SVT from VT.

contour premature beats had two distinct peaks in lead V_1, which they called "rabbit ears." They observed that the taller left peak pattern, which was commonly seen in ventricular premature beats, was a valuable clue in distinguishing ventricular ectopy from right bundle branch block aberration because the latter typically had a taller right peak pattern.

Wellens also found lead V_6 to be useful in distinguishing right bundle branch block contour beats. Typically, in the supraventricular complex, the QRS in lead V_6 started with a Q wave; the Q wave was followed by an R wave which was greater in amplitude than the subsequent broad S wave (R:S ratio greater than 1.0). In contrast, the ectopic ventricular complex showed an R:S ratio of less than 1.0 , or a QS complex in lead V_6.

QRS morphology in left bundle branch block contour tachycardias. Kindwall and co-workers (1988) identified four characteristics of left bundle branch block contour beats which were helpful in distinguishing ventricular tachycardia. These criteria included: (a) an R wave in V_1 or V_2 of more than 30 ms; (b) a slurred or notched S descent in V_1 or V_2; (c) greater than 60 ms from QRS onset to S nadir in V_1 or V_2; and, (d) any Q in V_6.

Kindwall and associates (1988) reported that the presence of all four of these criteria during a tachycardia had a high predictive value for diagnosing VT (96 to 100 percent) and specificity (94 to 100 percent) but a rather low sensitivity (36 to 63 percent). All 91 VT tracings had at least one of the four criteria; therefore, the sensitivity of observing one or more of the four criteria was 100 percent. Only three of the 27 SVTs with aberration had any one of the four criteria, and most had none. Thus, the specificity for VT when any one of the criteria was present was 89 percent.

QRS concordance in the precordial leads. Vera and co-workers (1972) were the first to describe concordancy of the precordial QRS complexes (i.e., when the complexes from V_1 to V_6

were either all positive or all negative). These authors found concordancy to be nearly diagnostic of ventricular ectopy; however, it occurred so seldom during VT that it was of little clinical value.

Value of the 12-Lead ECG in Diagnosing Tachycardias with a Wide QRS Complex

Wellens and associates (1981) conducted a prospective analysis of 62 episodes of regular wide QRS complex tachycardia to determine the accuracy of the 12-lead ECG in making the correct diagnosis. In no patient was information on the ECG during sinus rhythm or findings from the physical examination available. The site of origin of tachycardia was verified by intracardiac recordings. The site of origin was correctly predicted in 57 patients (92%). Of the five tachycardias which were misdiagnosed, four had SVT with preexistent bundle branch block exhibiting left axis deviation and a QRS width of more than 140 ms. These four tachycardias no doubt would have been correctly diagnosed had the researchers had the benefit of previous ECGs taken during sinus rhythm, which would have raised the accuracy to over 98 percent. The one remaining SVT which was indistinguishable from VT occurred in a patient who had an atrial tachycardia with antegrade conduction over an accessory pathway. This patient's tachycardia exhibited a QRS width of 160 ms, a positive concordant pattern, and left axis deviation. These data suggest that if a wide QRS complex tachycardia is captured in all 12 leads of a conventional ECG, and if previous tracings are available for comparison, the arrhythmia can be correctly predicted nearly 100 percent of the time.

Operational Definitions

Selection of Leads for Analysis

Because a limited number of leads are available for continuous bedside monitoring, the question arises as to which single lead or two, three, or four-lead set should be selected. The leads evaluated in the present study were those routinely used in clinical practice and those hypothesized to be of value in diagnosing wide QRS complex tachycardia based on the following assumptions:

1. The ability to diagnose bundle branch block is desirable. Thus, a right or left precordial lead such as V_1, MCL_1, V_6, MCL_6, or lead I are advantageous. Leads II, III, aVR, aVL, aVF, and V_2 to V_5 are less desirable.
2. The observation of QRS morphology in V_1 or MCL_1 and V_6 or MCL_6 is desirable.
3. The ability to observe precordial QRS concordancy from simultaneous V_1 and V_6 or MCL_1 and MCL_6 is slightly advantageous (i.e., the finding is specific for VT, but seldom occurs).
4. The ability to detect abnormal frontal plane QRS axis is desirable. Since monitoring leads are not standardized, precise measurement of QRS axis in degrees is not possible from the bedside. The observation of QRS polarity in certain limb leads, however, can elucidate an axis *range* the tachycardia complex falls within. Leads helpful in this regard include lead II, aVR, combined II and aVR, and, combined I and aVF (Figure 11). Leads unhelpful in this regard include the precordial leads, III, and, aVL.
5. All other criteria such as QRS width, AV dissociation, fusion beats, ventricular capture beats, and, a change in QRS contour from baseline with preexisting bundle branch block, can be observed from any lead.

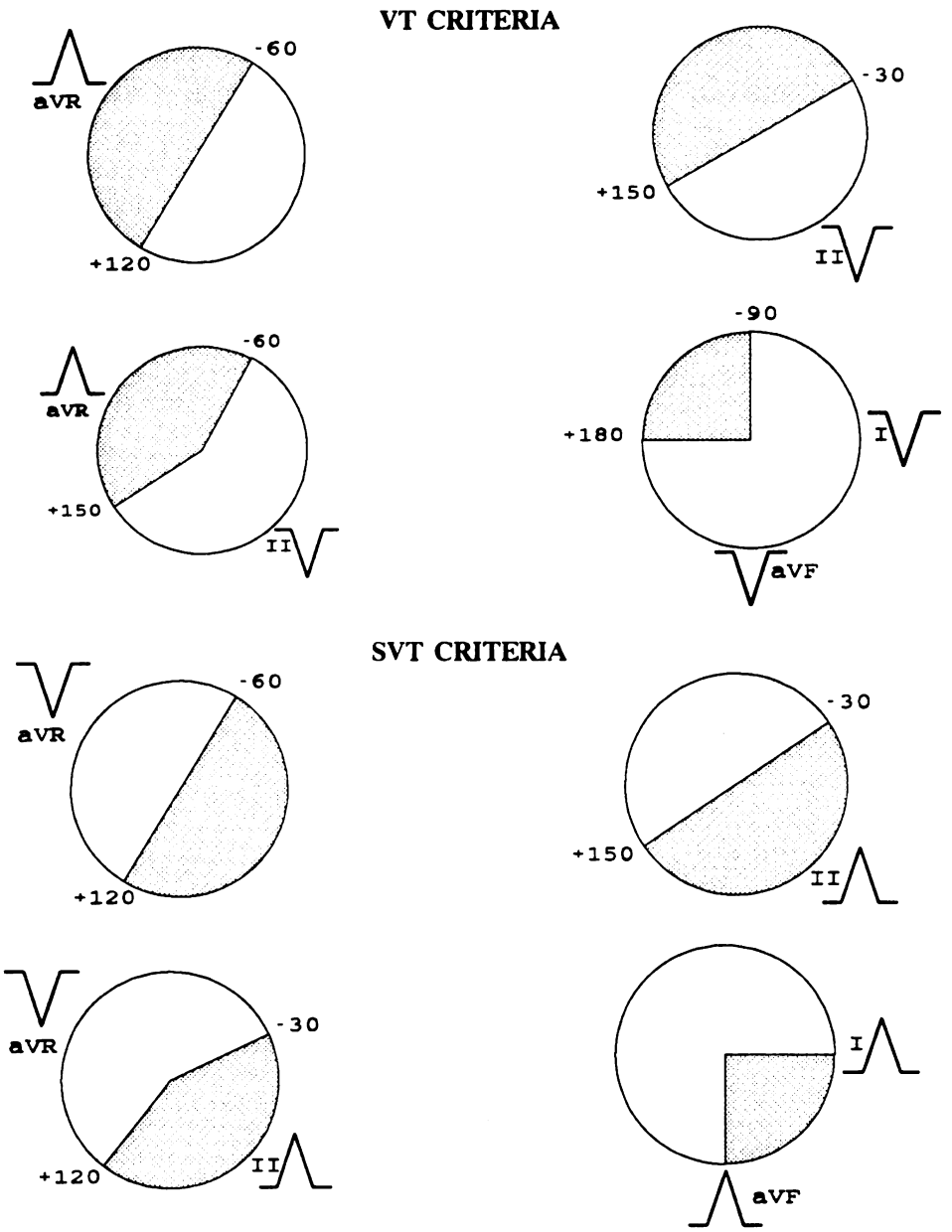


Figure 11. Determination of axis range suggestive of aberrant SVT or VT by noting QRS polarity from single or dual limb leads. QRS polarities suggestive of VT include: (a) a positive QRS in aVR, (b) a negative QRS in lead II, (c) a negative QRS in lead II plus a positive QRS in aVR, and, (d) a negative QRS in lead I and aVF. QRS polarities suggestive of aberrant SVT include: (a) a negative QRS in aVR, (b) a positive QRS in lead II, (c) a positive QRS in lead II plus a negative QRS in aVR, and, (d) a positive QRS in lead I and aVF. The actual degree ranges such polarities represent are designated by the stipled areas. VT = ventricular tachycardia; SVT = supraventricular tachycardia.

Thus, the following leads were selected for analysis:

Single lead monitoring:

1. MCL_1
2. MCL_6
3. V_1
4. V_6
5. II

Dual lead monitoring:

1. MCL_1 and MCL_6; V_1 and V_6
2. MCL_1 and II; V_1 and II
3. MCL_6 and II; V_6 and II

Triple lead monitoring:

1. MCL_1, II, and aVR; V_1, II, and aVR
2. MCL_6, II, and aVR; V_6, II, and aVR
3. MCL_1, I and aVF; V_1, I and aVF
4. MCL_6, I and aVF; V_6, I and aVF
5. MCL_1, MCL_6 and II; V_1, V_6, and II
6. MCL_1, MCL_6, and aVR; V_1, V_6, and aVR

Quadruple lead monitoring:

1. MCL_1, MCL_6, I and aVF; V_1, V_6, I and aVF

Sensitivity, Specificity, Predictive Value, and Predictive Accuracy

The sensitivity of an ECG criterion for diagnosing VT measures the proportion of those with VT (as measured by the His bundle electrogram) correctly identified by the criterion. In other words, it measures how often the criterion is observed from the surface ECG during VT. Likewise, the sensitivity of a particular lead or lead set for diagnosing VT measures the proportion of those with VT correctly identified by observing criteria from the lead(s) in question. The value is given as a percentage and the formula is as follows:

$$\text{Sensitivity} = \frac{\text{the number of true detections of VT}}{\text{the total number of VTs}}$$

The specificity of a criterion for diagnosing VT measures the proportion of those who do not have VT (i.e., those who have SVT as measured by the His bundle electrogram) who are correctly eliminated by the criterion.

$$\text{Specificity} = \frac{\text{the number of SVTs without the VT criteria}}{\text{the total number of SVTs}}$$

The predictive value of a criterion's positive finding measures the proportion of those exhibiting the VT criterion who truly have VT (as measured by the His bundle electrogram). It answers the clinically relevant question: When I observe this VT criterion in the clinical setting, how likely is it that the individual really has VT?

$$\text{Predictive value} = \frac{\text{the number of true detections of VT}}{\text{the number rated VT by the criterion}}$$

Predictive accuracy is the measure of the capability of a criterion (or particular lead) to yield correct results. It gives a general or overall figure-of-merit for the criterion (or lead), since it includes the ability of the criterion (or lead) to recognize VT as well as SVT.

$$\text{Predictive accuracy} = \frac{\text{the number of true detections of VT } \textit{and} \text{ SVT}}{\text{the total number of wide QRS tachycardias}}$$

Research Questions

1. Are the modified precordial leads MCL_1 and MCL_6 as valuable as the conventional precordial leads V_1 and V_6 in distinguishing aberrant SVT from VT? Specifically, are the characteristic QRS patterns suggestive of aberrant SVT or VT in V_1 and V_6 also present in MCL_1 and MCL_6 during wide QRS complex tachycardia?
2. Which ECG criteria are most useful in distinguishing aberrant SVT from VT?
3. Which single lead is most valuable for diagnosing wide QRS complex tachycardia? Which lead set is most valuable for making the diagnosis? How many leads are necessary to approximate the diagnostic accuracy of a full 12-lead ECG?

CHAPTER THREE: METHODS

Sample and Setting

One hundred and twenty one morphologically distinct wide (greater than 120 ms) QRS tachycardias were recorded from 92 adults undergoing cardiac electrophysiology study at the University of California, San Francisco during a one year period. Informed verbal consent was obtained as approved by the Committee on Human Research (Appendices A & B). Only monomorphic tachycardias lasting at least six beats at a rate greater than 100 beats per minute were selected for analysis. Ventricular tachycardias in the flutter range of 300 beats per minute or greater were excluded from analysis. Supraventricular tachycardias induced in patients who had a bundle branch block at baseline were excluded because the QRS complex during tachycardia exhibited the same bundle branch block configuration. Because it is theoretically impossible to distinguish SVT with antegrade conduction over an accessory pathway from VT, antidromic tachycardia was also excluded from the analysis. More than one tachycardia from the same patient was used in the analysis if the patient developed: (a) both SVT and VT, (b) SVT with both right and left bundle branch block-type aberration, or, (c) VT with clearly different morphology. Tachycardias were defined as morphologically distinct if they exhibited different bundle branch block contours or if they had a markedly different frontal plane QRS axis.

Instruments, Procedure, and Analysis

A conventional 12-lead ECG was recorded with a Marquette (MAC 12) instrument at a paper speed of 25 millimeters per second (mm/sec) (the paper speed routinely used in clinical practice). A baseline ECG was recorded and subsequent ECGs were recorded during all episodes of wide QRS complex tachycardia. The Marquette ECG recorder allowed for a continuous rhythm strip

of all leads so that non-sustained tachycardias were documented in all twelve leads. The bedside leads MCL_1 and MCL_6, the conventional leads V_1 and V_6, a high right atrial electrogram, and three low right atrial septal electrograms including the His bundle electrogram were recorded continuously throughout the entire study with an Electronics for Medicine multichannel monitor and strip recorder, and stored on a Hewlett-Packard eight-channel reel-to-reel tape recorder.

When an episode of tachycardia was induced, the tape recorder counter number was noted on the conventional 12-lead ECG recording so that the same tachycardia episode could later be examined from the bedside leads, 12-lead ECG, and His bundle electrogram recordings. Because the same electrode position was required for more than one lead (e.g., both MCL_1 and V_1 require an electrode in the fourth intercostal space to the right of the sternum), electrode wires were soldered together so that the same skin electrode could be connected to more than one lead cable.

Two independent observers rated the QRS complexes as being identical, similar, or clearly different between simultaneous recordings of MCL_1 and V_1 and between MCL_6 and V_6 during baseline sinus rhythm and during wide QRS complex tachycardia. An identical rating applied when the complex from the two leads being compared contained identical component waves of equal or nearly equal width and height. A clearly different rating applied when the QRS complexes were obviously dissimilar (e.g., the complex was primarily positive in one lead while primarily negative in the comparable lead). A similar rating applied when the QRS complex in the two leads exhibited the same predominant polarity but contained minor variations in the component waves. For example, if the complex was a rsR′ pattern in MCL_1 and a rR′ pattern in V_1, the complexes were rated as similar. Inter-rater reliability was assessed by Cohen's Kappa to determine the

statistical significance of the agreements (i.e., to confirm that the proportion of agreement was greater than chance).

Each of the 121 wide QRS complex tachycardias were analyzed blinded from the His bundle electrogram diagnosis in the two modified and twelve conventional ECG leads for evidence of AV dissociation, (i.e., presence of dissociated P waves, fusion complexes, or ventricular capture complexes). QRS width was measured in all leads; the greatest width being used in the analysis. Frontal plane QRS axis was determined and the change in axis from baseline was also noted. Because a limited number of leads are available for continuous bedside monitoring, it was important to determine whether axis information gleaned from a single limb lead might be helpful in distinguishing aberrant SVT from VT. Thus, the predominant QRS polarity during tachycardia in leads II and aVR was noted. Likewise, to determine whether axis information gleaned from dual limb leads would be useful in the differential diagnosis, the predominant QRS polarity in combined II and AVR and combined I and AVF was assessed.

QRS morphology during tachycardia was analyzed in several ways. First, the modified precordial leads MCL_1 and MCL_6 and the conventional precordial leads V_1 and V_6 were examined for presence of the previously-proposed morphologic criteria suggestive of aberrant supraventricular or ventricular origin. The configuration of QRS complexes which did not fall into the classic patterns were also examined for new morphologic criteria which might be useful in making the diagnosis. When QRS morphology was more clearly evident in the first complex of a tachycardia, the morphology chosen for analysis was that of subsequent complexes because the onset of tachycardia is rarely captured in the clinical setting, and a diagnosis must be made from such subsequent complexes. Second, presence of positive or negative QRS concordance was noted in

the six precordial leads. Moreover, to determine whether a concordant pattern observed in just two precordial leads (i.e., without information from the remaining four mid-precordial leads) would be helpful in making the diagnosis, concordance in MCL_1 and MCL_6 and V_1 and V_6 was noted. Third, to test whether the initial deflection of the QRS complex would discriminate between aberrant SVT and VT, a measurement of QRS onset to tallest peak (positive complexes) or nadir (negative complexes) was measured in MCL_1, MCL_6, V_1, and V_6. Since these leads were recorded on tape, they could be played back at fast paper speeds (100 mm/sec) for greater measurement precision. Fourth, in patients with a preexisting bundle branch block, a change in QRS morphology from baseline rhythm was noted during VT.

Each of the ECG criteria was compared to the His bundle electrogram diagnosis using the Fisher's Exact test to determine statistical significance. A diagnosis of ventricular site of origin was made from the His bundle electrogram if: (a) the His deflection fell during or after the QRS complex, (b) the H-V interval measured less than the H-V interval during sinus rhythm, or, (c) H-V dissociation was present. A diagnosis of supraventricular site of origin was made if the H-V interval was equal to or longer than the H-V interval during sinus rhythm. In some instances, the His bundle electrogram was not recorded during restudy of patients with known VT. The diagnosis of VT in these cases was made by observing sustained VT resulting in hemodynamic instability requiring termination by DC shock or ventricular overdrive pacing. The sensitivity, specificity, predictive value of a positive finding, and predictive accuracy were used to determine the value of the criteria for making the differential diagnosis.

Using the well-established ECG criteria as well as new criteria observed in the present study, a diagnosis of supraventricular, ventricular, or indeterminate was made from the conventional 12-

lead ECG while blinded from the His bundle electrogram diagnosis. In addition, a diagnosis was made from single leads routinely used in current practice for continuous bedside monitoring including MCL_1, V_1, MCL_6, V_6, and lead II. Furthermore, a diagnosis was made from dual, triple, and quadruple lead sets hypothesized to be valuable in making the differential diagnosis. The diagnosis of indeterminate was made when there were no criteria observed in the particular lead or lead set with which to make the diagnosis or when the criteria from a particular lead or lead set was contradictory. For example, if a wide complex exhibited a triphasic rsR′ contour in V_1 suggestive of aberrant SVT, but also had a width of 180 ms suggestive of VT, the diagnosis was labelled indeterminate. The diagnosis of SVT, VT, or indeterminate from the conventional 12-lead ECG and selected monitoring leads and lead sets was compared to the His bundle diagnosis of SVT or VT in 2 X 3 crosstabs tables using the chi square statistic to determine statistical significance. The value of various leads was determined by which had the highest predictive accuracy in diagnosing wide QRS complex tachycardia when criteria were observed from the lead in question. A test for the difference between correlated proportions (McNemar test) was used to determine whether the differences in predictive accuracies between various leads and lead sets were statistically significant.

CHAPTER FOUR: RESULTS

Subject and Sample Characteristics

Sixty seven percent of the 92 patients were males with a mean age of 57 years (range = 17-90 years). A majority (55%) of the patients had a history of ischemic heart disease, and an additional twelve percent had a diagnosis of dilated cardiomyopathy, valvular or congenital heart disease. The remainder had a history of paroxysmal tachycardia without evidence of heart disease, including thirteen percent with accessory AV pathways. Forty percent had a history of aborted sudden death.

Of the 121 wide QRS complex tachycardias, 35 were aberrant supraventricular and 86 were ventricular. Three quarters of the aberrant SVTs were sinus, atrial, AV nodal, or orthodromic tachycardia; the remaining were aberrant conduction produced by atrial overdrive pacing (Table 5). Three VT mechanisms involved the His-Purkinje conduction system. Of these, two were bundle branch reentry with a left bundle branch block pattern; the third originated on or near the left bundle branch resulting in a right bundle branch block pattern. Ninety-five of the spontaneous tachycardias were sustained lasting 30 seconds or more (Waldo, et al., 1986). Of the sustained

Table 5
Mechanisms of 35 Supraventricular Tachycardias with Aberrant Conduction

Tachycardia	Frequency	Percent
Orthodromic	15	43
Atrial overdrive pacing	9	26
AV nodal reentrant	5	14
Atrial	5	14
Sinus	1	3

AV = atrioventricular

tachycardias, 83 percent required an intervention to terminate the tachycardia; the remainder terminated spontaneously. The method of termination included ventricular overdrive pacing in 60 percent, DC shock cardioversion in 21 percent, and administration of a drug in 2 percent. Sixteen of the tachycardias were non-sustained, lasting an average of 19 beats (range = 7-42). Heart rate during tachycardia was similar in SVT and VT; i.e., mean heart rate of spontaneous SVT was 192 beats per minute (bpm) (range = 110-260); mean heart rate of VT was 199 bpm (range = 105-290).

Tachycardias exhibited a right bundle branch block pattern more often than a left bundle branch block pattern, especially those that were aberrant SVTs; i.e., 55 percent of VTs and 63 percent of SVTs had a right bundle branch block contour. The VT patients were more likely to be treated with antiarrhythmic agents at the time of electrophysiology study than the SVT group. In fact, 59 percent of the VTs compared to only 17 percent of the SVTs were recorded from patients treated with one or more antiarrhythmic agents. As stated previously, SVTs from patients with preexisting bundle branch block were excluded from analysis, however, ten of the VTs were recorded from patients with a preexisting right (n=7) or left (n=3) bundle branch block. Moreover, 27 of the 121 tachycardias (22%) were recorded from patients with a left intraventricular conduction delay.

Comparison of Modified and Conventional Precordial Leads

A total of 424 comparisons were made by the two observers including: (a) 92 comparisons between MCL_1 and V_1, and 92 between MCL_6 and V_6 during baseline sinus rhythm, and, (b) 120 comparisons between MCL_1 and V_1, and 120 between MCL_6 and V_6 during tachycardia (one of the 121 tachycardias was not recorded with MCL_1 and MCL_6). There was consensus between the two observers in rating the similarity between the modified and conventional leads in more than 80

percent ($P < .000$). Most disagreements occurred because one observer rated an episode as identical while the other rated the episode as similar. In these cases, both QRS complexes being compared had the same number, width, and order of Q, R, or S waves, but the height of the waves varied slightly with respect to other waves in the same complex compared to the comparable lead. There were no instances in which one observer rated an episode between the two leads as identical while the other rated the episode as clearly different. All differences were readily resolved by consensus.

Table 6
Comparison of QRS Morphology between Bedside and Conventional Leads

Rhythm	N	Leads	Identical (%)	Similar (%)	Different (%)
Baseline	92	MCL_1 vs. V_1	67	23	10
		MCL_6 vs. V_6	66	29.5	4.5
Aberrant SVT	35	MCL_1 vs.V_1	63	26	11
		MCL_6 vs. V_6	68	26	6
Ventricular tachycardia	85	MCL_1 vs. V_1	26	36	38
		MCL_6 vs. V_6	63	19	18
All wide complex tachycardias	120	MCL_1 vs. V_1	37	33	30
		MCL_6 vs. V_6	65	21	14

SVT = supraventricular tachycardia

During baseline rhythm, QRS morphology was identical or similar 90 percent of the time between MCL_1 and V_1, and 95.5 percent of the time between MCL_6 and V_6 (Table 6). Likewise, during SVT with aberrant conduction, QRS morphology was identical or similar 89 percent of the time between MCL_1 and V_1, and 94 percent of the time between MCL_6 and V_6. During VT,

however, QRS morphology was often clearly different between the modified and conventional leads, especially between MCL_1 and V_1. In fact, only 22 of the 85 VTs (26%) had identical QRS morphology in MCL_1 and V_1. It was not uncommon for QRS morphology to be identical in MCL_1 and V_1 during baseline sinus rhythm, while clearly different during VT (Figure 12).

One dramatic difference between QRS morphology in MCL_1 and V_1 was discovered serendipitously during ventricular overdrive pacing from the right ventricular outflow tract area. While QRS morphology in V_1 always exhibited a primarily negative, left bundle branch block contour characteristic of right ventricular rhythms, MCL_1 frequently exhibited a primarily positive right bundle branch block contour which typically had a monophasic R or taller left peak pattern (Figure 13). This dramatic difference was reproduced in numerous patients, and was also observed during spontaneous VT originating from the right ventricular outflow tract area (Figure 14).

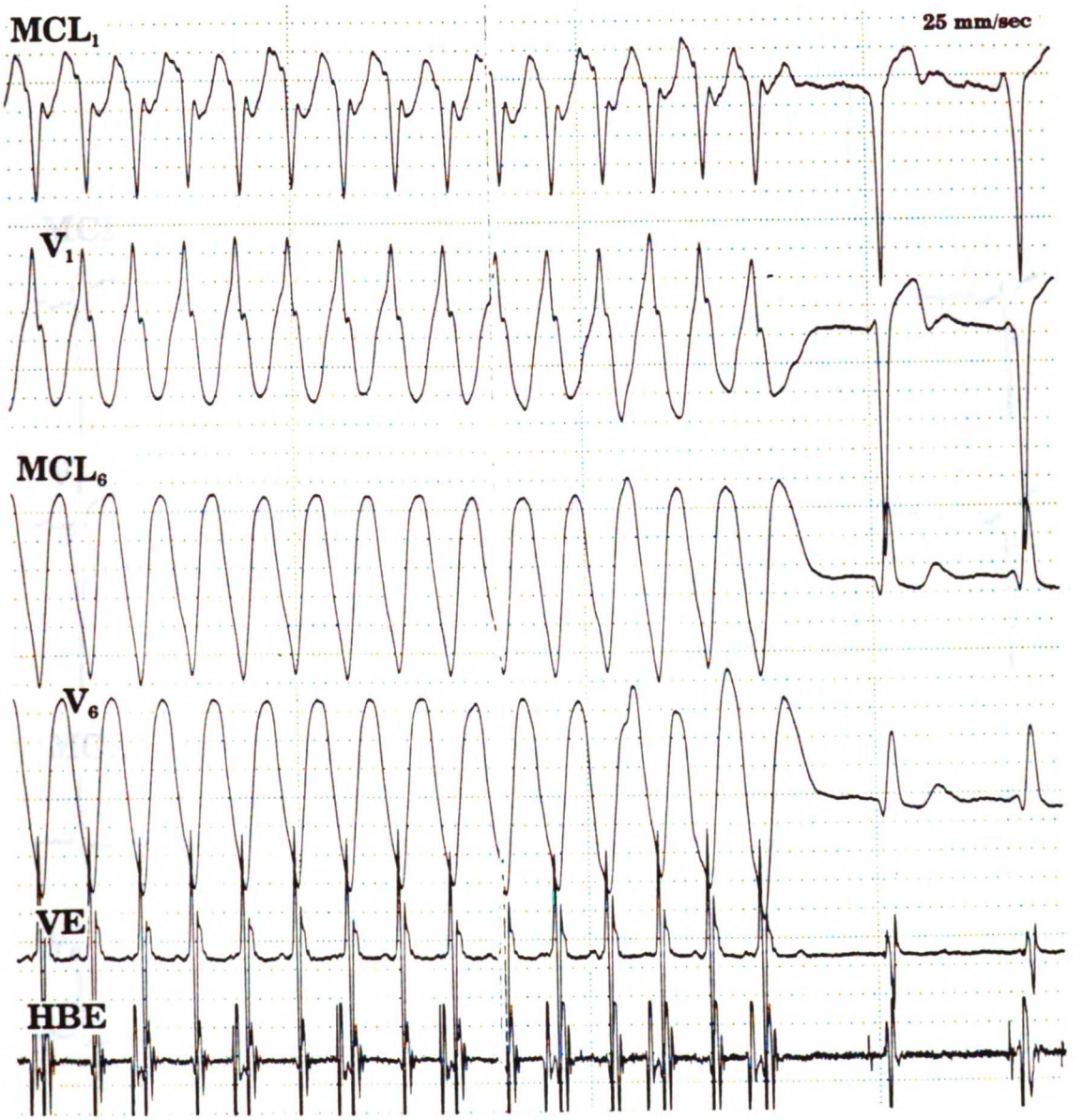


Figure 12. Nonsustained VT with clearly different QRS morphology in MCL_1 and V_1. QRS morphology in MCL_6 and V_6 is identical during both sinus rhythm and VT. Although MCL_1 and V_1 are identical during sinus rhythm, they exhibit different bundle branch block contours during VT (i.e., MCL_1 has a right bundle branch block contour while V_1 has a left bundle branch block contour).

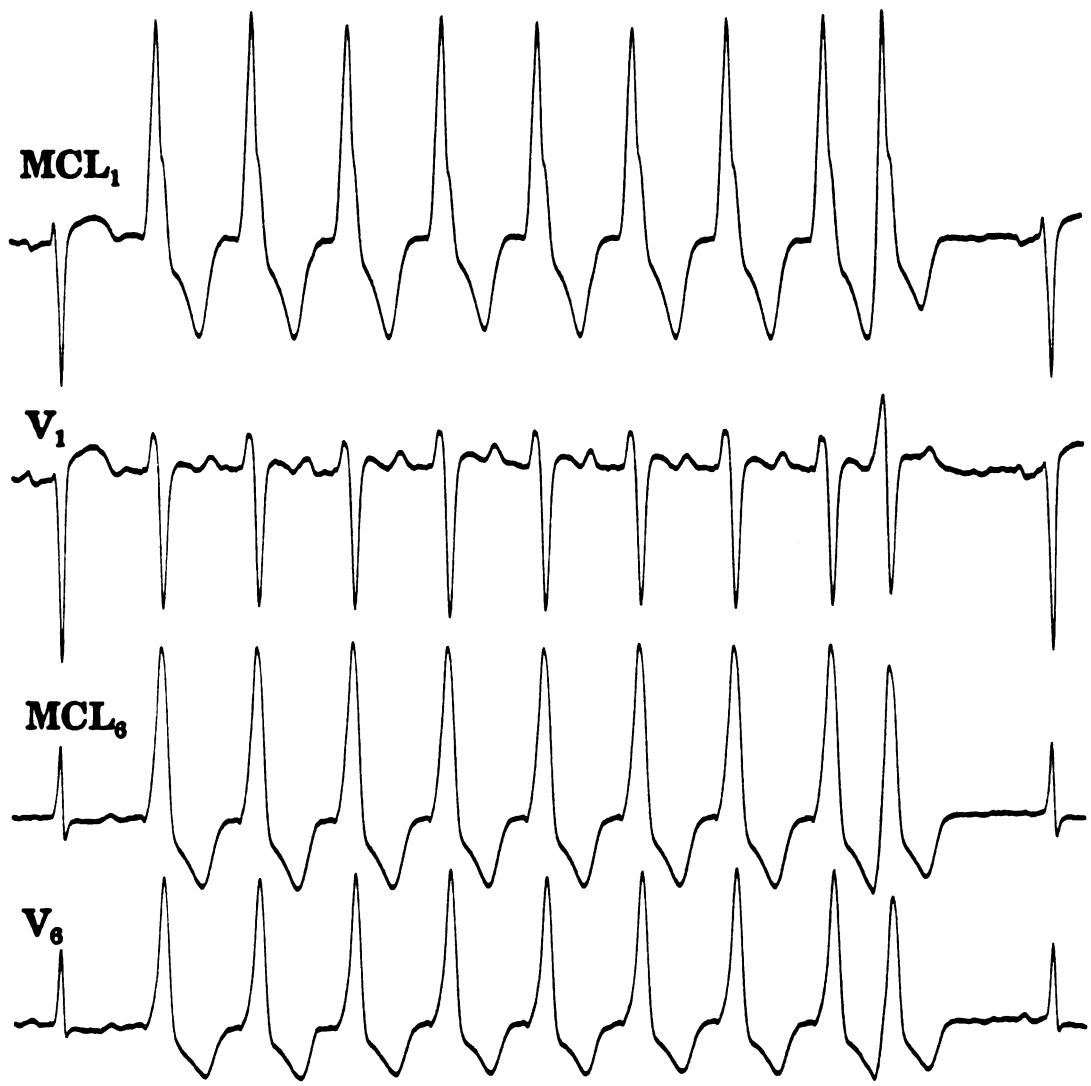


Figure 13. Pacing from the right ventricular outflow tract area showing dramatically different QRS morphology in MCL_1 and V_1 despite identical morphology during sinus rhythm. While V_1 has a left bundle branch block contour characteristic of rhythms originating from the right ventricle, MCL_1 has a right bundle branch block contour characteristic of rhythms originating from the left ventricle.

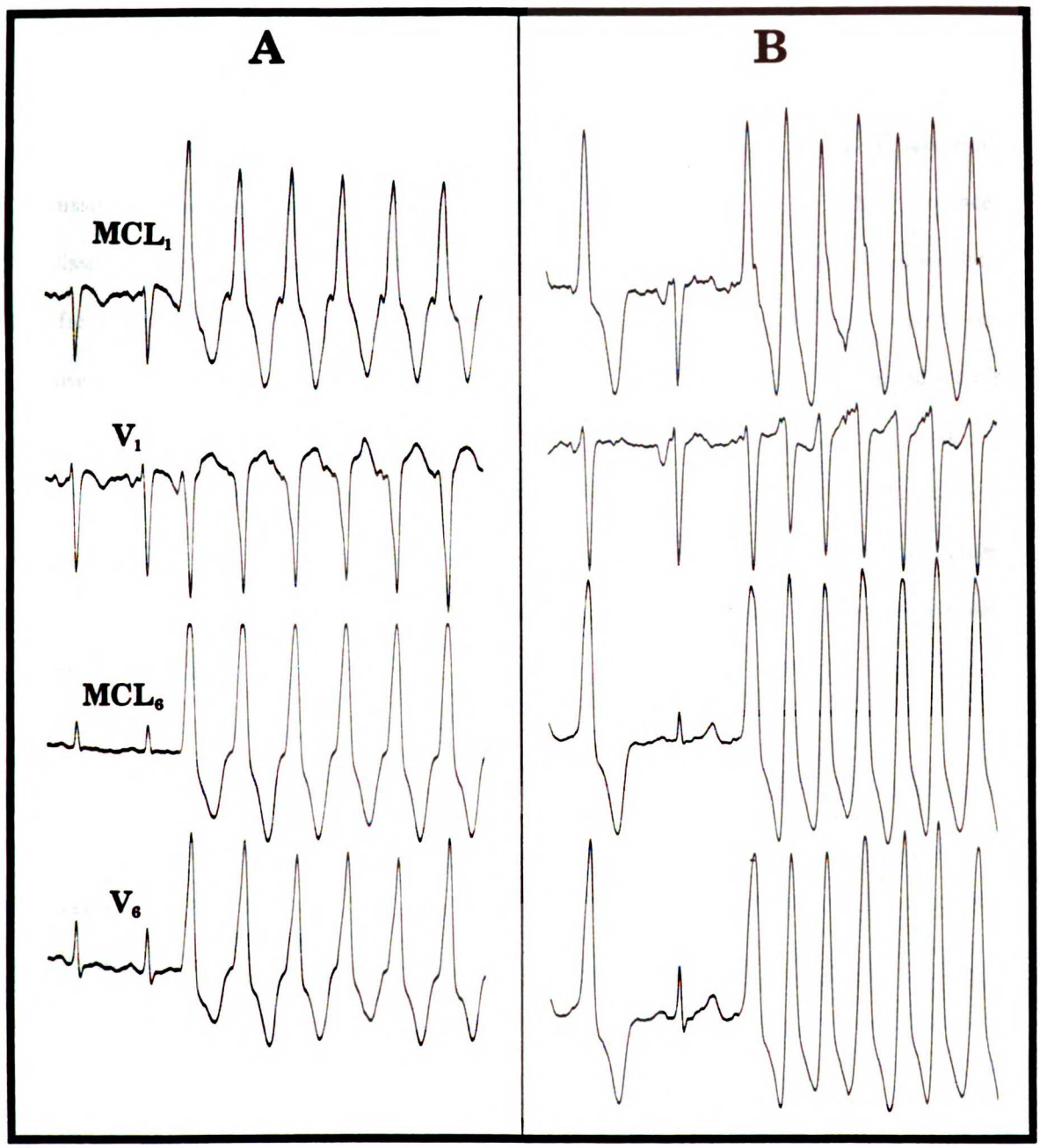


Figure 14. Pacing from the right ventricular outflow tract (A) and spontaneous non-sustained VT (B) in a 26 year old woman with right ventricular dysplasia. Pace mapping of the right ventricle during electrophysiology study revealed that the VT originated from the right ventricular outflow tract. Notice that while QRS morphology is identical in MCL_1 and V_1 during sinus rhythm, it is clearly different during the ventricular ectopic rhythms. In contrast to the right precordial leads, QRS morphology is identical in MCL_6 and V_6 during both sinus and ectopic rhythms.

Value of the ECG Criteria

Relationship Between the Atria and Ventricles

Results from the present study concur with previous studies showing that the observation of AV dissociation is diagnostic for VT (Wellens et al., 1978). None of the SVTs had evidence of AV dissociation on the intracardiac tracings, whereas 30 of the 86 VTs (35%) had AV dissociation. The frequency of AV dissociation during VT was probably underestimated in the present series because during restudies of patients with known VT, an atrial and His bundle electrogram were not always recorded. Thus, the atrial potential was not clearly visible from intracardiac leads. AV dissociation was visible in one or more leads of the 12-lead ECG in 19 percent of VTs. Only one VT exhibited ventricular capture beats, and only three had evidence of fusion. The low incidence of ventricular capture and fusion beats may have been due to the limited number of complexes recorded during VT in any one lead with the conventional 12-lead ECG format. Thus, evidence of AV dissociation was typically the observation of dissociated P waves.

Two findings of interest regarding AV dissociation were as follows. First, dissociated P waves were more likely to be visible in V_1 or MCL_1. In fact, of the 16 VTs in which AV dissociation was observed in one or more leads of the 12-lead ECG, 11 (69%) were observed in V_1 and MCL_1. Thus, AV dissociation was a more powerful criterion for diagnosing VT in V_1 and MCL_1. Second, the lead in which dissociated P waves were observed was very often a lead which had a low amplitude or nearly isoelectric QRS complex (Figure 15).

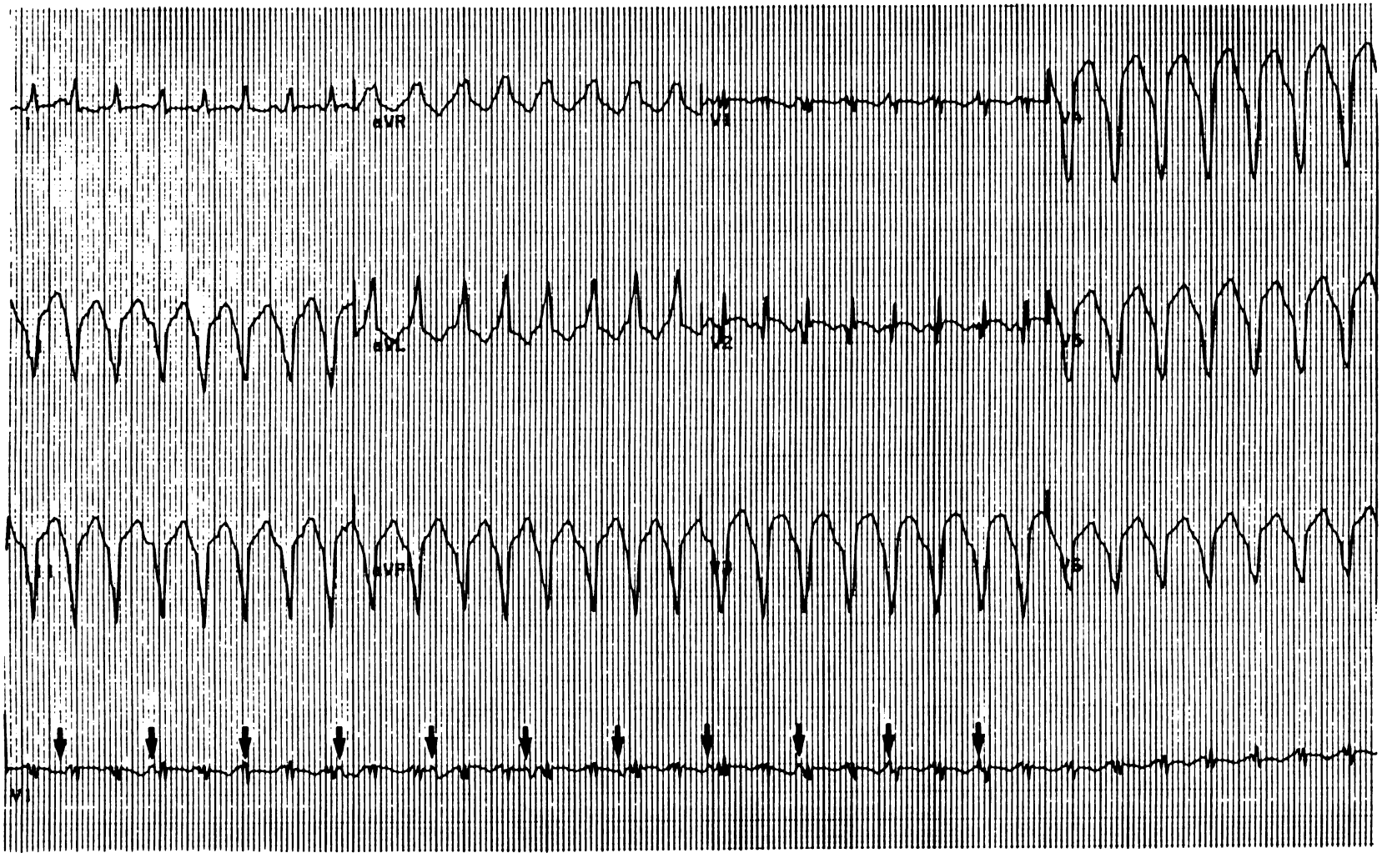


Figure 15. Wide QRS complex tachycardia with AV dissociation, which is diagnostic of VT. In the present study, dissociated P waves (arrows) were more often observed in V_1 or MCL_1 or from a lead in which the QRS complexes were nearly isoelectric (e.g., as in V_1 shown here).

Table 7
Value of QRS Width in Diagnosing Ventricular Tachycardia

QRS Width (ms)	HBE Diagnosis	
All Tachycardias	**Aberrant SVT** n = 35	**Ventricular Tachycardia** n = 86
> 140	14	76
> 160	1	59
Without drug therapy, BBB or LIVCD	**Aberrant SVT** n = 28	**Ventricular Tachycardia** n = 21
> 140	11	16
> 160	1	6
With drug therapy and/or BBB or LIVCD	**Aberrant SVT** n = 7	**Ventricular Tachycardia** n = 65
> 140	3	60
> 160	0	53

HBE = His bundle electrogram
drug therapy = class IA, IB, IC, II, III agents or digitalis
BBB = bundle branch block
LIVCD = left intraventricular conduction delay
SVT = supraventricular tachycardia

QRS Width

The previously suggested width criterion of greater than 140 ms for diagnosing VT in patients without preexisting bundle branch block or drug therapy proved to be falsely positive in numerous aberrant SVTs (Table 7). In fact, 14 of the 35 SVTs (40%) had QRS widths measuring more than 140 ms in one or more leads. Moreover, the 14 SVTs which exceeded 140 ms were recorded from patients who would not be expected to have depressed conduction. For example, none of the patients in this group had a previous myocardial infarction, or other types of heart disease such as a cardiomyopathy. The average age of patients comprising this group was 46, and

Table 8
Effect of Antiarrhythmic Drugs and Intraventricular Conduction Abnormalities on Mean QRS Width during Wide QRS Complex Tachycardia

Drugs	LIVCD	BBB	ASVT		VT	
			N	Mean QRS Width (ms)	N	Mean QRS Width (ms)
-	-	-	28	143	21	155
-	+	-	1	140	12	166
-	-	+	0	--	2	170
+	-	-	5	149	30	192
+	+ or +		1	135	21	208
+	-	+	0	--	8	224

drugs = class IA, IB, IC, II, III agents or digitalis
BBB = bundle branch block
LIVCD = left intraventricular conduction delay
ms = milliseconds
ASVT = aberrant supraventricular tachycardia
VT = ventricular tachycardia
\+ = present
\- = absent

their mean ejection fraction was near normal (55%). Only three patients were being treated with antiarrhythmic agents, and two of these were receiving digitalis, which is less likely to depress conduction than the Class IA, IC, or III agents. The third patient had been taken off Amiodarone two weeks prior to electrophysiology study.

The average QRS width during SVT in patients with antiarrhythmic therapy or preexisting left intraventricular conduction delay was very nearly the same as the average width during SVT from patients without such conditions (Table 8). In contrast, the presence of drug therapy, preexisting bundle branch block or left intraventricular conduction delay did dramatically increase

mean QRS width during VT. For example, mean QRS width during VT in patients without drug therapy or intraventricular conduction abnormalities was 155 ms compared to a mean width of 224 ms in patients with bundle branch block plus drug therapy.

In the present study, a QRS width of greater than 160 ms was most useful in discriminating between aberrant SVT and VT regardless of drug therapy or intraventricular conduction abnormalities (Table 7). In fact, only one SVT had a QRS width exceeding 160 ms (Figure 16).

QRS Axis

During tachycardia, a normal or right axis was statistically significant for predicting aberrant SVT; whereas a left or northwest quadrant axis was statistically significant in predicting VT (Table 9). The axis criteria discriminated between SVT and VT better in tachycardias with a right bundle branch block contour than in tachycardias with a left bundle branch block contour (Table 10). In fact, none of the axis criteria were statistically significant for predicting SVT or VT in the tachycardias with a left bundle branch block contour.

The two most valuable criteria were an axis in the northwest quadrant and left axis deviation in tachycardias with a right bundle branch block contour, both of which indicated a diagnosis of VT. In fact, if all tachycardias with an axis in the northwest quadrant were diagnosed as ventricular in origin, the diagnosis would be correct 92 percent of the time. Moreover, about one in four (26%) VTs had such an axis, which made it a useful criterion for diagnosing VT. In contrast to previous reports which stated that aberrant SVT *never* displayed a highly abnormal axis in the northwest quadrant except in patients with preexisting bundle branch block (Akhtar et al., 1988, Caceres et al., 1987; Wellens et al., 1981), two SVTs in the present study had such an axis (Figures 17 A & B, 18 A, B, & C).

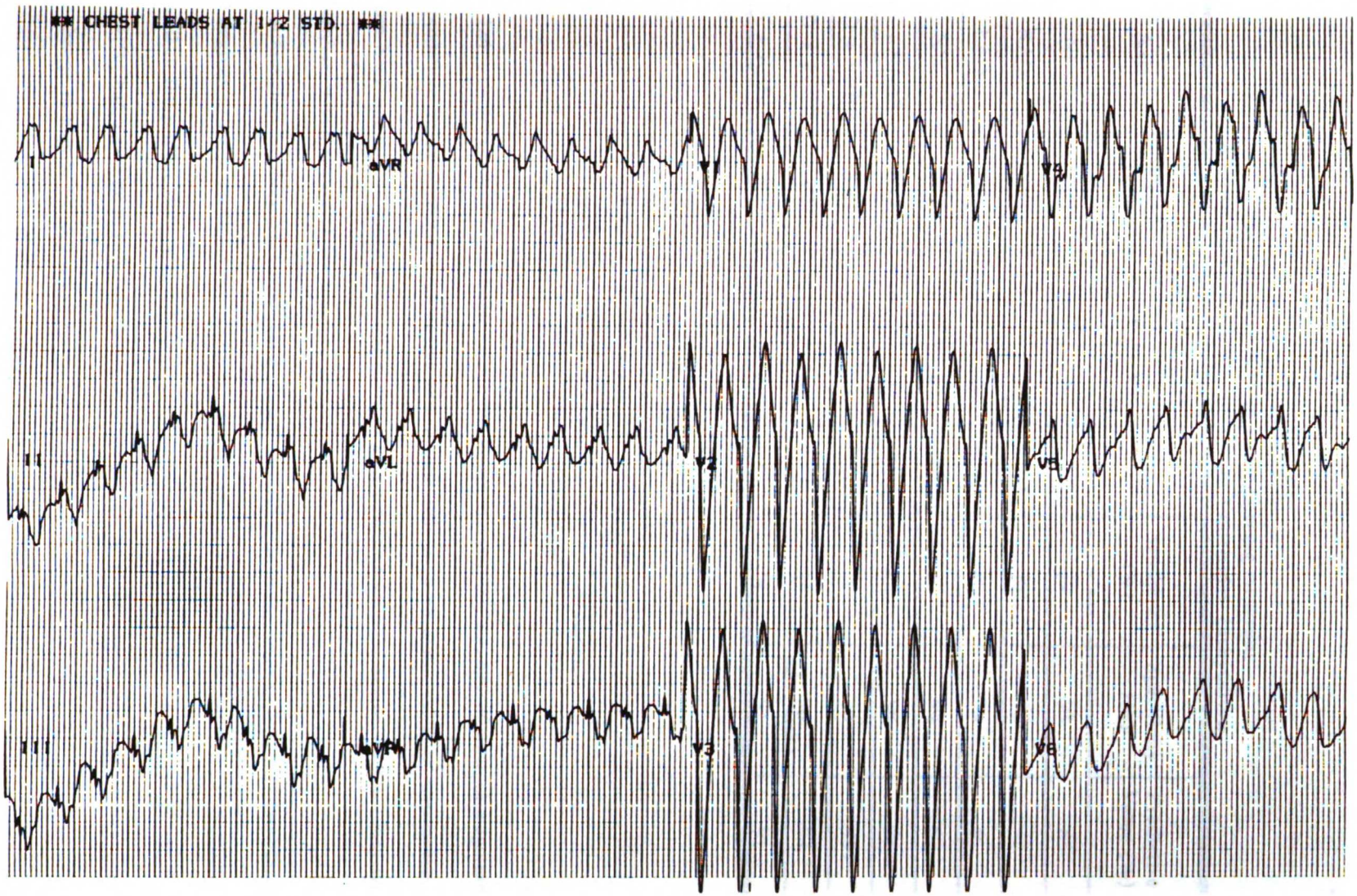


Figure 16. Orthodromic tachycardia with left bundle branch block-type aberration recorded from a 33 year old male without heart disease, preexisting bundle branch block, or drug therapy. QRS intervals measured 120 to 160 ms in all leads but one (lead II), in which the QRS measured 180 ms. The same patient had a second orthodromic tachycardia with right bundle branch block-type aberration and QRS widths ranging from 120 to 155 ms.

Table 9
Value of QRS Axis in Distinguishing Aberrant Supraventricular Tachycardia from Ventricular Tachycardia

Axis	Implied Diagnosis	HBE Diagnosis		P	Sensitivity (%)	Specificity (%)	Predictive Value (%)
		ASVT N = 35	VT N = 86				
NL	SVT	10	6	< .005	29	93	63
RAD	SVT	14	19	< .05	40	78	42
LAD	VT	9	39	< .05	45	74	81
NW	VT	2	22	< .01	26	94	92
NL + RBBB	SVT	6	1	< .005	27	98	86
RAD + RBBB	SVT	13	10	< .005	59	79	57
LAD + RBBB	VT	1	16	< .01	34	96	94
NW + RBBB	VT	2	21	< .005	44	91	91

HBE = His bundle electrogram
ASVT = aberrant supraventricular tachycardia
VT = ventricular tachycardia
NL = normal axis (0 to +89 degrees)
RAD = right axis deviation (+90 to +179 degrees)
LAD = left axis deviation (- 1 to - 90 degrees)
NW = northwest quadrant axis (- 91 to - 180 degrees)
RBBB = right bundle branch block contour

Left axis deviation in right bundle branch block-type tachycardias was also of value in diagnosing VT (predictive value = 94%). Additional axis criteria that were of some value in distinguishing between SVT and VT included a normal or right axis deviation in right bundle branch block-type tachycardias, which suggested SVT.

Although it has been suggested that a change in QRS axis from baseline rhythm to tachycardia of 40 degrees or more predicted VT (Griffith, Mickelwright, Linder, Ward & Camm, 1989), such an axis shift in the present study was unhelpful in distinguishing SVT from VT. In fact, 18 of the 35 SVTs (51%) had a change in axis from baseline of 40 degrees or more. Moreover, seven SVTs (20%) had a change in axis of 90 degrees or more.

Table 10
QRS Axis in Tachycardias with Right and Left Bundle Branch Block Contour

Axis	BBB Contour	HBE Diagnosis		P
		ASVT N = 35	VT N = 84*	
NL	RBBB	6	1	< .005
	LBBB	4	5	NS
RAD	RBBB	13	10	< .005
	LBBB	1	9	NS
LAD	RBBB	1	16	< .01
	LBBB	8	21	NS
NW	RBBB	2	21	< .005
	LBBB	0	1	NS

* two of the 86 VTs had indeterminate bundle branch block contour
NL = normal axis (0 to +89 degrees)
RAD = right axis deviation (+90 to +179 degrees)
LAD = left axis deviation (- 1 to - 90 degrees)
NW = northwest quadrant axis (- 91 to - 180 degrees)
RBBB = right bundle branch block contour, N = 69
LBBB = left bundle branch block contour, N = 50
HBE = His bundle electrogram
ASVT = aberrant supraventricular tachycardia
VT = ventricular tachycardia

Axis information provided from a single lead II or aVR was helpful in distinguishing aberrant SVT from VT (Figure 19). Localization of the axis to an abnormal hemisphere by observing a negative QRS in lead II or a positive QRS in aVR was more helpful in diagnosing VT than was localization of the axis to a normal hemisphere in diagnosing aberrant SVT. For example, nearly 90 percent of tachycardias with a negative QRS in lead II or a positive QRS in aVR were ventricular in origin (predictive value = 88%). Moreover, the predictive value of observing a negative QRS in lead II plus a positive QRS in aVR for diagnosing VT was 94 percent. The best

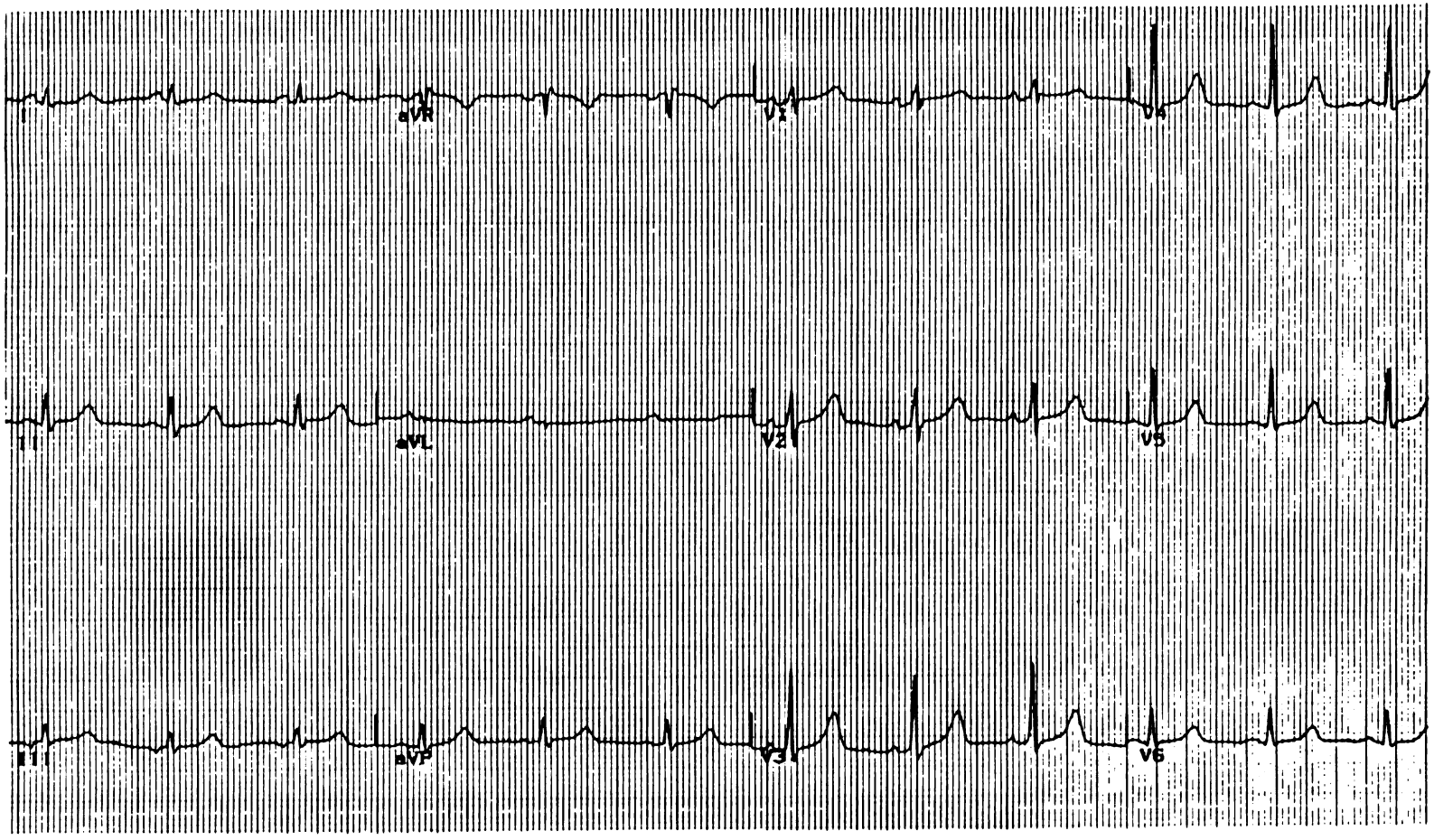


Figure 17a. Baseline ECG from a 55 year old woman without heart disease or preexisting bundle branch block, and a normal axis (+60°) during sinus rhythm who developed supraventricular tachycardia with an axis in the highly abnormal northwest quadrant (see Figure 17b, next page).

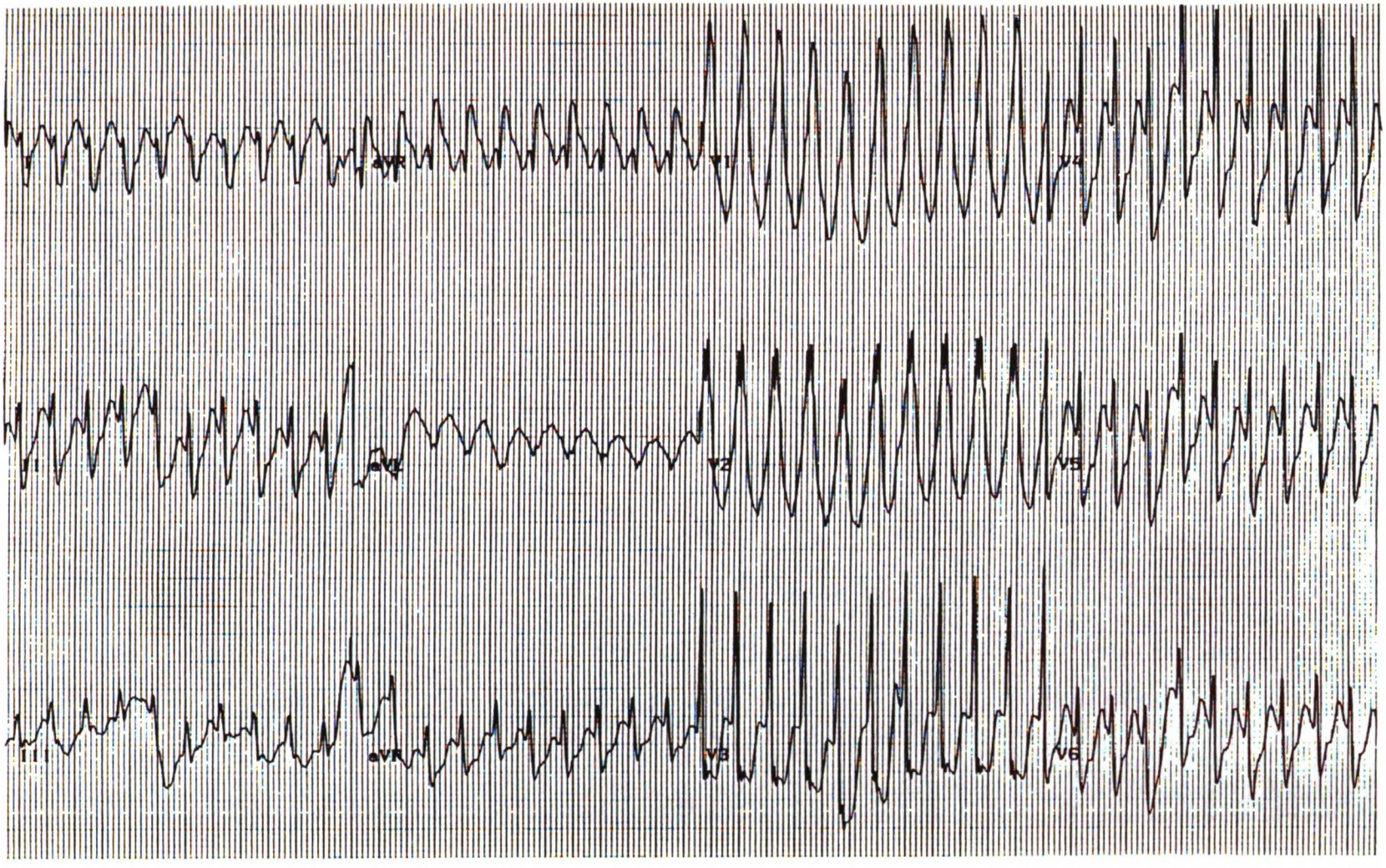


Figure 17b. AV nodal reentrant tachycardia in the same patient with an unusual pattern of right bundle branch block-type aberration (i.e., a monophasic R wave pattern in V_1). QRS axis during tachycardia is - 175 to - 180 degrees, a change in axis of nearly 130 degrees from baseline.

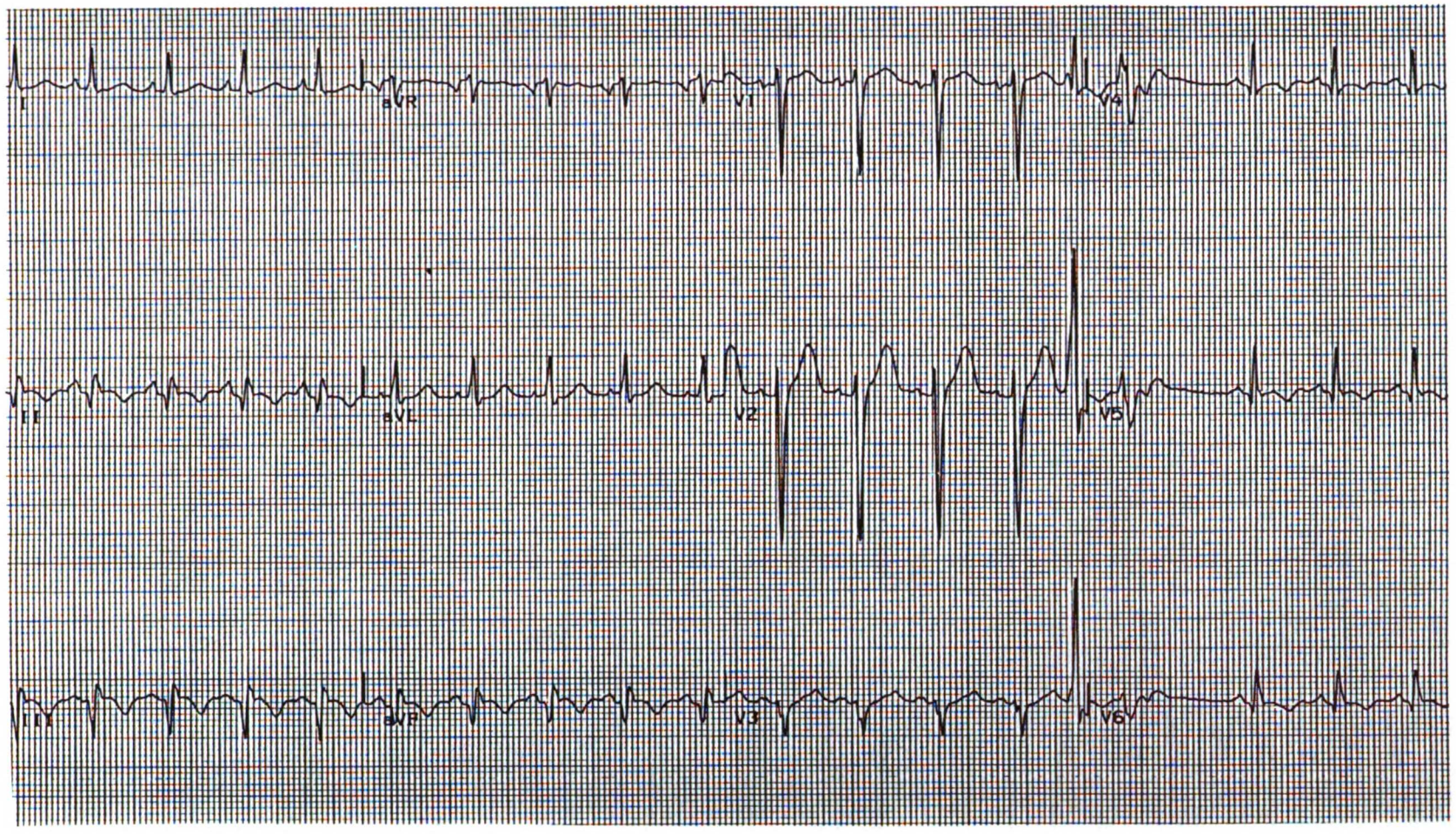


Figure 18a. Baseline ECG recorded from a 71 year old male with a history of a previous inferior wall myocardial infarction, and an axis of about −10 degrees during sinus rhythm, who developed supraventricular tachycardia with an axis in the northwest quadrant (see Figure 18b, next page).

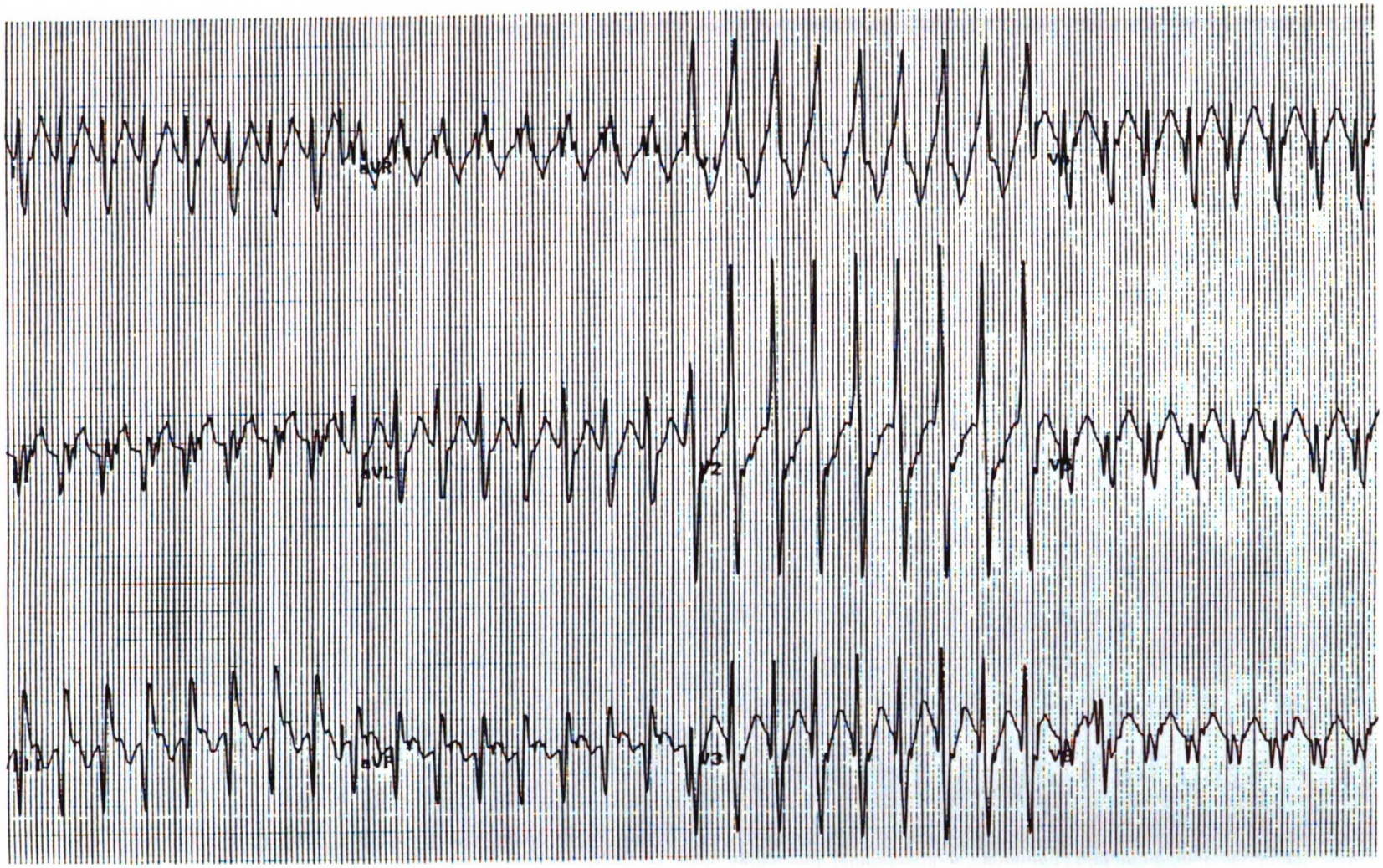


Figure 18b. Atrial over-drive pacing in the same patient with an unusual pattern of right bundle branch block-type aberration, and an axis of −100 degrees.

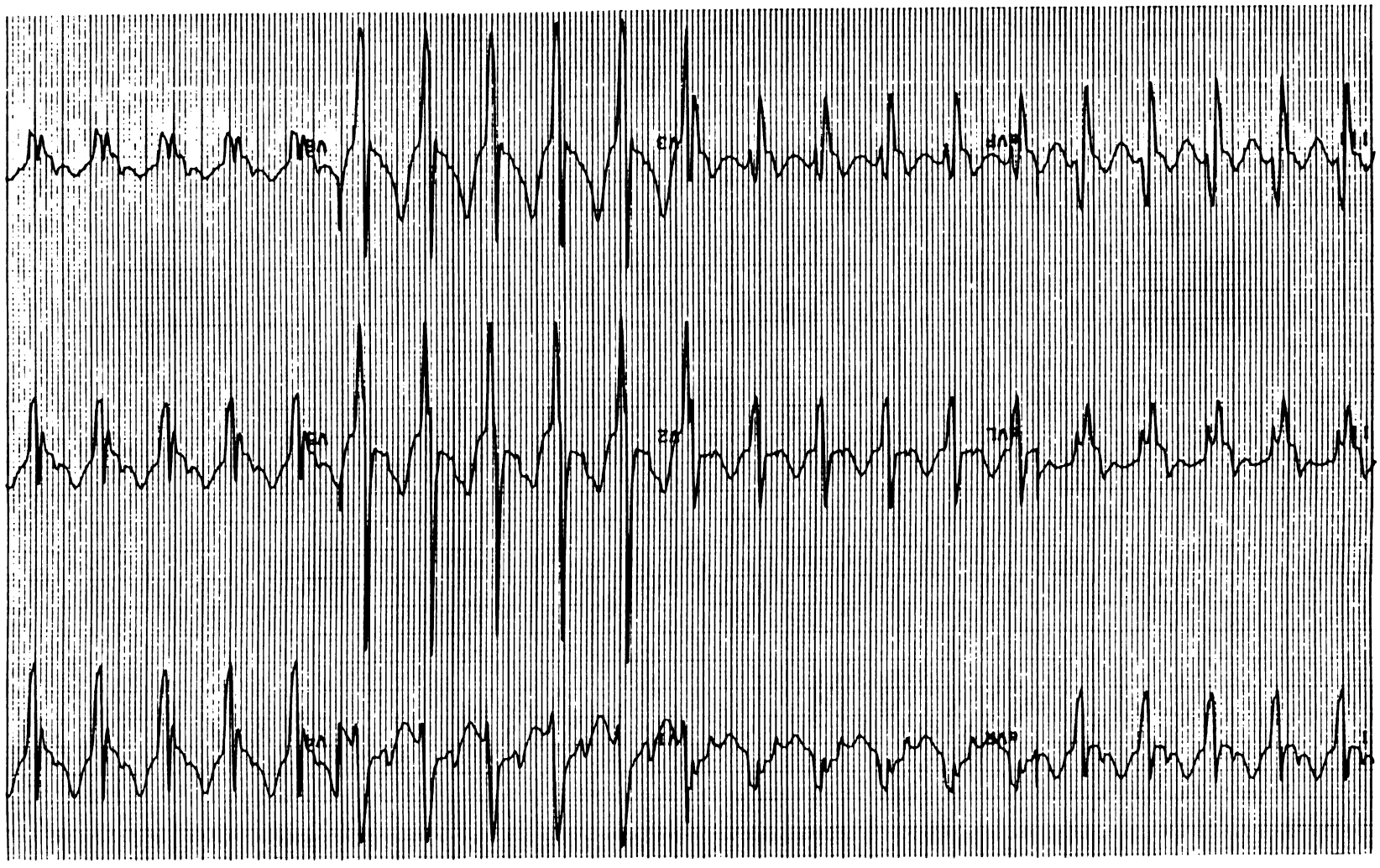
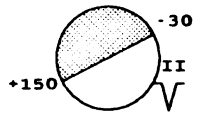
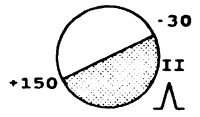
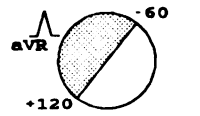
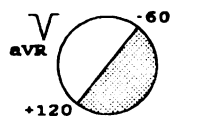
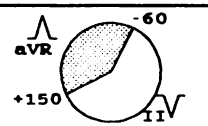
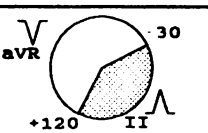


Figure 18c. Sinus tachycardia with aberrant conduction in the same patient showing the same markedly abnormal QRS axis.

Figure 19
Value of Axis Information from Single or Dual Limb Lead in Distinguishing Aberrant Supraventricular from Ventricular Tachycardia

QRS Polarity and Lead(s)1	Implied Axis Range	HBE Diagnosis		P	Sensitivity (%)	Specificity (%)	Predictive Value (%)	Predictive Accuracy (%)
		ASVT N = 35	VT N = 86					
Negative in II		8	57	< .000	66	77	88 (VT)	67
Positive in II		24	26	< .001	69	70	48 (ASVT)	
Positive in aVR		7*	51*	< .001	61	78	88 (VT)	60
Negative in aVR		21*	29*	< .005	66	66	42 (ASVT)	
Negative in II + positive in aVR		3*	46*	< .000	55	91	94 (VT)	54
Positive in II + negative in aVR		19*	20*	< .000	59	76	49 (ASVT)	

HBE = His bundle electrogram; ASVT = aberrant supraventricular tachycardia; VT = ventricular tachycardia
* Lead aVR had five missing values (in aVR, SVT = 32; VT = 84). Shaded area indicates axis range implied by lead II and/or aVR.

dual lead combination for diagnosing wide QRS complex tachycardia from axis criteria was leads I and aVF which had a predictive accuracy of 70 percent (Table 9).

QRS Morphology

Right bundle branch block-type tachycardias in MCL_1 or V_1. QRS patterns which were most discriminating in right bundle branch block-type tachycardias in V_1 and MCL_1 were a taller left peak or biphasic Rs or qR pattern suggestive of VT and a rR′ or rsR′ pattern suggestive of aberrant SVT (Figure 20). Tachycardias with a taller right peak or equal peaks in MCL_1 or V_1 were not helpful in distinguishing aberrant SVT from VT (Figure 21). Although QRS morphology often varied during wide complex tachycardia between MCL_1 and V_1, there were no instances in which the left peak was taller in one lead while the right peak was taller in the other, or when the peaks were equal in one while taller in the left or right in the other. What did differ occasionally between the two leads was the presence of a peak pattern in one lead and the absence of a peak pattern in the other (Figure 22).

A rR′ or rsR′ pattern was suggestive of aberrant SVT, however, such patterns were also seen in six VTs in MCL_1, and in seven VTs in V_1 (Figures 20, 23 A-C). Thus, the predictive value for the observation of a triphasic rsR′ or bimodal rR′ from MCL_1 or V_1 in diagnosing aberrant SVT was only about 70 percent. One case of VT which exhibited the classic features of right bundle branch block-type aberration occurred in a patient with fascicular VT (Figure 24). Two additional features of rR′ or rsR′ contours that resulted in confusion in distinguishing aberrant SVT from VT were observed in the study. First, it was often difficult to determine whether a complex in V_1 was a rsR′ contour (suggesting aberrant SVT) or a qR pattern (suggesting VT) (Figure 25). Second, triphasic patterns were often rate-related. That is, a clear-cut rsR′ pattern might be seen at slower tachycardia rates or during the first beat of a tachycardia, but such discrete QRS details were lost at faster rates resulting in monophasic R waves (Figures 26a-26d, 27a & 27b).

Figure 20
QRS Morphology in MCL_1 and V_1 During Wide QRS Complex Tachycardia

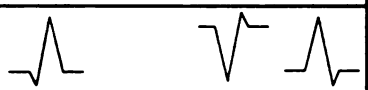
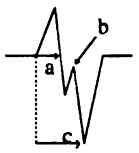
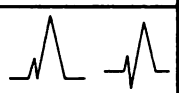
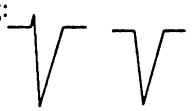
QRS Morphology	Lead	HBE Diagnosis		P
		ASVT N = 35	VT N = 86	
Monophasic R 	MCL_1	7	5	NS
	V_1	5	15	NS
Taller left peak 	MCL_1	0	9	< .05
	V_1	0	12	< .025
Biphasic qR, Qr or Rs 	MCL_1	0	18	< .005
	V_1	0	9	< .05
Biphasic rS with one or more of the following: (a) R > 30 ms (b) Slurred or notched S descent (c) QRS onset to S nadir > 60ms 	MCL_1	0	27	< .000
	V_1	0	31	< .000
Bimodal rR′ or triphasic rsR′ 	MCL_1	11	6	< .005
	V_1	16	7	< .000
Biphasic rS or Q with all of the following: (a) R ≤ 30 ms (b) Straight S descent (c) QRS onset to S nadir ≤ 60 ms 	MCL_1	12	8	< .005
	V_1	13	4	< .000
No diagnostic morphology	MCL_1	5	11	
	V_1	1	7	

Figure 21
Value of QRS Peak Deflections in Distinguishing Aberrant Supraventricular from Ventricular Tachycardia

Peak Deflection	Lead	HBE Diagnosis		P
		ASVT	VT	
Taller left peak 	MCL_1	0	11	< .025
	V_1	0	12	< .025
Taller right peak 	MCL_1	4	6	NS
	V_1	3	5	NS
Equal peaks 	MCL_1	1	4	NS
	V_1	0	3	NS

HBE = His bundle electrogram
ASVT = aberrant supraventricular tachycardia
VT = ventricular tachycardia

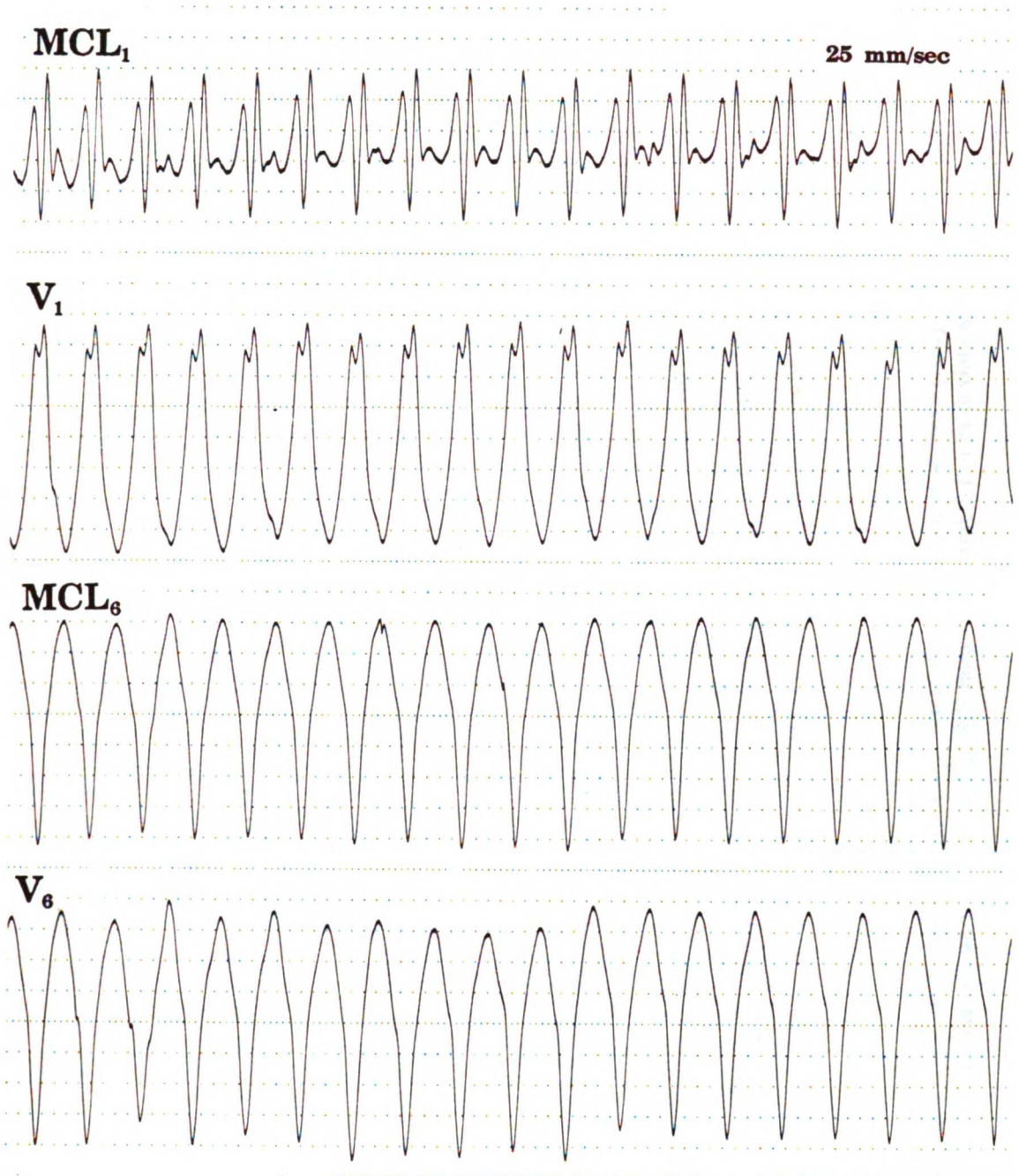


Figure 22. Tracings recorded during VT illustrating a taller right peak pattern in V_1 while MCL_1 shows a rsR′ pattern. Although the right peak is taller in the triphasic complex in MCL_1, it does not possess the classic features of the "rabbit ear" pattern which includes a monophasic R wave containing two distinct peaks. QRS morphology erroneously suggests aberrant SVT in MCL_1, and in V_1 morphology is unhelpful in making the diagnosis. In MCL_6 and V_6, however, a QS pattern is diagnostic of VT.

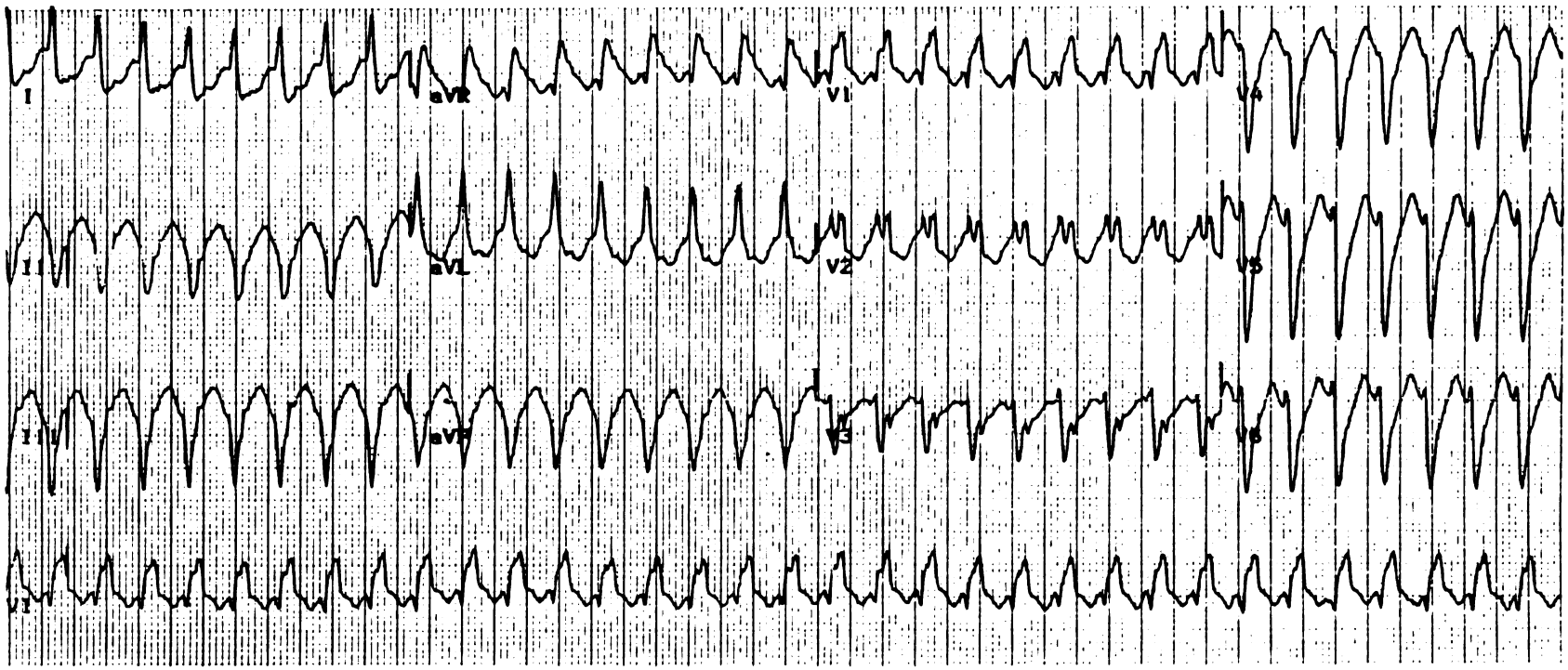


Figure 23a. Example of VT with a rsR′ pattern in V_1. While the rsR′ pattern in V_1 erroneously suggests aberrant SVT, two other ECG criteria indicate VT. They include an rS pattern in V_6 (R:S ratio < 1.0), and, left axis deviation during right bundle branch block-type tachycardia.

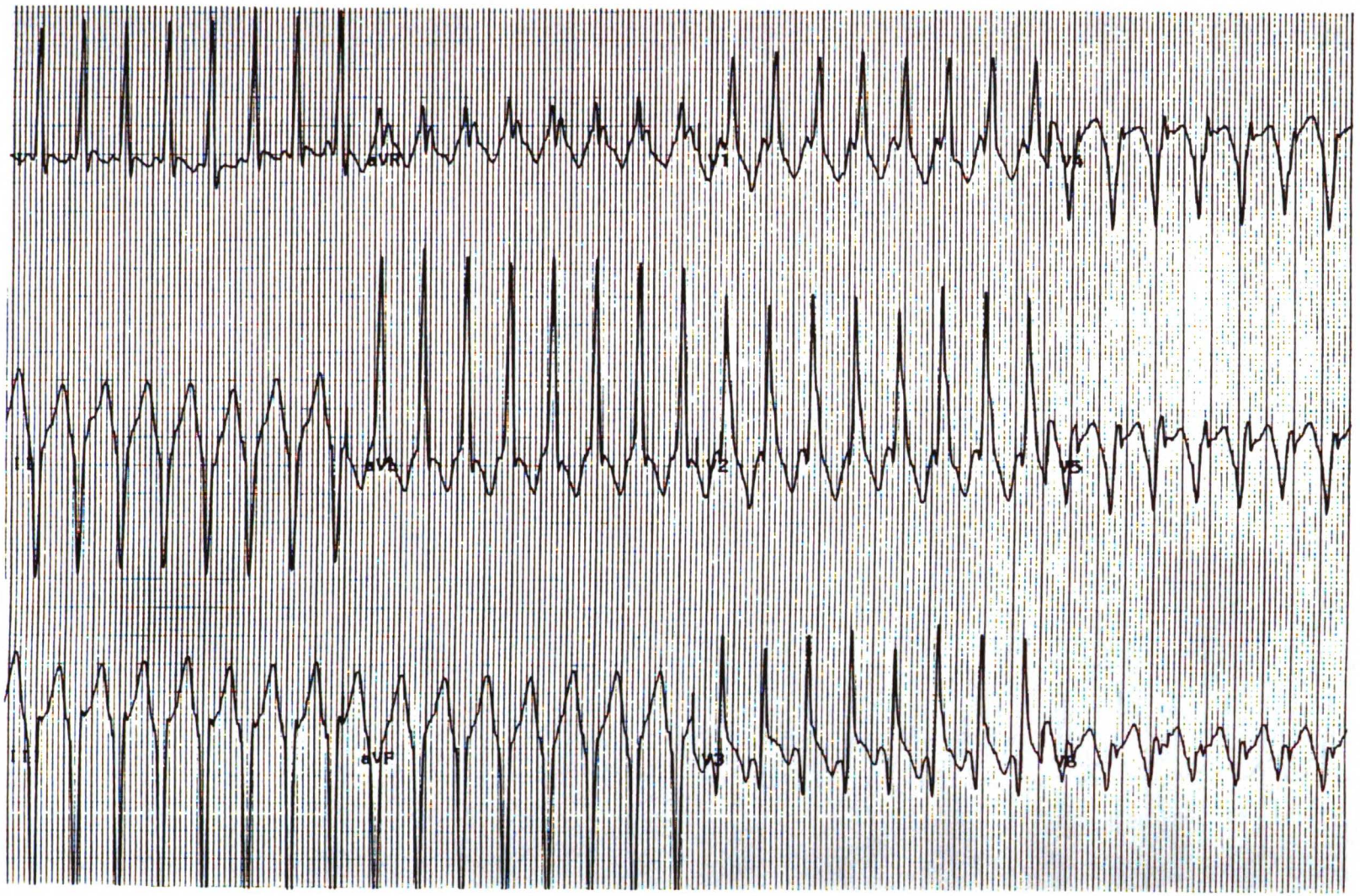


Figure 23b. Second example of VT with a rsR′ pattern in V_1. While the rsR′ pattern in V_1 erroneously suggests aberrant SVT, two other ECG criteria indicate VT. They include a QS pattern in V_6, and, left axis deviation during right bundle branch block-type tachycardia.

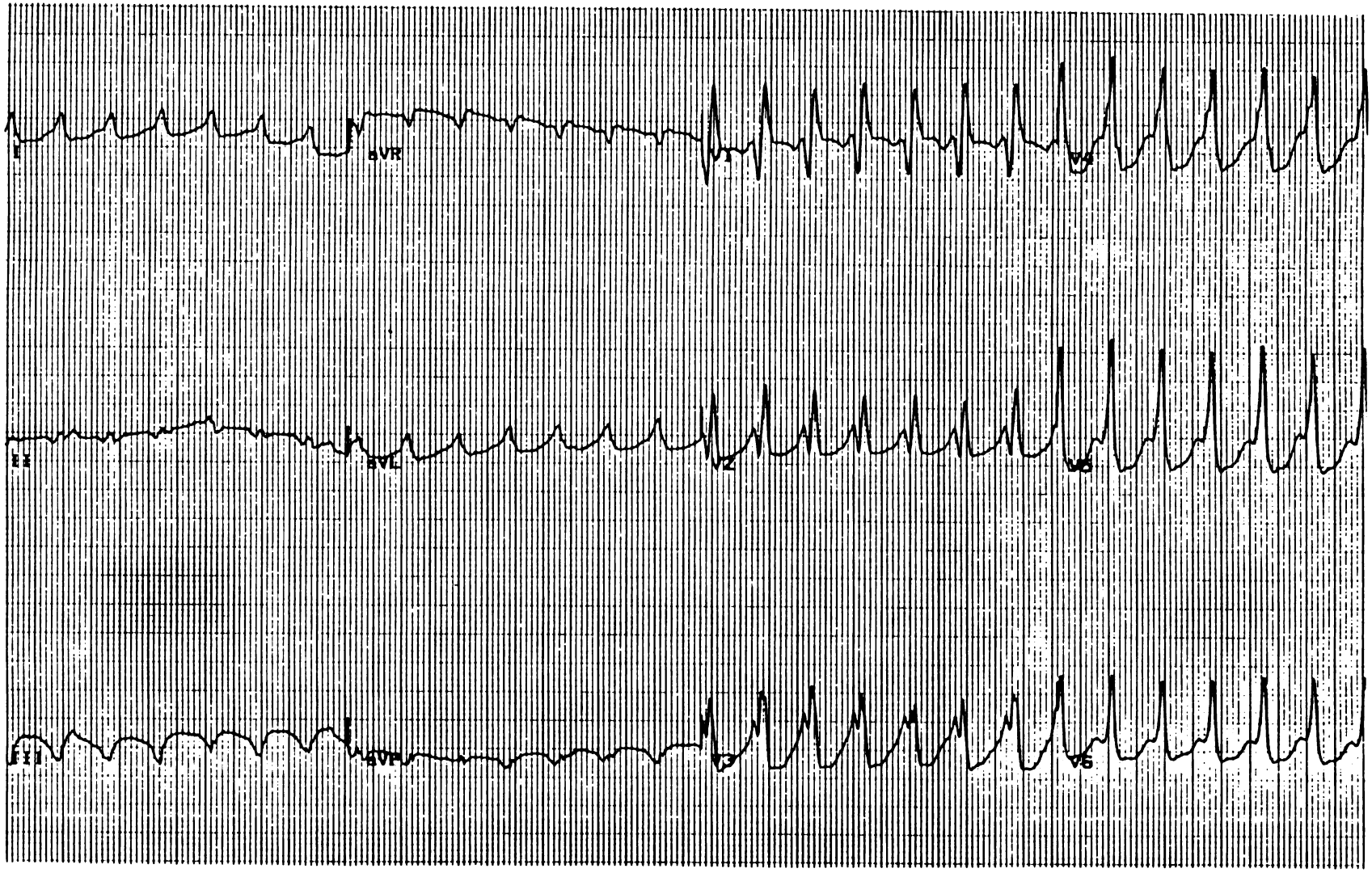


Figure 23c. Third example of VT with a rsR′ pattern in V_1. Ventricular tachycardia in a 54 year old male with a prior inferior wall myocardial infarction treated with Amiodarone. An rsR′ pattern is present in V_1, and a Rs pattern in V_6 is unhelpful in making the diagnosis.

Figure 24. Ventricular tachycardia in a 42 year old male without heart disease and a history of exercise-induced tachycardia. The tracing is a continuous rhythm strip of all twelve leads at 25 mm/sec paper speed. This VT, which was reproducibly and easily inducible from either atrial or ventricular extrastimulation, most likely represented a triggered mechanism. The tachycardia was terminated with verapamil, and its rate was directly proportional to the pacing drive cycle length which induced it, both characteristics typical of a triggered mechanism. QRS morphology identical to right bundle branch block with left anterior hemiblock indicated that the site of origin was located on or near the posterior fascicle of the left bundle branch. The atrial-ventricular relationship varied between 2:1 ventriculo-atrial block and AV dissociation with fusion and ventricular capture beats.

Figure 25. Ventricular tachycardia illustrating the difficulty in judging whether QRS morphology is a rsR′ pattern (suggestive of aberrant SVT) or a qR pattern (suggestive of VT). The Q waves from V_2 to V_4 make it likely that the V_1 complex is a qR pattern as well with the preceding T wave superimposed on the QRS which results in a triphasic-appearing complex.

Figure 26a-d. Rate-dependent morphological criteria during SVT. **a.** Baseline sinus rhythm illustrating the classic triphasic contour of right bundle branch block and a sizable initial R wave. **b.** Orthodromic tachycardia at a rate of 169 bpm with a rR′ pattern and a smaller amplitude initial R wave than during sinus rhythm. **c.** Orthodromic tachycardia at a rate of 198 bpm following isuprel infusion. Notice that QRS morphology in V_1 is a taller right peak pattern which is unhelpful in distinguishing aberrant SVT from VT. **d.** Continuous rhythm strip of MCL_1 and V_1 in the same patient showing the initiation of SVT following isuprel infusion at a rate of 214 bpm. Although initial complexes exhibit a triphasic contour, such discrete QRS details are lost at this faster rate and subsequent complexes become monophasic.

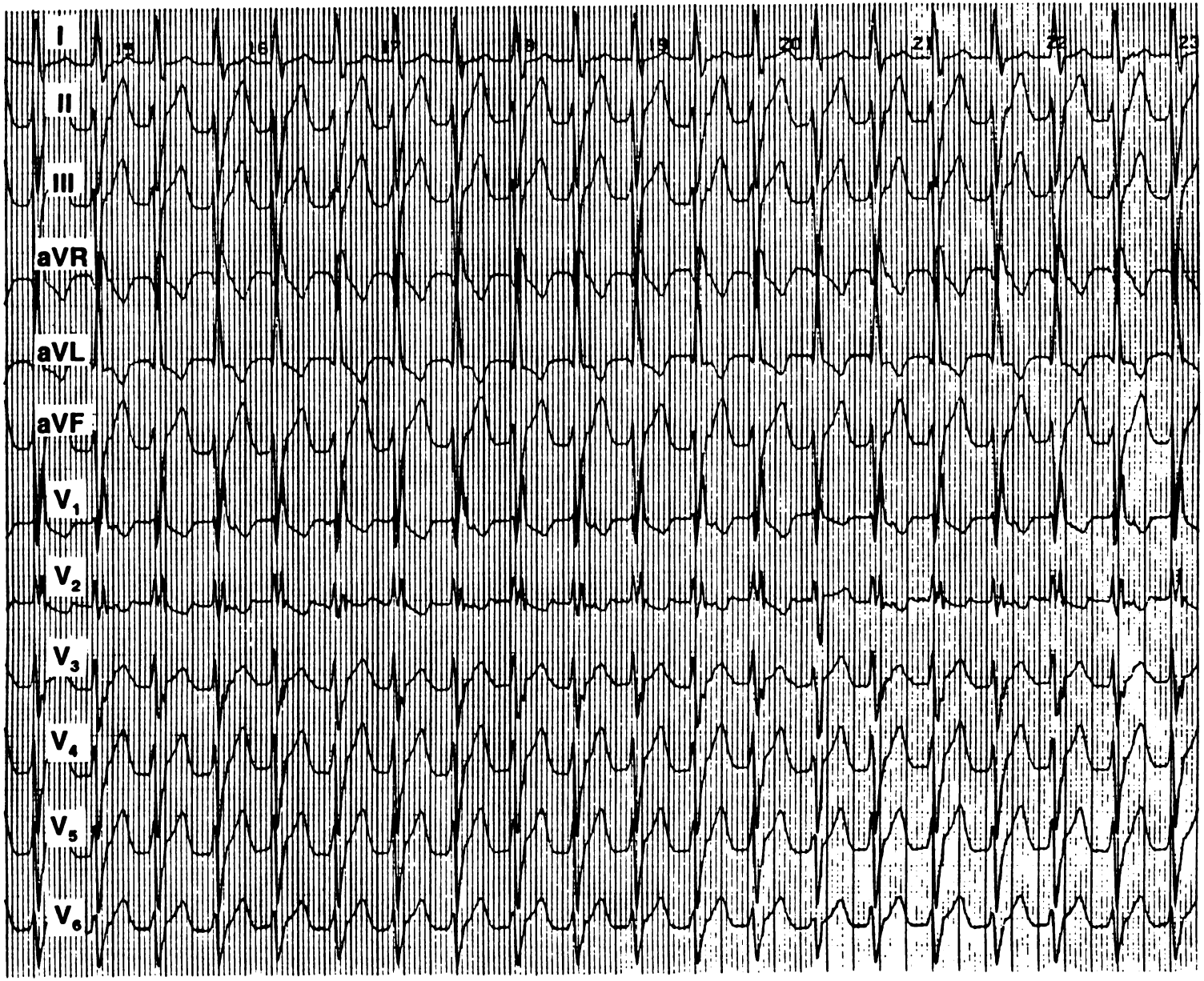


Figure 24

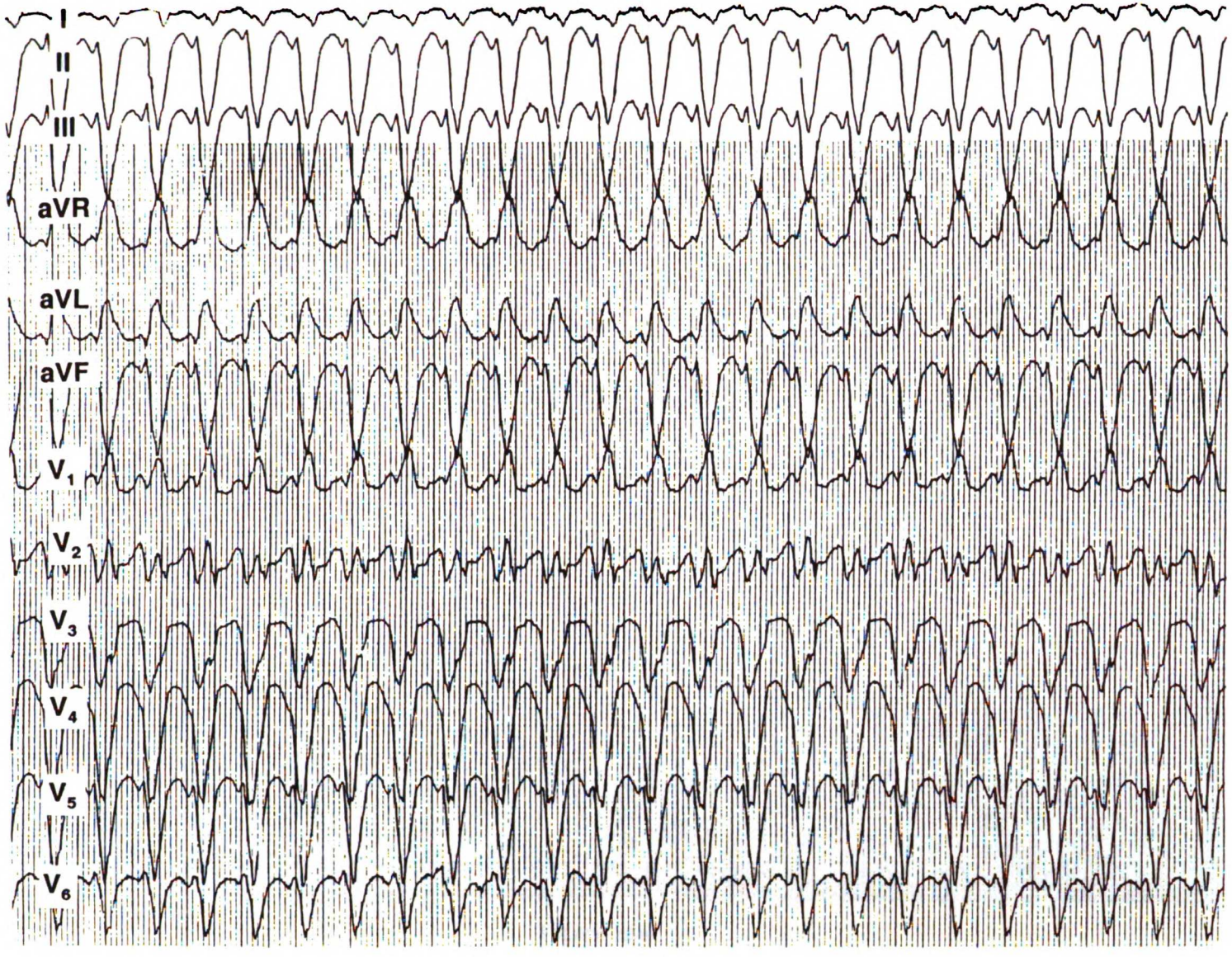


Figure 25

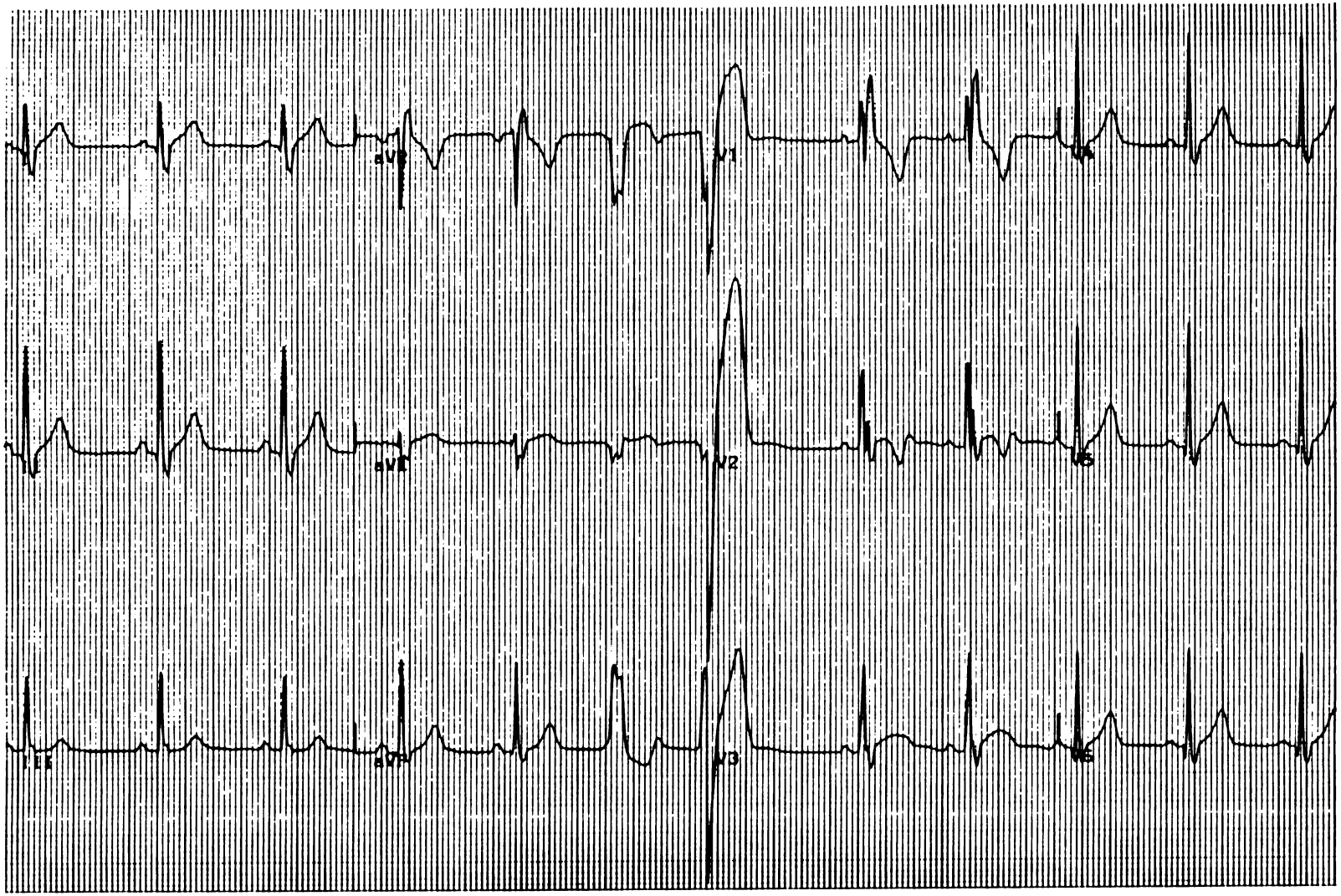


Figure 26a

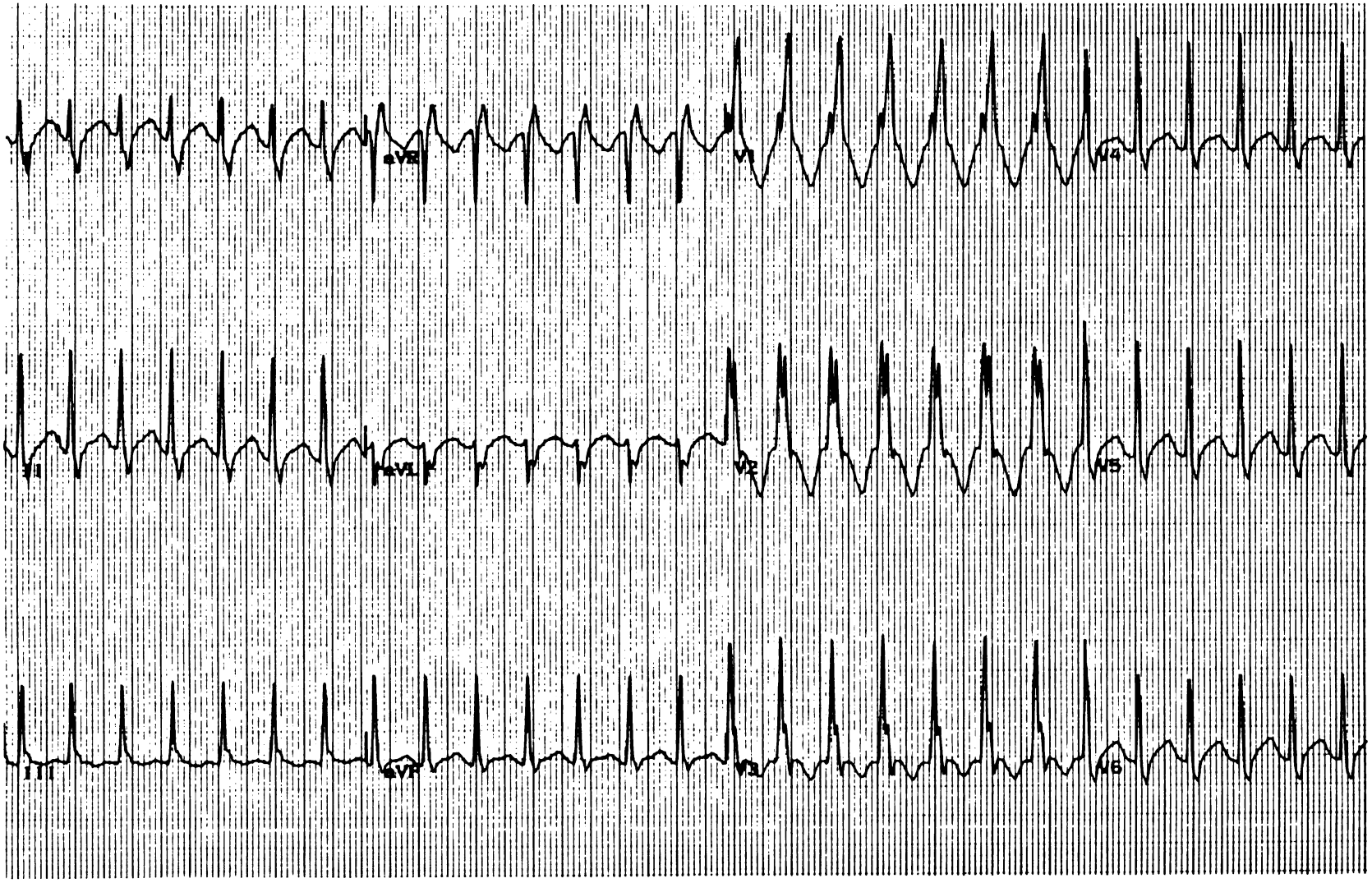


Figure 26b

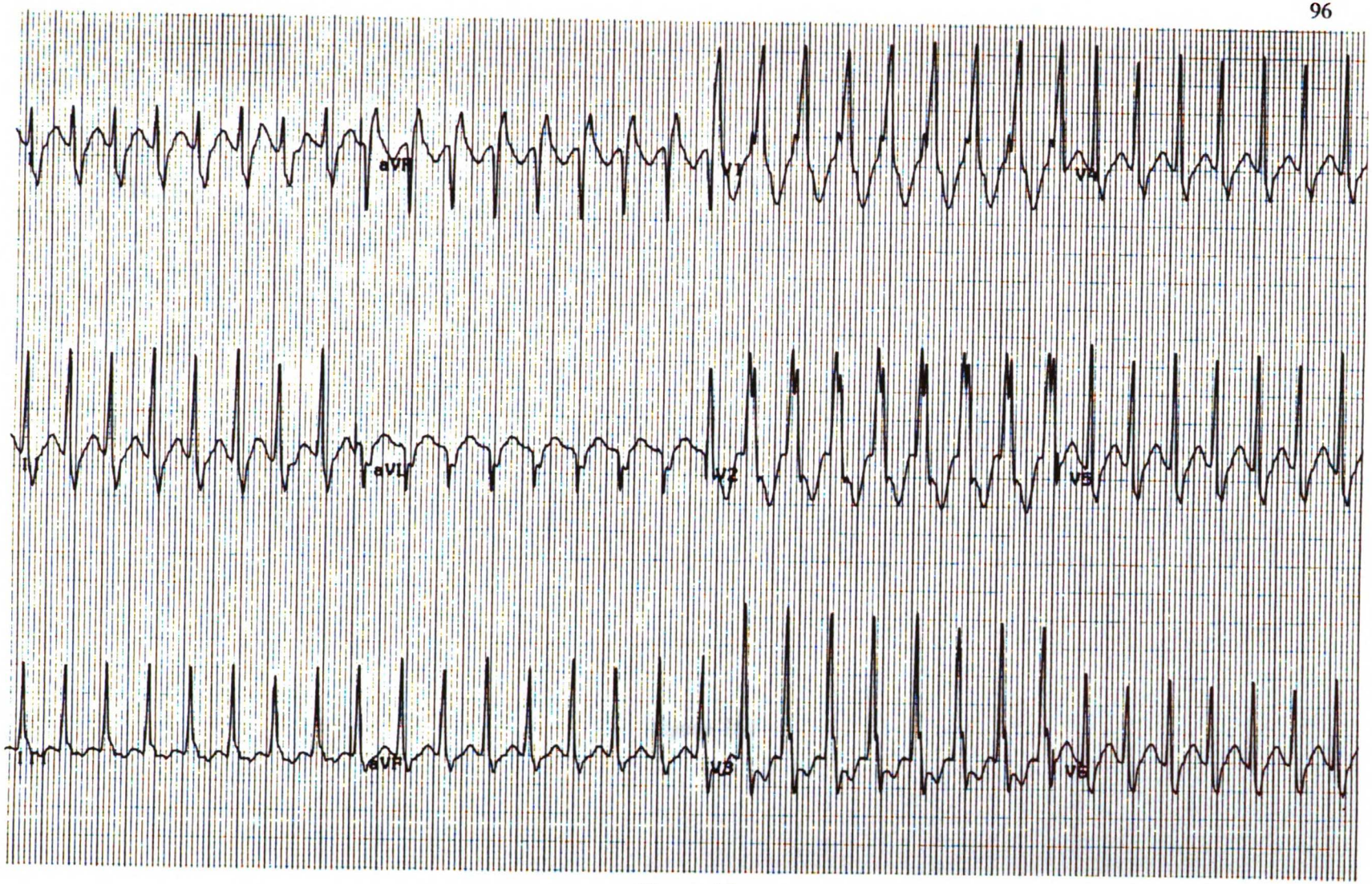


Figure 26c

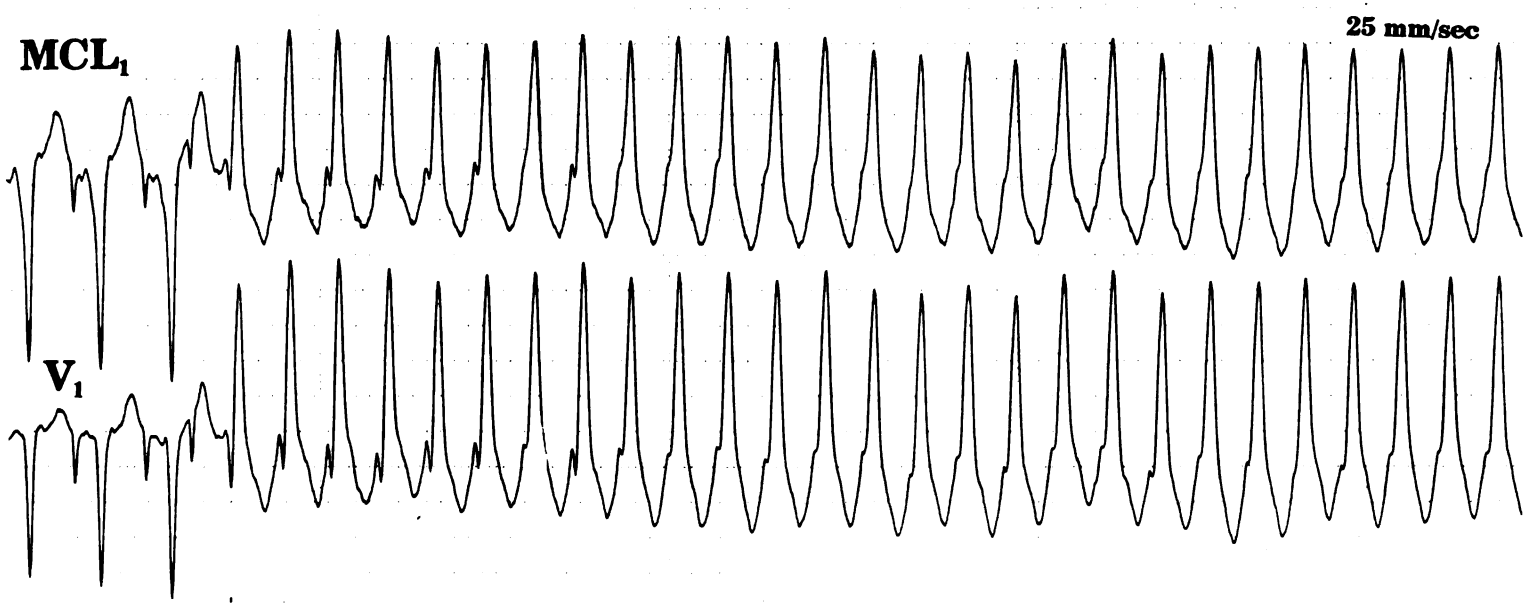


Figure 26d

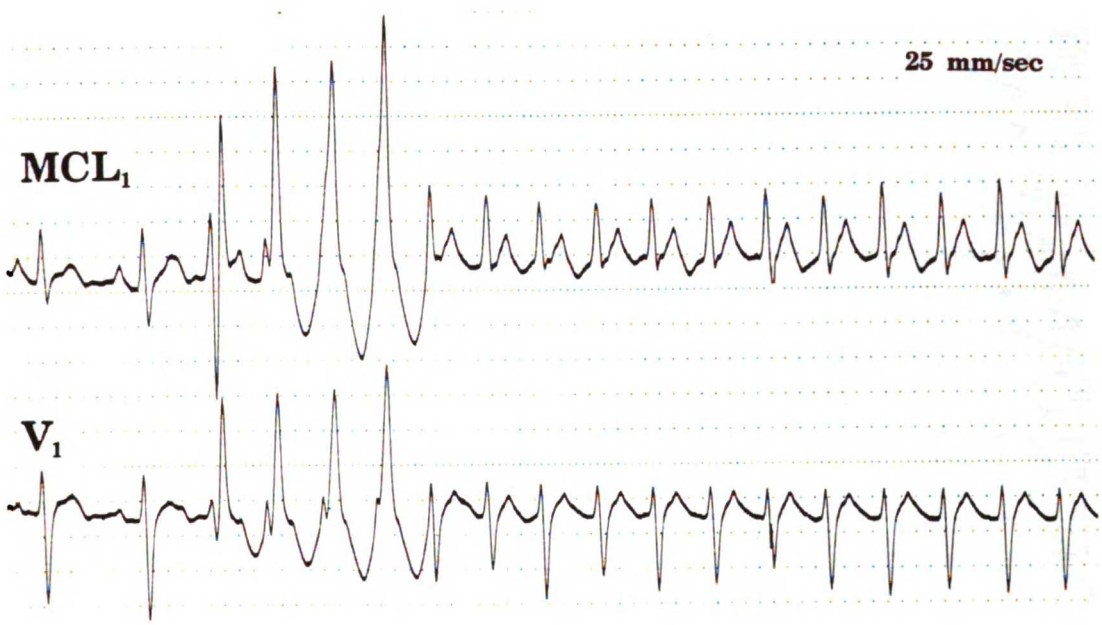


Figure 27a

Figure 27. **a.** Simultaneous recording of MCL_1 and V_1 showing a burst of atrial tachycardia in which the first four beats are aberrantly conducted. This tracing demonstrates the importance of noting QRS morphology in the first beat of a train of rapid aberrant complexes, because QRS details (i.e., triphasic rsR′ contour) may be lost in the barrage of ensuing wide complexes. **b.** 12-lead ECG from the same patient during atrial tachycardia with right bundle branch block-type aberration illustrating a monophasic R wave pattern in V_1 except for the complex following the non-aberrant beat (arrow) which suggests a triphasic contour.

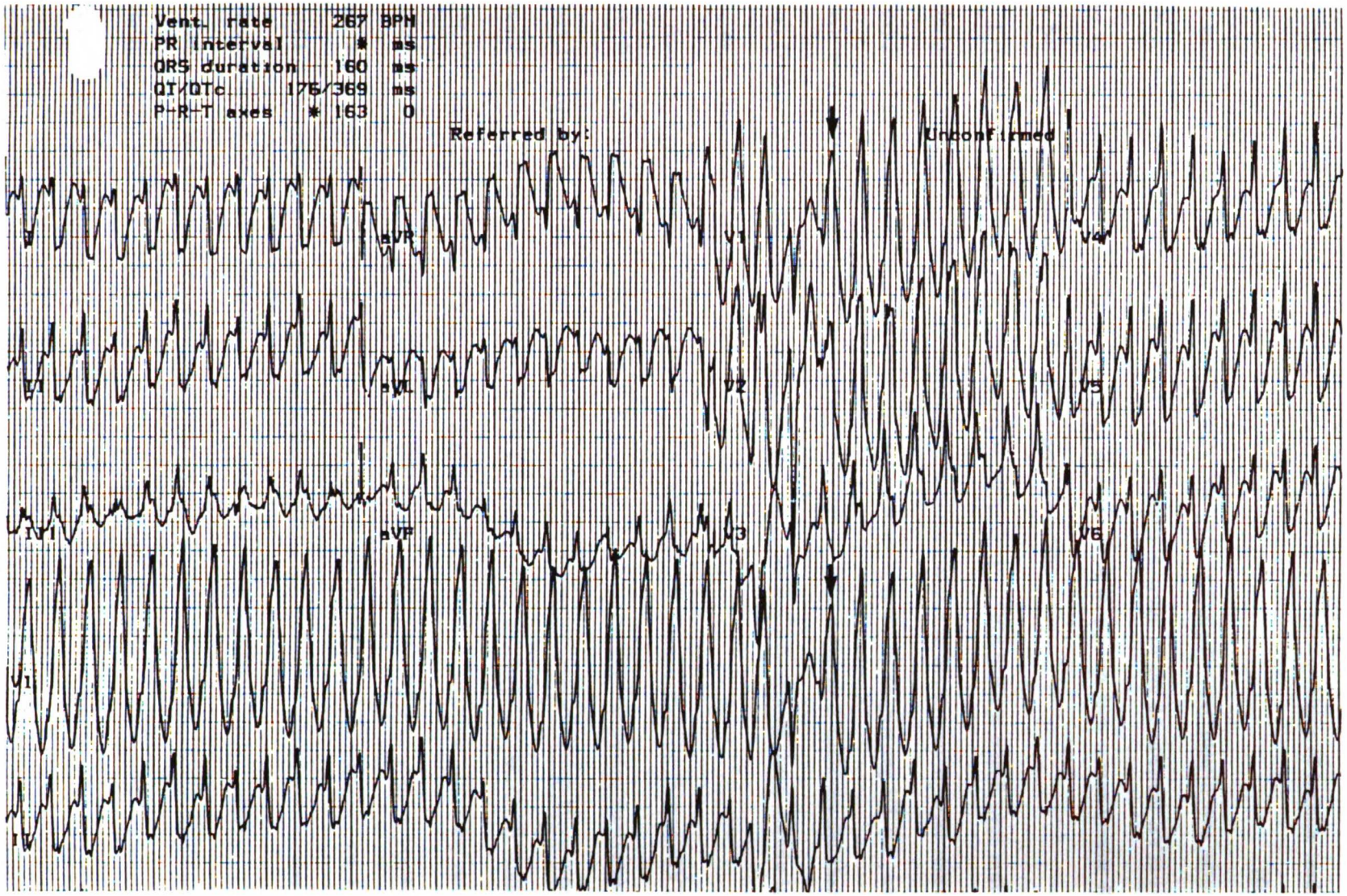

Vent. rate 267 BPM
PR interval * ms
QRS duration 160 ms
QT/QTc 176/369 ms
P-R-T axes * 163 0
Referred by:
Unconfirmed
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

Figure 27b

In contrast to Wellens' findings (1978, 1982) that a monophasic R wave pattern was never observed during aberrant SVT, such a pattern was observed in seven of the 35 aberrant SVTs in MCL_1 and in five SVTs in V_1 (Figures 17 B, 18 B & C, 27 A & B). In fact, a monophasic R wave pattern was not statistically significant for predicting VT.

Left bundle branch block-type tachycardias in MCL_1 or V_1. QRS patterns which were most discriminating in complexes with a left bundle branch block contour in MCL_1 and V_1 were those that exhibited the criteria described by Kindwall and associates (1988) for diagnosing VT (i.e., an R wave greater than 30 ms, slurred or notched S descent, or QRS onset to S nadir greater than 60 ms) (Figure 28). In fact, not one of the 35 aberrant SVTs had any one of these criteria in MCL_1 or V_1 (Figure 20).

Absence of the Kindwall criteria in MCL_1 or V_1 was less helpful in diagnosing aberrant SVT (Figure 20); however, the criteria described by Kindwall and associates (1988) includes observation of these findings in V_1 *or* V_2 as well as noting any Q wave during left bundle branch block-type tachycardia in V_6. When the criteria was searched for in all three conventional precordial leads (i.e., V_1, V_2, and V_6), the proportion of correct diagnoses that could be made from observing one or more of the criteria was 94 percent (sensitivity = 91%; specificity and predictive value = 100%) (Figure 29). When the criteria was searched for in the two modified precordial leads (i.e., MCL_1 and MCL_6), the proportion of correct diagnoses that could be made from observing one or more of the criteria was 88 percent (sensitivity = 83%; specificity and predictive value = 100%).

Only three of the 35 left bundle branch block-type VTs exhibited absence of the Kindwall criteria in V_1, V_2, and V_6. Two of these VTs used the His-Purkinje conduction system; that is, they were bundle branch reentry VTs which would be expected to look identical to SVT with left

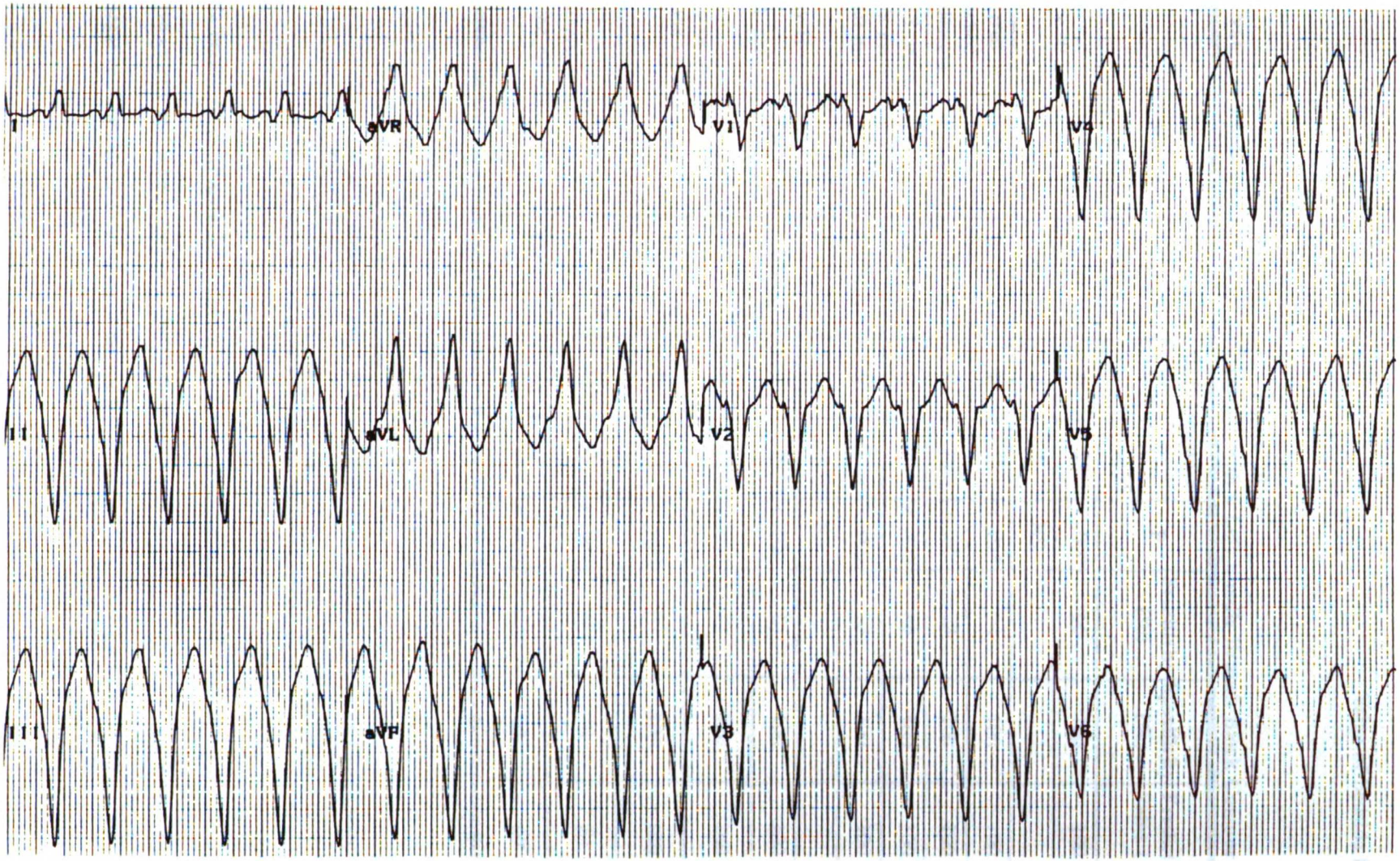
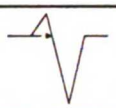
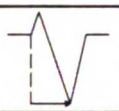


Figure 28. QRS morphology during left bundle branch block contour tachycardia highly suggestive of VT (Kindwall et al., 1988). This VT has an R wave greater than 30 ms in V_1, a QRS onset to S nadir of greater than 60 ms in V_1 and V_2, and a monophasic Q in V_6.

Figure 29
Value of Kindwall Criteria in Tachycardias with a Left Bundle Branch Block Contour

Criteria	Lead	HBE Diagnosis		P	Sensitivity (%)	Specificity (%)	Predictive value (%)
		ASVT N = 13	VT N = 35				
R > 30 ms 	V_1 or V_2	0	23	< .000	66	100	100
	MCL_1	0	8	NS			
Slurred or notched S descent 	V_1 or V_2	0	12	< .025	34	100	100
	MCL_1	0	10	< .05	28	100	100
QRS onset to S nadir > 60 ms 	V_1 or V_2	0	31	< .000	89	100	100
	MCL_1	0	27	< .000	75	100	100
Any Q 	V_6	0	8	NS			
	MCL_6	0	19	< .001	53	100	100
One or more of the criteria	V_1, V_2, & V_6	0	32	< .000	91	100	100
	MCL_1^* & MCL_6	0	30	< .000	83	100	100
Absence of all of the criteria	V_1, V_2, & V_6	13	3†‡	< .000	100	91	81
	MCL_1^* & MCL_6	12**	6‡	< .000	100	83	67

HBE = His bundle electrogram
ASVT = aberrant supraventricular tachycardia
VT = ventricular tachycardia

* Note: MCL_2 was not recorded
** One of the thirteen SVTs with left bundle branch block aberration in V_1 had a right bundle branch block configuration in MCL_1
† In one VT labelled as having no criteria present in V_1, V_2 or V_6, lead V_2 was missing
‡ Two VTs with negative Kindwall criteria were bundle branch reentry VTs

bundle branch block (Figure 30). The third VT had missing data from V_2, which may have provided positive criteria had it been recorded. When the criteria was searched for in MCL_1 and MCL_6, six VTs had negative Kindwall criteria. Two of the six VTs were bundle branch reentry mechanisms, and two

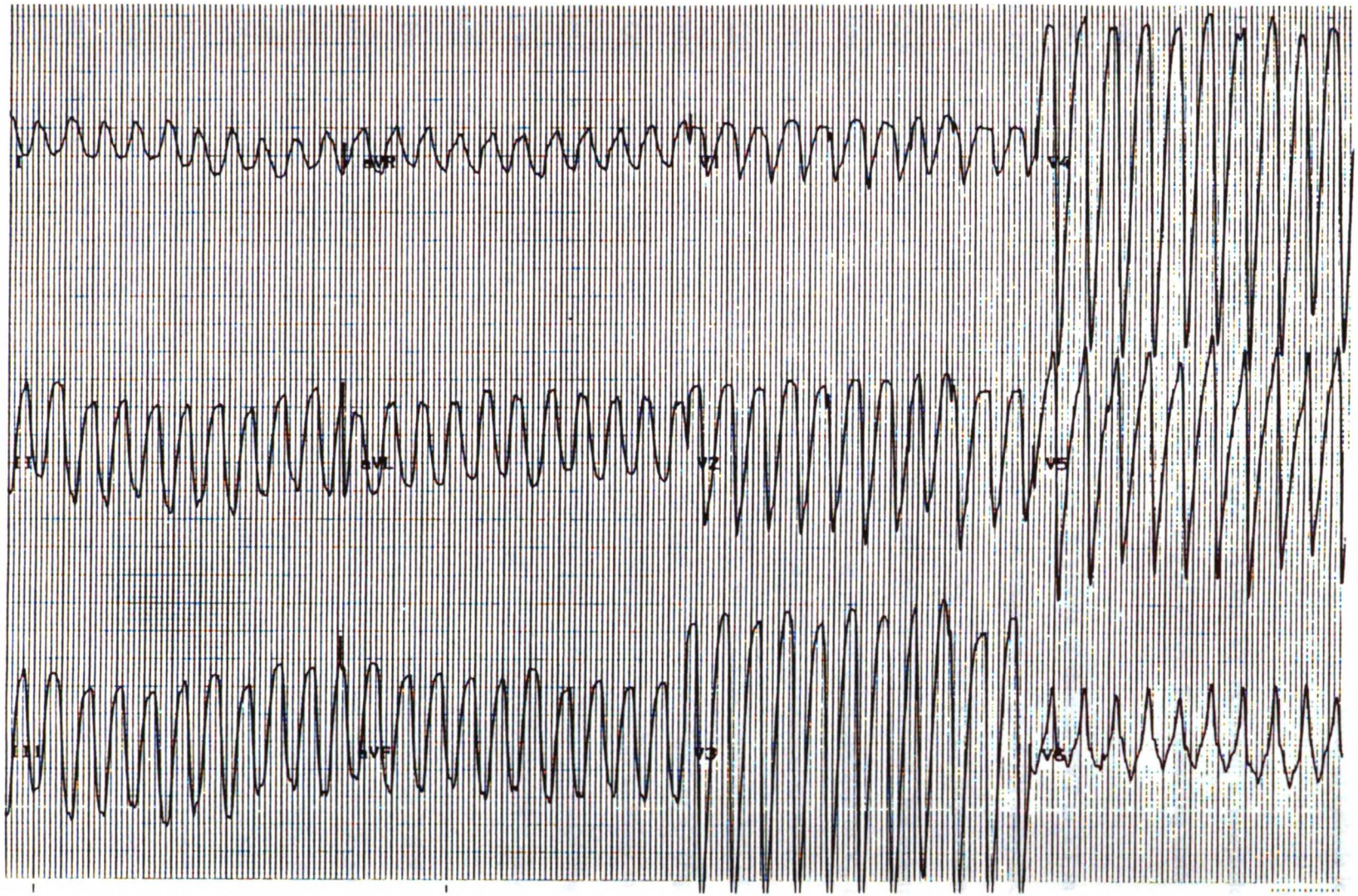


Figure 30. Bundle branch reentry VT without the QRS morphological criteria suggestive of VT (i.e., no R wave, S notch, or QRS onset to S nadir > 60 ms in V_1 or V_2, and, no Q wave in V_6). Left axis deviation present during tachycardia was also present during sinus rhythm with a left intraventricular conduction delay. Thus, VT which uses the His-Purkinje system is indistinguishable from SVT with left bundle branch block-type aberration.

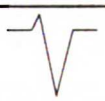
exhibited S notches in V_2 (MCL_2 was not recorded). A fifth VT exhibited a morphology suggestive of VT in MCL_6 (i.e., a biphasic rS with R:S ratio of < 1.0). The remaining sixth VT with negative Kindwall criteria was a false negative.

In summary, QRS morphology in MCL_1 alone was diagnostic of aberrant SVT or VT in 85 of the 120 tachycardias (71%), and V_1 morphology alone was diagnostic in 89 of 121 tachycardias (74%). Observation of morphology in additional leads (i.e., V_2, V_6, and MCL_6) improved only slightly the ability to diagnose tachycardias with a left bundle branch block contour. For example, only one additional VT was correctly diagnosed by observing V_2 and V_6 in addition to V_1, and only two additional VTs were correctly diagnosed by observing MCL_6 in addition to MCL_1.

QRS morphology in MCL_6 and V_6. In contrast to MCL_1 and V_1, only about one-half of the tachycardias exhibited QRS morphologies in MCL_6 or V_6 suggestive of aberrant SVT or VT (Figure 31). QRS morphology was frequently a monophasic R or biphasic Rs, patterns as likely to be aberrant SVT as VT. In addition, MCL_6 and V_6 were less likely to exhibit diagnostic morphologies during aberrant SVT than during VT. For example, only eight of the 35 SVTs had QRS patterns suggestive of SVT.

Never-the-less, there were three patterns indicative of VT: (a) a biphasic rS with a R:S ratio less than one, (b) a monophasic or notched QS, and, (c) a biphasic qR. The biphasic qR pattern rarely occurred, and thus, was not statistically significant; however, it was never seen during aberrant SVT. The one SVT which had a rS pattern occurred during atrial overdrive pacing with right bundle branch block aberration and an axis shift to +150 degrees. A QS pattern was particularly valuable in diagnosing VT since it occurred in about one third of VTs and it was never seen during aberrant SVT.

Figure 31
QRS Morphology in MCL_6 and V_6 During Wide QRS Complex Tachycardia

QRS Morphology	Lead	HBE Diagnosis		P
		ASVT N = 35	VT N = 86	
Biphasic rS with R:S ratio < 1.0 	MCL_6	1	14	< .05
	V_6	1	20	< .005
Monophasic or notched QS 	MCL_6	0	32	< .000
	V_6	0	27	< .000
Biphasic qR 	MCL_6	0	6	NS
	V_6	0	2	NS
Triphasic qRs with R:S ratio > 1.0 	MCL_6	8	1	< .001
	V_6	8	3	< .005
No diagnostic morphology	MCL_6	26	32	
	V_6	26	34	

HBE = His bundle electrogram
ASVT = aberrant supraventricular tachycardia
VT = ventricular tachycardia

One pattern, a triphasic qRs with a R:S ratio of greater than one, was suggestive of SVT with right bundle branch block aberration, however, it occurred in only eight of the 35 SVTs. Moreover, one VT in MCL_6 and three VTs in V_6 exhibited this supraventricular pattern (Figure 32).

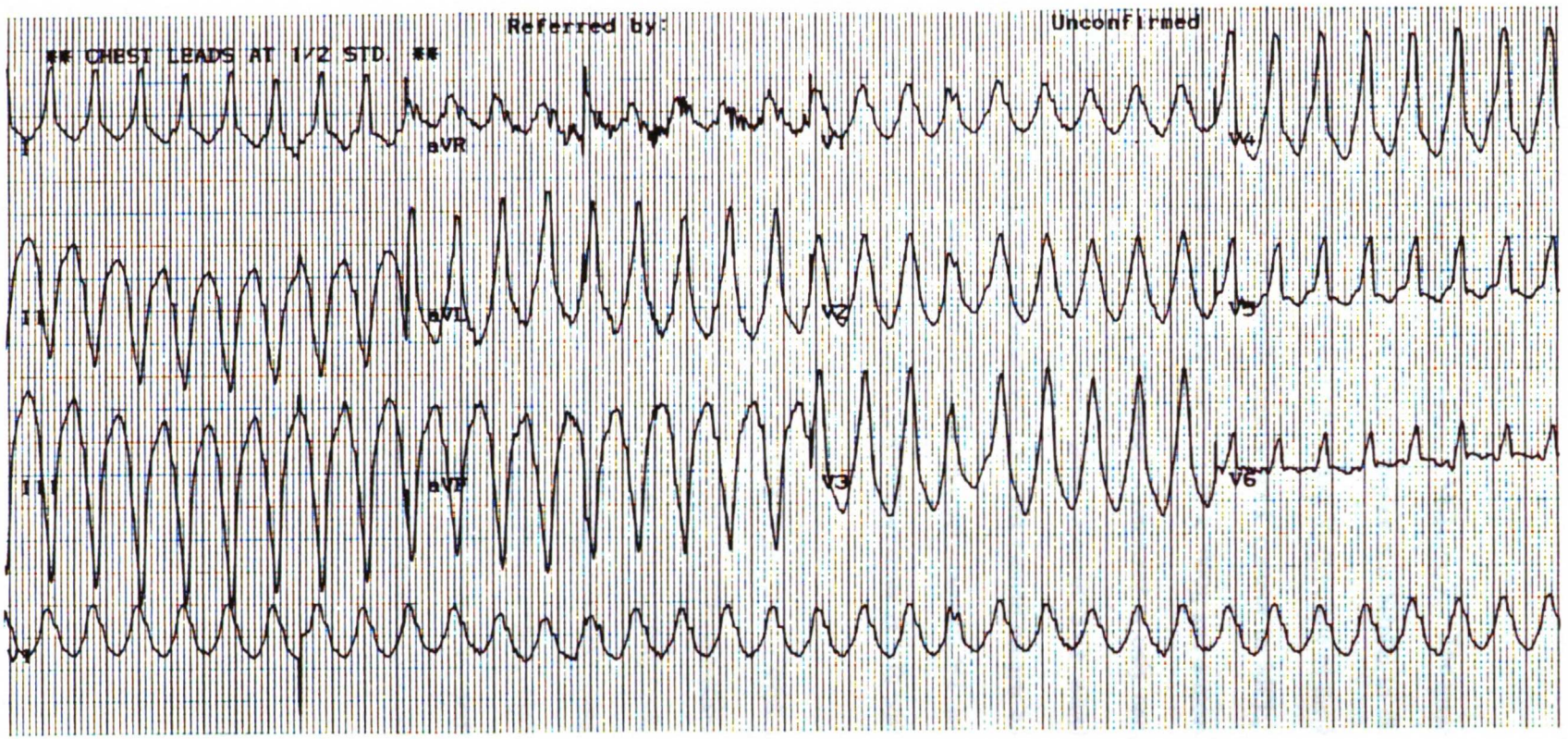
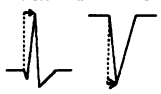


Figure 32. Ventricular tachycardia illustrating a qRs with a R:S ratio > 1.0, erroneously suggestive of SVT with right bundle branch block-type aberration. The presence of a taller left peak pattern in V_1 as well as left axis deviation during right bundle branch block contour tachycardia are, however, strongly suggestive of VT.

Measurement of QRS onset to predominant peak or nadir in MCL_6 or V_6. The measurement of QRS onset to the predominant peak (or nadir) of the wide complex was helpful in diagnosing only tachycardias with left bundle branch block contour in MCL_1 and V_1 (i.e., one of the Kindwall criteria). In tachycardias of either right or left bundle branch block contour in MCL_6 and V_6, however, a measurement of 50 ms or less predicted aberrant SVT ($P < .000$), and a measurement of 70 ms or more was nearly diagnostic of VT ($P < .000$) since it occurred in only one SVT in MCL_6 and in only two SVTs in V_6 (Figures 33 & 34). Both cases of SVT which had a measurement of 70 ms or more erroneously suggesting VT were left bundle branch block-type aberration with a prolonged R wave in MCL_6 and V_6. The several cases of VT which had measurements of 50 ms or less erroneously suggesting SVT generally occurred in VTs with narrow QRS complexes (Figure 35 A & B).

A measurement of 50 ms or less was highly specific for SVT (specificity = 93% and 92% in MCL_6 and V_6, respectively), as was a measurement of 70 ms or more for VT (specificity = 97% and 94% in MCL_6 and V_6, respectively). Moreover, the new criterion occurred frequently enough during wide complex tachycardia to be of value in making the diagnosis. For example, the sensitivity of the supraventricular criterion was 71 and 77 percent in MCL_6 and V_6, respectively; the sensitivity of the ventricular criterion was 71 and 72 percent in MCL_6 and V_6, respectively. A majority of tachycardias could be accurately diagnosed using this criterion alone (predictive accuracy in MCL_6 and V_6 = 71% and 74%, respectively).

Figure 33
Value of Measuring QRS Onset to Predominant Peak (or Nadir) in Distinguishing Aberrant Supraventricular from Ventricular Tachycardia

QRS Onset to Predominant Peak/Nadir (ms)	Lead	HBE Diagnosis		P	Sensitivity (%)	Specificity (%)
		ASVT N = 35	VT N = 86			
 ≤ 50	MCL_6	25	6	< .000	71	93
	V_6	27	7	< .000	77	92
 ≥ 70	MCL_6	1	60	< .000	71	97
	V_6	2	62	< .000	72	94

Lead	Predictive Accuracy (%)
MCL_6	71
V_6	74

ms= milliseconds
HBE= His bundle electrogram
ASVT= aberrant supraventricular tachycardia
VT = ventricular tachycardia

Precordial QRS concordance. As indicated in Table 11, concordance of the QRS complexes in MCL_1 and MCL_6 was suggestive of VT. Concordance was infrequently observed in V_1 and V_6, and in all six precordial leads of the conventional ECG; however, whenever it was present, the diagnosis was always VT. A concordant pattern was present in 23 of 85 VTs in MCL_1/MCL_6 leads (27%); in eight VTs in V_1/V_6 leads (9%); and in six VTs in the conventional ECG (7%). The low incidence of concordance in the 12-lead ECG may have been due to inferior displacement of leads V_3 and V_4 since a large defibrillation electrode placed over the left precordium precluded their accurate placement.

Comparison of QRS morphology during tachycardia with preexisting bundle branch block. Ten patients with VT had a preexisting bundle branch block during baseline sinus rhythm. None of these VTs had identical QRS morphology in all twelve leads as in baseline rhythm.

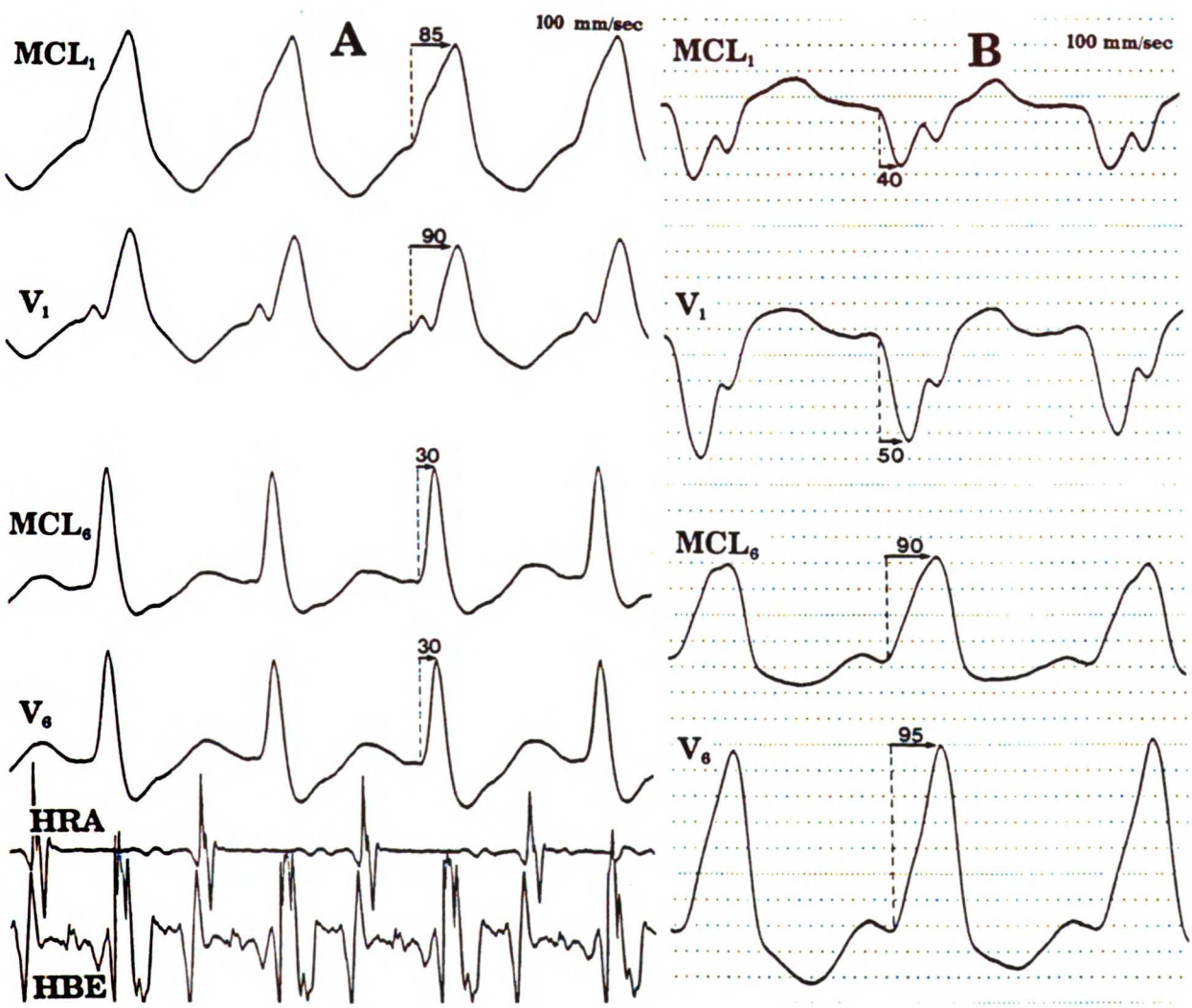


Figure 34. Measurement of QRS onset to predominant peak/nadir in distinguishing aberrant SVT from VT. A. Orthodromic tachycardia with right bundle branch block aberration has a long interval from QRS onset to R wave peak in MCL_1 and V_1, which does not discriminate between a supraventricular and ventricular origin. The measurement of 50 ms or less in MCL_6 and V_6 is, however, suggestive of SVT. B. Ventricular tachycardia has a short interval from QRS onset to S nadir in MCL_1 and V_1 which is erroneously suggestive of SVT with left bundle branch block aberration. The measurement in MCL_6 and V_6 of QRS onset to R wave peak of greater than 70 ms, however, strongly suggests VT. HRA = high right atrial electrogram; HBE = His bundle electrogram.

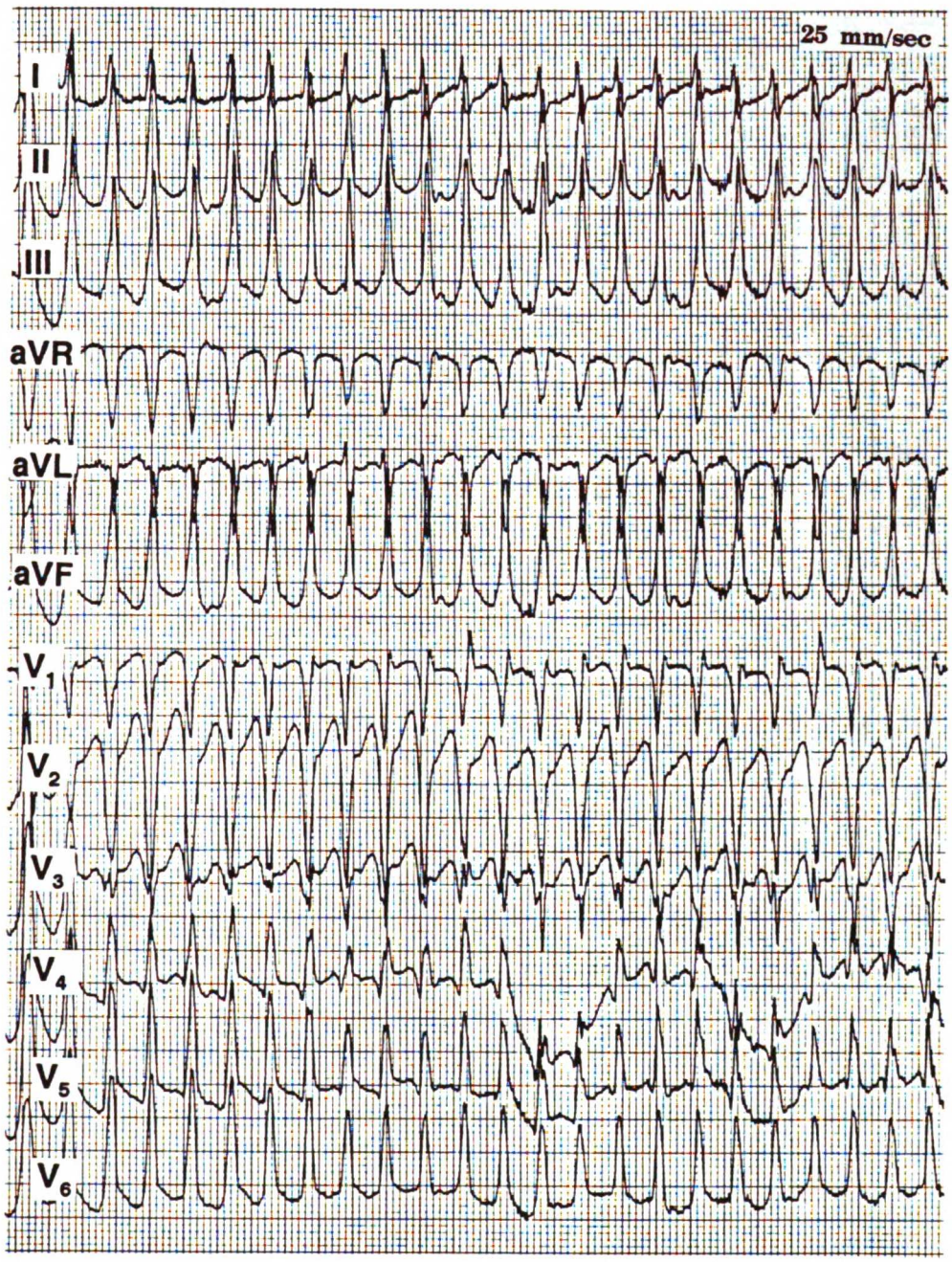


Figure 35a. Narrow QRS VT with QRS onset to R wave peak in V_6 less than 50 ms, erroneously suggestive of SVT. Interestingly, this VT occurred in a patient with a prolonged QRS (160 ms) at baseline (see Figure 35b, next page).

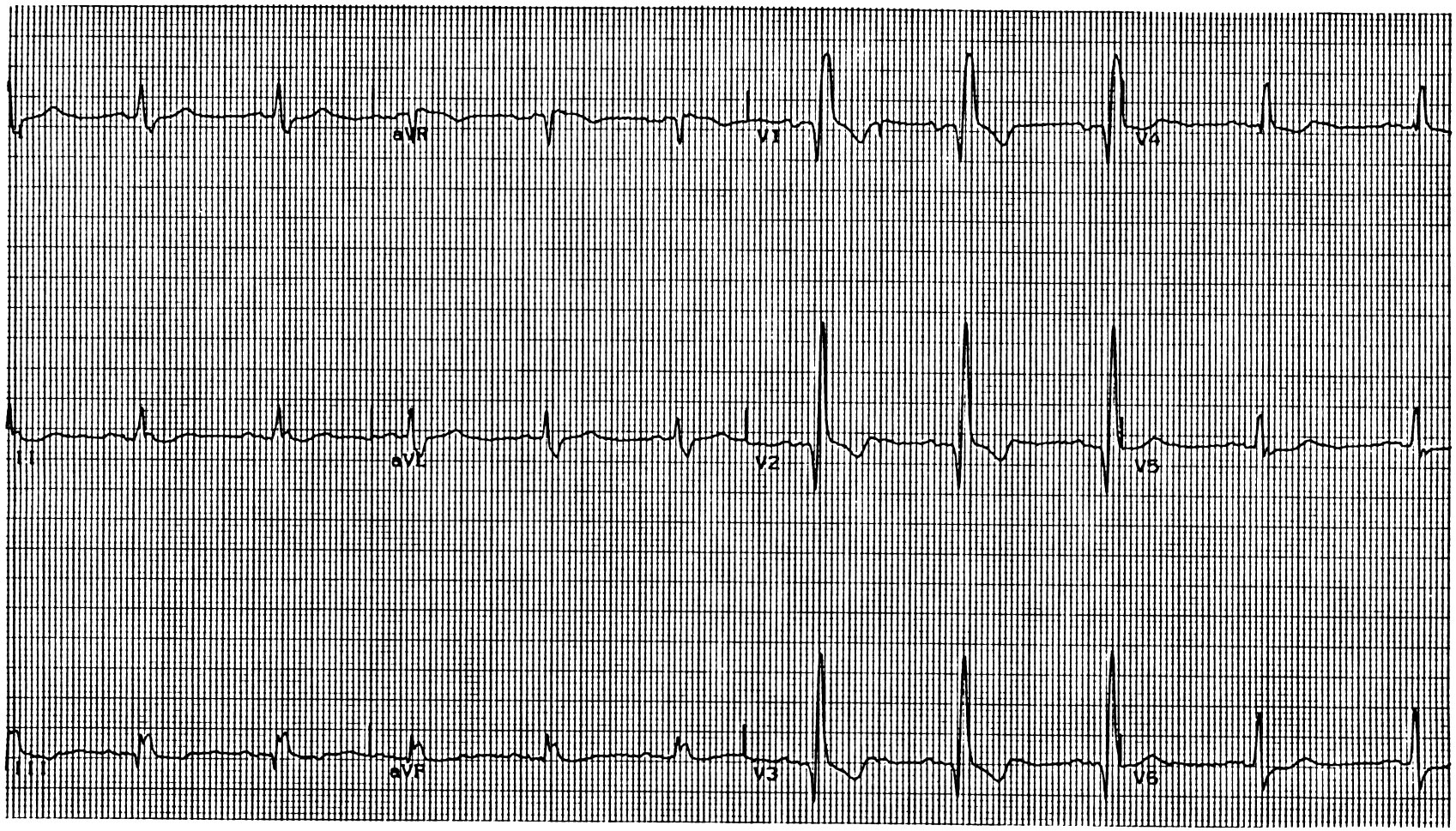


Figure 35b. Baseline ECG in a patient who developed narrow QRS VT (Figure 35a). The clearly different QRS morphology during VT compared to sinus rhythm with right bundle branch block rules out a diagnosis of SVT because SVT would also have right bundle branch block with identical QRS contours in all 12 leads.

Table 11
Value of Precordial QRS Concordance in Distinguishing Aberrant Supraventricular from Ventricular Tachycardia

QRS Concordance	Leads	HBE Diagnosis		P
		ASVT N = 35	VT N = 85	
present	12-lead ECG	0	6	NS
	MCL_1 & MCL_6	1	23	< .005
	V_1 & V_6	0	8	NS
absent	12-lead ECG	35	79	NS
	MCL_1 & MCL_6	34	62	NS
	V_1 & V_6	35	78	NS

HBE = His bundle electrogram
ASVT = aberrant supraventricular tachycardia
VT = ventricular tachycardia
12-ld ECG = V_1 through V_6

Several VTs had nearly identical morphology in one or two leads, however, the majority of leads were clearly different (Figure 36 A & B). Thus, a changed QRS morphology during tachycardia in one or more leads from baseline rhythm with bundle branch block was diagnostic of VT.

Value of the 12-Lead ECG and Selected Bedside Monitoring Leads

Criteria used to make the diagnosis of SVT with aberrant conduction were the presence of: (a) a normal axis in right bundle branch block-type tachycardias, (b) QRS patterns suggestive of SVT (i.e., rR′ or rsR′ in V_1/MCL_1, qRs in V_6/MCL_6, and, negative Kindwall criteria in V_1/MCL_1, V_2, and V_6/MCL_6), and, (c) QRS onset to predominant peak (or nadir) of 50 ms or less in MCL_6 or V_6. In addition, axis ranges suggestive of SVT, as indicated by QRS polarity in a single or dual bedside limb lead, were used when such leads were part of the particular lead or lead set being evaluated.

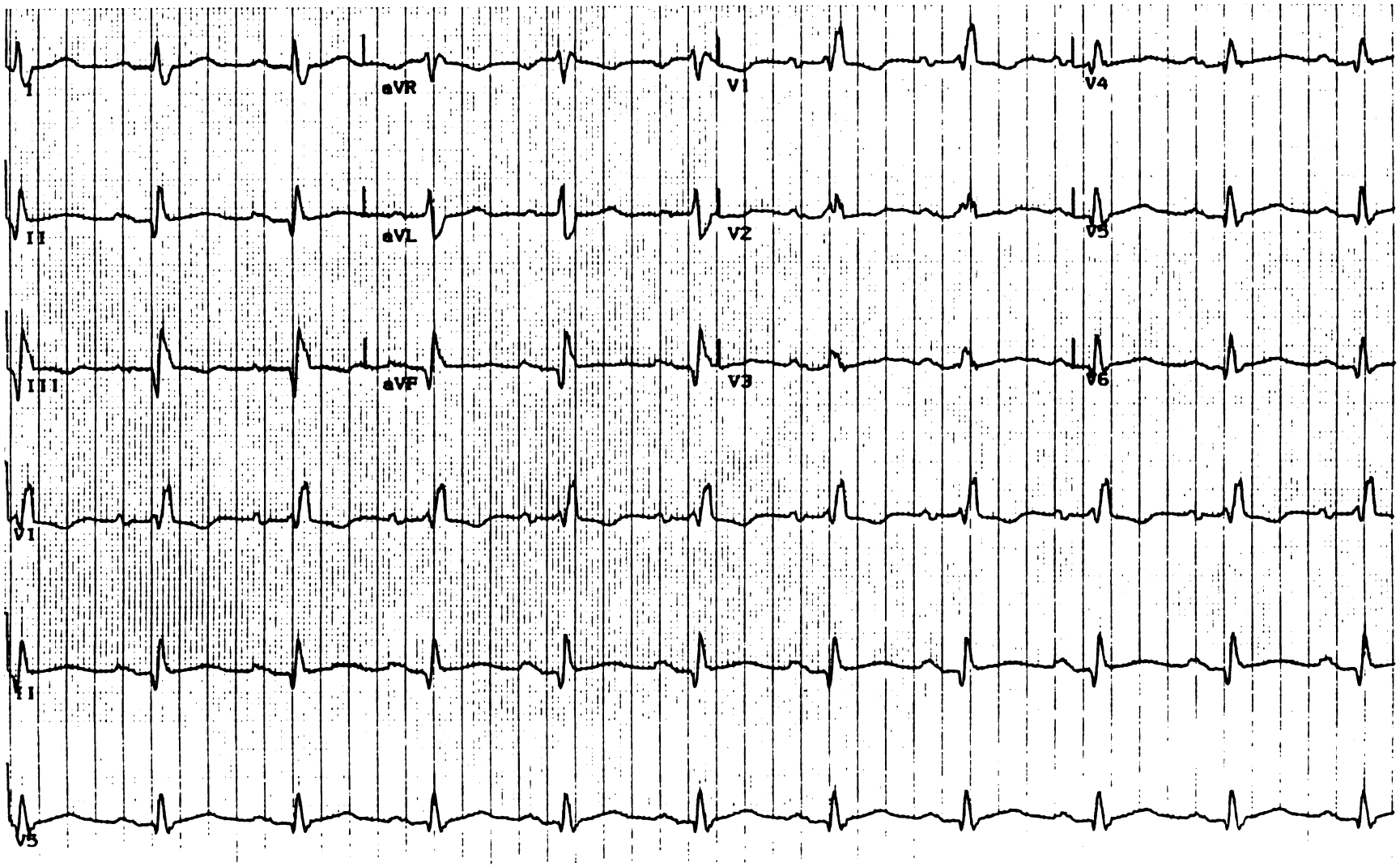


Figure 36a. Baseline sinus rhythm with right bundle branch block in a patient who developed VT (see Figure 36b, next page).

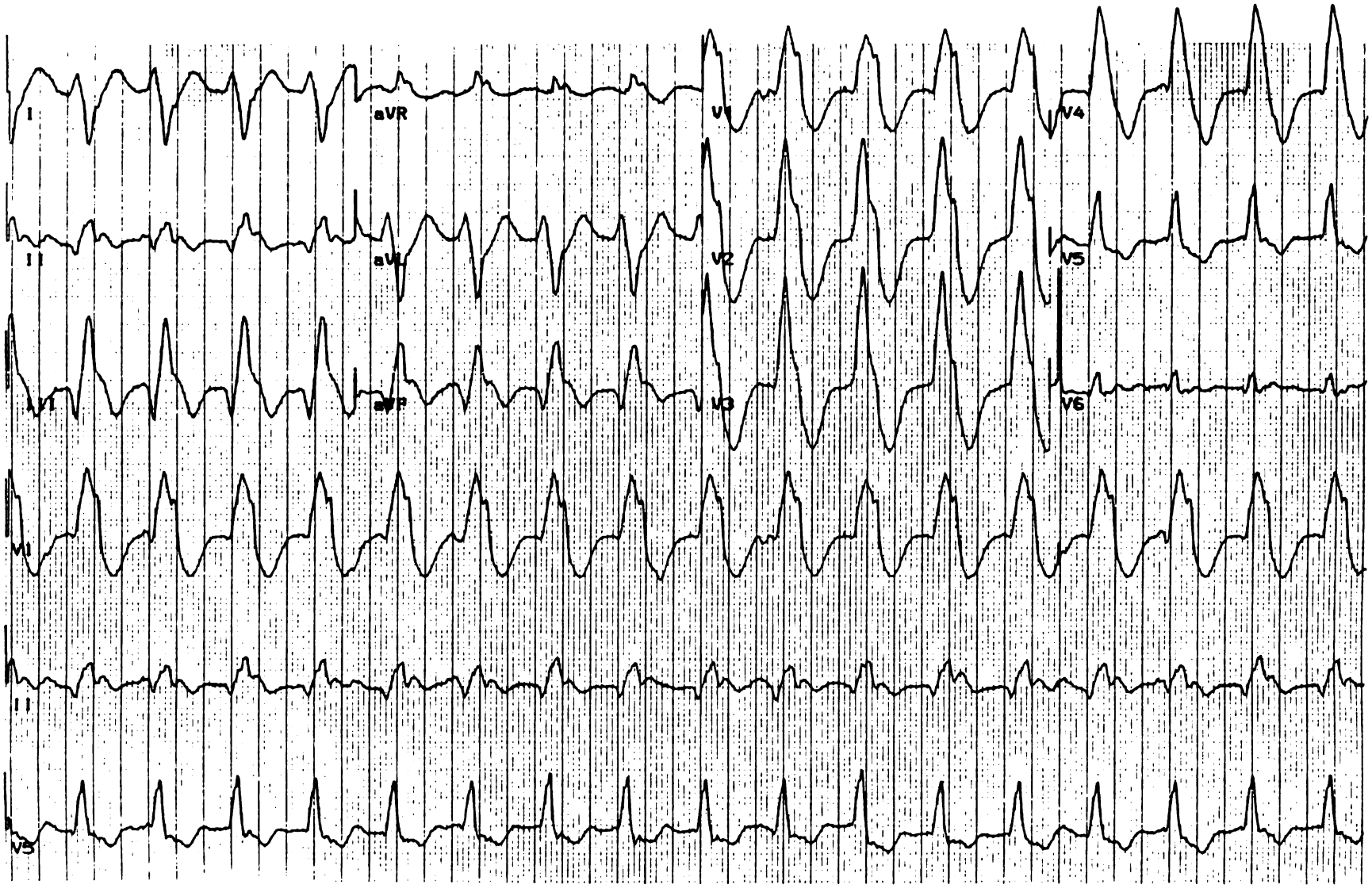


Figure 36b. During VT with a right bundle branch block contour, QRS morphology was clearly different in one or more leads (especially the right precordial leads). QRS morphology was identical during sinus rhythm and VT, however, in V_6.

Criteria used to make the diagnosis of VT were the presence of: (a) AV dissociation, (b) QRS width greater than 160 ms, (c) an axis in the northwest quadrant, (d) left axis deviation in right bundle branch block-type tachycardias, (c) a change in QRS morphology during tachycardia from baseline rhythm with preexisting bundle branch block, (d) precordial QRS concordance, (e) QRS patterns suggestive of VT (i.e., taller left peak, biphasic RS or QR, and Kindwall criteria in V_1/MCL_1, and rS (R:S < 1.0), qR or QS in V_6/MCL_6, and, (f) QRS onset to predominant peak/nadir of 70 ms or more in V_6/MCL_6. The presence of AV dissociation was considered diagnostic. In addition, axis ranges suggestive of VT, as indicated by QRS polarity in a single or dual bedside limb lead, were used when such leads were part of the particular lead or lead set being evaluated.

There were times when an ECG had criteria suggestive of both aberrant SVT and VT. For example, an ECG might have a rsR′ morphology in V_1 suggestive of aberrant SVT, while also having a bizarre axis and a QRS width of 200 ms, suggestive of VT. In such cases, a diagnosis was made based on which criteria (i.e., the SVT or VT criteria) were more numerous. Thus, if a tachycardia had three or four VT criteria, but only one SVT criteria, the diagnosis was VT. When equal numbers of SVT and VT criteria were observed in a particular tachycardia, a diagnosis of indeterminate was made. The diagnosis was based purely on electrocardiographic data, and the patient's medical history, age, or physical findings were not a part of the decision.

Value of the 12-lead ECG. Of the 121 tachycardias, 114 (94%) were correctly diagnosed from the conventional 12-lead ECG. The seven tachycardias which could not be diagnosed from the surface ECG were as follows. Two tachycardias were bundle branch reentry VT (one is pictured in Figure 30), and both were indistinguishable from SVT with left bundle branch block

aberration. Two additional tachycardias had conflicting criteria present. The first was SVT with left bundle branch block aberration. The ECG appropriately showed negative Kindwall criteria in V_1 and V_6 (V_2 was missing), but QRS onset to the predominant R wave peak in V_6 was 80 ms. The second conflict was a VT which appropriately showed a QRS onset to predominant R wave peak in V_6 of 80 ms, but a negative Kindwall pattern in V_1 and V_2 (i.e., no R wave, no S notch, and, less than 60 ms to S nadir). A fifth case in which a diagnosis could not be made from the surface 12-lead ECG was an atrial tachycardia with right bundle branch block aberration which had no criteria present (i.e., no AV dissociation, QRS width of 135 ms, axis = 100°, a monophasic R in V_1, a qRS in V_6 with R:S < 1.0, and QRS onset to S nadir between 50 and 70 ms). What was interesting about this SVT was that the monophasic R wave pattern in V_1 during rapid SVT became a rsR' pattern at slower rates which would have been helpful in making the diagnosis (this SVT is pictured in Figure 27). Two remaining tachycardias were aberrant SVTs which were misdiagnosed as VT from the surface ECG are pictured in Figures 16 and 19. One (Figure 16) had appropriate negative Kindwall criteria in V_1 but had a QRS width of 180 ms and a QRS onset to R wave peak in V_6 of 80 ms. The second (Figure 19) had an axis of - 100 degrees suggestive of VT.

Value of selected single bedside leads. Eighty-nine out of 120 tachycardias (74%) were correctly identified from a single MCL_1; 95 out of 121 tachycardias (79%) were correctly identified from a single V_1 (Table 12). This difference in diagnostic accuracy between MCL_1 and V_1 was not statistically significant. Moreover, in the 36 tachycardias with clearly different QRS morphology in MCL_1 and V_1, no statistically significant difference in diagnostic accuracy was

Table 12
Value of Selected Single Leads in Distinguishing Aberrant Supraventricular from Ventricular Tachycardia

Lead	Diagnosis from the Lead	HBE Diagnosis		Predictive Accuracy (%)
		ASVT N = 35	VT N = 86*	
MCL_1	ASVT	23	9	74
	VT	0	66	
	Indeterminate	12	10	
V_1	ASVT	29	7	79
	VT	0	66	
	Indeterminate	6	13	
MCL_6	ASVT	25	3	81
	VT	2	72	
	Indeterminate	8	10	
MCL_6 (without new criterion†)	ASVT	8	1	62
	VT	2	66	
	Indeterminate	25	18	
V_6	ASVT	27	4	83
	VT	2	74	
	Indeterminate	6	8	
V_6 (without new criterion†)	ASVT	8	1	60
	VT	1	65	
	Indeterminate	26	20	
II	ASVT	24	16	73
	VT	8	64	
	Indeterminate	3	6	
II (without axis criterion††)	ASVT	0	0	34
	VT	1	41	
	Indeterminate	34	45	

* One VT was not recorded in MCL_1 and MCL_6
† New criterion = QRS onset to to predominant peak (or nadir) of ≤ 50 ms suggests SVT; ≥ 70 ms suggests VT.
†† axis criteria = positive QRS in lead II suggests SVT; a negative QRS suggests VT.
HBE = His bundle electrogram
ASVT = aberrant supraventricular tachycardia
VT = ventricular tachycardia

present between the two leads. For example, predominately negative complexes in one lead might exhibit the Kindwall criteria while predominately positive complexes in the comparable lead might demonstrate a taller left peak pattern, both suggestive of VT (e.g., Figure 14). In addition, in the 40 tachycardias with similar QRS morphology in MCL_1 and V_1, no statistically significant difference in diagnostic accuracy resulted. For example, a rsR′ pattern might be observed in one lead while the comparable lead might demonstrate a rR′ pattern, both suggestive of aberrant SVT (Figure 37).

The proportion of correct diagnoses made from a single MCL_6 or V_6 was 81% and 83% respectively. This difference in diagnostic accuracy between MCL_6 and V_6 was not statistically significant. The new criterion (i.e., measurement of QRS onset to predominant peak/nadir) greatly improved the accuracy of diagnoses made from MCL_6 and V_6. For example, the proportion of correct diagnoses made from MCL_6 with and without the new criterion was 81% and 62% respectively ($P < .0000$); the proportion of correct diagnoses made from V_6 with and without the new criterion was 83% and 60% respectively ($P < .0000$). In fact, inclusion of the new criterion made a single left precordial lead (i.e., MCL_6 or V_6) as valuable as a single right precordial lead (i.e., MCL_1 or V_1) in distinguishing aberrant SVT from VT. (Prior to the inclusion of the new criterion, the high proportion of characteristic aberrant SVT and VT QRS morphologies in MCL_1 and V_1 compared to MCL_6 and V_6 made the right precordial leads superior to the left precordial leads).

The proportion of correct diagnoses made from a single lead II was 73%, which was not statistically different from a single MCL_1 or V_1, but was inferior to a single MCL_6 or V_6 ($P < .05$). In contrast to the precordial leads, however, lead II analysis resulted in a high number (24) of false

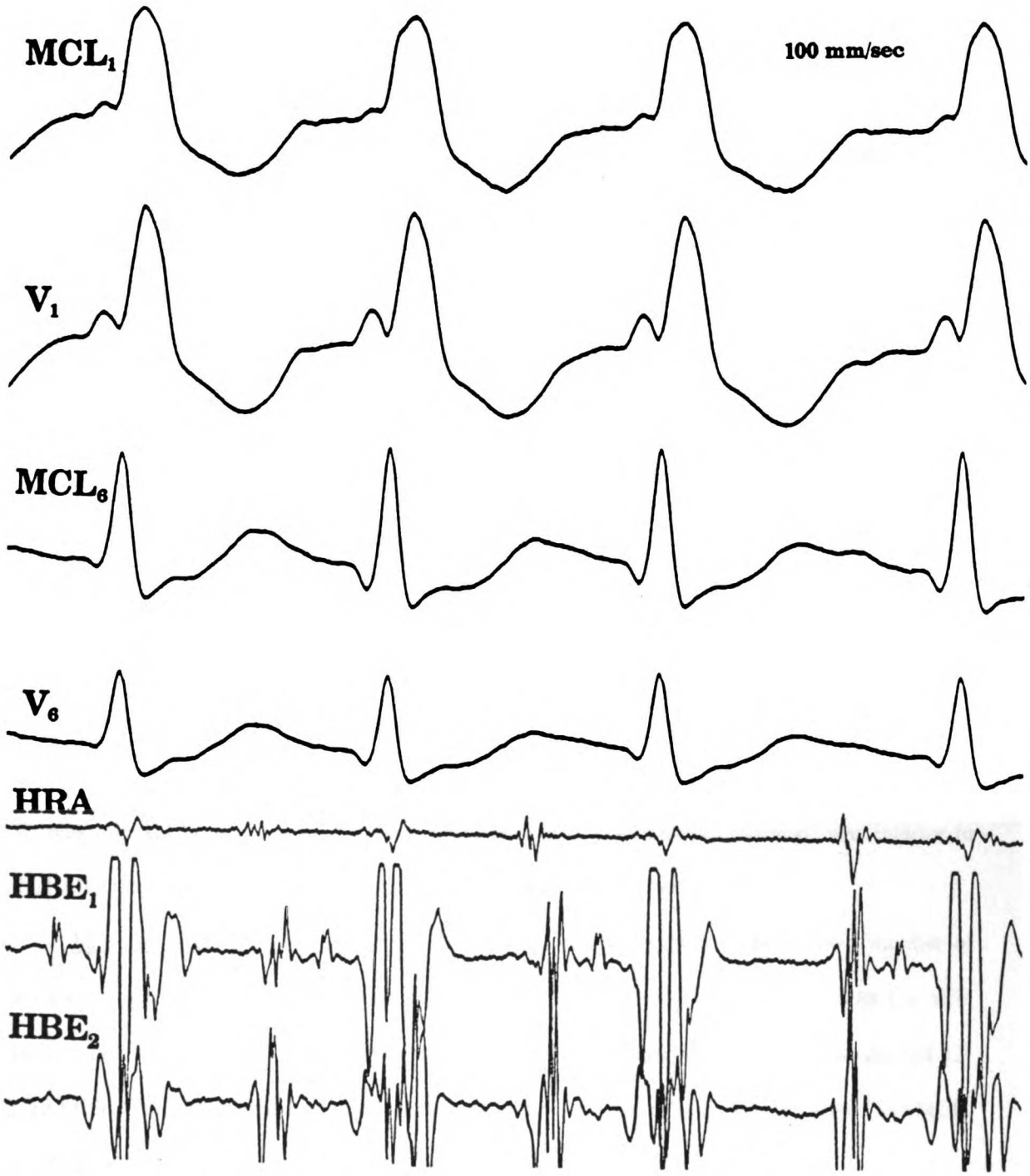


Figure 37. Atrial tachycardia with aberrant conduction. QRS morphology in MCL_1 and V_1 is not identical, however, both rR′ patterns are suggestive of SVT with right bundle branch block-type aberration. HRA = high right atrial electrogram; HBE_1 and HBE_2 = two His bundle electrogram recordings.

negatives and positives, which made it a less desirable lead. For example, 16 VTs were misdiagnosed as aberrant SVT from analysis of lead II. Analysis of the precordial leads resulted in few misdiagnoses (range = 5-9), and more situations in which there were no criteria present. The inclusion of the axis criteria (i.e., a positive QRS in lead II suggested aberrant SVT; a negative QRS suggested VT) greatly improved the diagnostic sensitivity of lead II. For example, without the axis criteria, predictive accuracy of diagnoses made from lead II fell from 73% to 34% ($P < .0000$). In fact, lead II was worthless in diagnosing aberrant SVT without the axis criteria, because no other supraventricular criteria could be observed in this lead.

Value of selected dual leads. A remarkable finding was that 108 of 120 tachycardias (90%) could be accurately diagnosed from a combined MCL_1 plus MCL_6 lead set (Table 13). In fact, the high diagnostic accuracy achieved from observing criteria in MCL_1 plus MCL_6 was not statistically different from making the diagnosis using the full 12-lead ECG. Dual MCL_1/MCL_6 leads were superior to a single lead II ($P < .000$), MCL_1 ($P < .000$), V_1 ($P < .005$), or MCL_6 ($P < .025$). Although seven additional tachycardias were diagnosed from analysis of dual MCL_1/MCL_6 leads than a single V_6, this difference did not quite reach statistical significance ($P = .07$). Combined V_1 plus lead II, the lead set used most often in units with dual-channel monitors, was inferior to dual MCL_1/MCL_6 leads in distinguishing aberrant SVT from VT ($P < .05$).

Value of selected multi-lead sets. The lead set which resulted in the greatest number of accurate diagnoses was a combined MCL_1 + MCL_6 + lead I + aVF, or V_1 + V_6 + lead I + aVF. In fact, this four-lead set was not statistically different than making the diagnosis from the full 12-lead ECG. Four additional lead sets which were comparable to 12-lead analysis were: (a) MCL_1 + MCL_6, (b) V_1 + V_6 + lead II, (c) MCL_1 + MCL_6 + aVR, and, (d) V_1 + V_6 + aVR.

Table 13
Value of Selected Lead Sets in Distinguishing Aberrant Supraventricular from Ventricular Tachycardia

Lead(s)	Diagnosis from Lead(s)	HBE Diagnosis		Predictive Accuracy (%)
		ASVT N = 35	VT N = 86	
A. Dual Lead Sets				
$MCL_1 + MCL_6$	ASVT	30	2	90
	VT	0	78	
	indeterminate	5	5	
$V_1 + V_6$	ASVT	31	2	88
	VT	0	75	
	indeterminate	4	9	
MCL_1 + II	ASVT	27	7	78
	VT	3	66	
	indeterminate	5	12	
V_1 + II	ASVT	27	4	82
	VT	4	72	
	indeterminate	4	10	
MCL_6 + II	ASVT	26	6	83
	VT	5	74	
	indeterminate	4	5	
V_6 + II	ASVT	26	4	83
	VT	4	75	
	indeterminate	5	7	
B. Triple Lead Sets				
MCL_1 + II + aVR	ASVT	27	12	80
	VT	4	69	
	indeterminate	4	4	
V_1 + II + aVR	ASVT	27	9	82
	VT	4	72	
	indeterminate	4	5	
MCL_6 + II + aVR	ASVT	26	9	80
	VT	6	72	
	indeterminate	3	6	
V_6 + II + aVR	ASVT	26	9	82
	VT	4	73	
	indeterminate	5	4	

Table 13 (cont.)

B. Triple Lead Sets (cont.)				
MCL_1 + I + aVF	ASVT	23	4	78
	VT	1	71	
	indeterminate	11	10	
V_1 + I + aVF	ASVT	29	4	88
	VT	2	77	
	indeterminate	4	5	
MCL_6 + I + aVF	ASVT	24	3	84
	VT	2	77	
	indeterminate	9	5	
V_6 + I + aVF	ASVT	26	3	87
	VT	3	79	
	indeterminate	6	4	
MCL_1 + MCL_6 + II	ASVT	30	4	88
	VT	2	76	
	indeterminate	3	5	
V_1 + V_6 + II	ASVT	31	3	93
	VT	3	81	
	indeterminate	1	2	
MCL_1 + MCL_6 + aVR	ASVT	32	4	90
	VT	1	76	
	indeterminate	2	5	
V_1 + V_6 + aVR	ASVT	32	3	92
	VT	2	79	
	indeterminate	1	4	
C. Quadruple Lead Sets				
MCL_1 + MCL_6 + I + aVF	ASVT	32	2	93
	VT	0	80	
	indeterminate	3	3	
V_1 + V_6 + I + aVF	ASVT	31	2	93
	VT	1	81	
	indeterminate	3	3	
D. Twelve Leads				
12-lead ECG	ASVT	31	2	94
	VT	3	83	
	indeterminate	1	1	

HBE = His bundle electrogram; ASVT = aberrant supraventricular tachycardia; VT = ventricular tachycardia

CHAPTER FIVE: DISCUSSION

This is the first study to validate use of the modified precordial leads, MCL_1 and MCL_6, in clinical practice. The vast majority of both normal and wide QRS complexes have identical (or nearly identical) patterns in MCL_6 and V_6. Although QRS morphology clearly differs between MCL_1 and V_1 in about one third of wide QRS complex tachycardias, no statistically significant difference in diagnostic accuracy results. In fact, the previously-proposed QRS patterns for distinguishing aberrant SVT from VT in V_1 and V_6 also are valuable in making the diagnosis from MCL_1 and MCL_6. Although an identical QRS pattern may not be recorded from the modified leads as in their conventional lead counterparts during a wide QRS complex tachycardia, both types of leads exhibit morphological criteria suggesting the same diagnosis.

In contrast to what is observed in V_1, right ventricular rhythms do not always have a predominately negative, left bundle branch block contour in MCL_1. Results from the present study indicate that a monophasic R wave or taller "left rabbit ear" pattern in MCL_1 is characteristic of rhythms originating from the right ventricular outflow tract area. Marriott and Fogg (1970) proclaimed that a major diagnostic advantage of monitoring patients with MCL_1 was the ability to recognize the ventricle being paced with temporary transvenous pacemakers. Figure 38 shows a MCL_1 tracing published by Marriott of a "partially ineffective pacemaker pacing the *left* ventricle" (1972, p. 545). The author explains that if the pacing pattern changes from right ventricular (i.e., left bundle branch block pattern) to left ventricular (i.e., right bundle branch block pattern) in MCL_1, one may be alerted to otherwise silent perforation of the right ventricular wall or septum by the catheter tip. Such a perforation in a patient receiving thrombolytic or anticoagulation therapy necessitates immediate removal of the pacemaker catheter to avoid cardiac tamponade. The

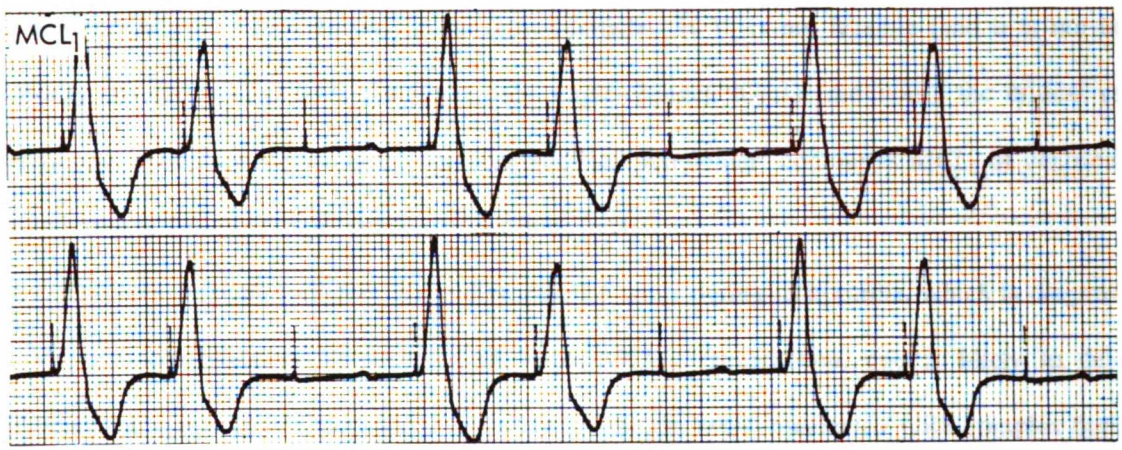


Figure 38. MCL_1 tracing illustrating intermittant failure of a pacemaker to capture the ventricle. The author states that QRS morphology in MCL_1 confirms pacing of the left ventricle, possibly through a perforation of the intraventricular septum or right ventricular wall. Reproduced from: Marriott, H. J. L. (1972) *Workshop in Electrocardiography*, Tampa Tracings: Oldsmar: FL, Review Tracing # 3, p. 507.

current study indicates that a more likely explanation for the change in pacing pattern from negative to positive complexes in MCL_1 is migration of the pacing catheter from the right ventricular apex to the right ventricular outflow tract, which is of much less consequence than perforation of the septum or right ventricular wall.

Coronary care unit nurses are routinely taught to monitor patients in MCL_1 during procedures involving the introduction of catheters into the right ventricle (e.g., insertion of transvenous pacemaker or pulmonary artery catheters). Such procedures are well-known to produce salvos of right ventricular tachycardia from mechanical stimulation of the myocardium. Ventricular tachycardia caused by mechanical stimulation does not carry the same prognostic significance and is not treated aggressively as VT arising spontaneously from an electrically unstable (often left

ventricular) focus. Results from the present study indicate that a true V_1 would be a better choice to monitor patients with catheters in the right ventricle. If V_1 is not feasible (and it is not in a majority of critical care units that have only single-channel, bipolar monitors), the critical care practitioner should be alerted to the characteristic "left ventricular" contour in MCL_1 of ventricular ectopy arising from the right ventricular outflow tract.

The present study indicates that a dissociated P wave during tachycardia, which is diagnostic of VT, is most likely to be observed in V_1 or MCL_1. In addition, one is likely to detect dissociated P waves from a lead which has a low amplitude, nearly isoelectric QRS complex during VT, because the small P wave is not lost in the barrage of wide, bizarre QRS complexes.

Data from the current study suggest serious limitations in use of the previously-proposed QRS width criterion for distinguishing aberrant SVT from VT. For example, 40 percent of aberrant SVTs in the present study had a QRS width greater than 140 ms. This finding occurred in patients not expected to have delayed conduction (e.g., patients without preexisting bundle branch block, drug therapy, or intramyocardial disease). A width of greater than 160 ms was a better discriminator of aberrant SVT and VT than a width greater than 140 ms; however, QRS width may be a less reliable criteria for two reasons. First, it is difficult to measure the QRS width accurately at recording speeds used in clinical practice (i.e., 25 mm/sec), particularly since it is often difficult to determine precisely the initiation and termination of the QRS complex. For example, it is rare to see a sharp-angle change in waveform marking the end of the wide, distorted QRS complex and the beginning of the S-T segment. Second, QRS width should be measured in more than one lead, with the widest measurement considered most accurate. If the measurement is made from a single

lead which is perpendicular in axis to the tachycardia in question, much of the QRS will be isoelectric and an underestimation of the true width will be made.

Results from the present study confirm prior research (Kindwall et al., 1988; Wellens et al., 1982) that axis deviation is a more useful criterion to diagnose VTs with a right rather than a left bundle branch block contour. In contrast to previous researchers who have reported that an extremely abnormal axis in the northwest quadrant is never observed during aberrant SVT (Akhtar et al., 1988; Caceres et al., 1987), two such bizarre axes were observed in the present series during aberrant SVT. Despite these two rare exceptions, however, the current study indicates that an axis in the northwest quadrant strongly suggests VT in both right and left bundle branch block-type VTs. Two additional axis criteria are useful: a normal axis during right bundle branch block-type tachycardias suggests aberrant SVT, and, a left axis deviation during right bundle branch block-type tachycardias suggests VT.

The current study indicates that the ability to detect normal and abnormal axis *ranges* by observing QRS polarity from a single lead II or aVR; or from dual II and aVR leads, or dual I and aVF leads, is useful in distinguishing aberrant SVT from VT using a bedside monitor. Given that a single lead II is as useful in this regard as a single aVR, lead II is probably the best choice since aVR has other limitations such as lower signal-to-noise ratio. If simultaneous I and aVF are used for monitoring, a quick glance at QRS polarity in these two leads can tell one whether the tachycardia axis is in the normal, right, left, or northwest quadrant. Thus, three additional criteria are available to distinguish aberrant SVT from VT (i.e., normal and left axis deviation during right bundle branch block contour tachycardias, and, an axis in the northwest quadrant in either right or left bundle branch block contour tachycardias).

QRS morphologies helpful in distinguishing aberrant SVT from VT in right bundle branch block contour tachycardias in MCL_1 and V_1 are a taller left peak pattern and a biphasic RS or QR pattern, all of which strongly suggest VT. In fact, these morphologies were never observed during aberrant SVT in the current study. A bimodal rR′ or triphasic rsR′ pattern is suggestive of aberrant SVT; however, such patterns are occasionally observed during VT. For example, in the present study, eight percent of VTs had an rR′ or rsR′ pattern. In contrast to previous research (Wellens et al., 1978) which indicates that a monophasic R wave pattern is never seen during aberrant SVT, such a pattern was present in 20 percent of aberrant SVTs in MCL_1 and in 14 percent of aberrant SVTs in V_1. In fact, a monophasic R wave pattern was not statistically significant for predicting VT in the present study.

Two observations regarding QRS morphology in right bundle branch block contour tachycardias are worth noting for their clinical applicability. First, it is often difficult to distinguish between a qR pattern (suggestive of VT) and a rsR′ pattern (suggestive of aberrant SVT) in MCL_1 or V_1. This difficulty arises because the onset of the wide complexes are superimposed on the previous S-T segment and T wave during rapid tachycardia. Thus, it is unclear whether initial positivity of the complex is due to the prior T wave or to the QRS onset. Second, rR′ and rsR′ patterns may be rate-related. That is, at faster tachycardia rates, a triphasic contour suggestive of aberrant SVT may assume a monophasic R wave contour. This observation underscores the importance of recording the first complex of a tachycardia where one is most likely to observe the true details of the wide QRS, free of impingement from a prior distorted S-T segment and T wave.

The QRS morphology described by Kindwall and associates (1988) is valuable for diagnosing VT with a left bundle branch block contour in MCL_1 and V_1. Complete application of Kindwall's

criteria, however, requires careful scrutiny of the wide QRS complex in more than one precordial lead (i.e., V_1 and V_2), which is not feasible with current bedside monitors. In addition, the Kindwall criteria intervals of greater than 30 ms for the initial R wave and 60 ms or more from QRS onset to S nadir are difficult to measure at paper speeds used in clinical practice. Intervals of 40 and 80 ms (one and two small boxes on the ECG paper) might be more practical; however, the criteria would be less sensitive in detecting VT.

The previously-proposed morphological criteria for distinguishing aberrant SVT from VT are much less sensitive in MCL_6 and V_6 than the morphological criteria in MCL_1 and V_1, particularly criteria for diagnosing aberrant SVT. For example, in the present study, three-quarters of aberrant SVTs had no diagnostic QRS morphology present in MCL_6 or V_6. Never-the-less, the patterns which are useful in these left precordial leads are a biphasic rS with a R:S ratio of less than one, a monophasic or notched QS, and a biphasic qR pattern, all of which indicate VT, and a qRs with a R:S ratio of greater than one which indicates aberrant SVT. The most helpful ventricular morphology in the current study was a QS pattern which was present in one of three VTs and was never observed during aberrant SVT. The only aberrant SVT pattern in MCL_6 and V_6 (qRs with R:S ratio > 1.0), seldom occurs (e.g., it occurred in only 23% of aberrant SVTs in the present study), and it is, on rare occasions, observed during VT. Moreover, this qRs pattern is the contour representative of only right bundle branch block-type aberration. No helpful patterns emerged in the current study to identify left bundle branch block-type aberration in MCL_6 or V_6. Left bundle branch block aberration often assumed a monophasic R or Rs pattern in MCL_6 and V_6; patterns as likely to be VT as aberrant SVT.

The measurement of QRS onset to the predominant peak (or nadir) of the complex is a valuable new clue for distinguishing aberrant SVT from VT in MCL_6 and V_6. A measurement of 50 ms or less suggests aberrant SVT; a measurement of 70 ms or more is virtually diagnostic of VT. Moreover, the measurement is not difficult to make since the peak or nadir is a readily identifiable point. In theory, the criterion is logical for the following reasons. Initial activation of the ventricles from an aberrantly-conducted supraventricular impulse proceeds rapidly via the His-Purkinje conduction system to produce an initial sharp, rapid deflection. Subsequently, the impulse spreads through non-conduction system myocardium to activate the ventricle on the side of the temporary bundle branch block. Such conduction outside the conduction system produces a widened QRS complex. In contrast, activation of the ventricles from a ventricular focus spreads entirely through non-conduction system myocardium to produce a more slurred initial deflection. Additionally, a majority of aberrantly conducted tachycardias exhibit a right bundle branch block contour. Activation of the left ventricle occurs in a more or less normal fashion via the intact left bundle branch to produce a normal R wave in MCL_6 or V_6 reflective of left ventricular free wall activation. The current data indicates that the measurement from QRS onset to the peak of this R wave is likely to be 50 ms or less during aberrant SVT. A potential limitation of this new criterion is that aberrantly conducted SVT may be misdiagnosed as VT when, in the presence of diffuse myocardial disease, it exhibits a more slurred initial QRS deflection. Likewise, the rare VT which uses the conduction system (e.g., fascicular or bundle branch reentrant mechanisms) may be misdiagnosed as aberrant SVT because it exhibits an initial rapid QRS deflection, but this limitation extends to all proposed morphological criteria.

If observation of QRS morphology in MCL_6 or V_6 is limited to the previously-proposed criteria, only about one-half of the tachycardias exhibit patterns helpful in making the diagnosis. If, however, observation of QRS morphology in MCL_6 and V_6 includes the new criterion, the vast majority of tachycardias (88% in MCL_6 and 92% in V_6 in the current study) exhibit useful patterns. Thus, MCL_6 and V_6 become as valuable as MCL_1 and V_1 for continuous bedside monitoring. This information is important because MCL_1 or V_1 leads are not always a practical choice for monitoring patients in critical care units. For example, a post cardiac surgical patient's sternotomy incision may preclude placement of an electrode near the sternum. Moreover, a patient with chronic obstructive pulmonary disease and accompanying increased anterior-posterior chest dimension typically has a low signal-to-noise ratio in MCL_1 or V_1 which may obfuscate proper diagnosis.

Previous studies have shown that a concordant pattern across the entire precordium from V_1 to V_6 during wide QRS complex tachycardia is virtually diagnostic of VT (Vera et al., 1972; Wellens et al., 1978). Data from the present study indicates that the observation of QRS concordance in V_1 and V_6 alone (without observing the complexes in V_2 to V_5) is also a strong criterion for diagnosing VT.

Comparing QRS morphology during wide complex tachycardia with that of baseline rhythm in patients with a preexisting bundle branch block is valuable in distinguishing aberrant SVT from VT. If the tachycardia QRS complex is different in one or more leads, SVT is virtually ruled out since a SVT complex should have an identical QRS morphology to that of baseline rhythm with bundle branch block. The diagnosis is very likely to be VT in such instances. However, it must be remembered that a rare SVT with antegrade conduction over an accessory AV pathway could also produce a different QRS morphology during tachycardia than that of sinus rhythm with bundle

branch block. Another caution regarding use of this criterion in clinical practice requires emphasis. If one observes identical QRS morphology during tachycardia as in baseline rhythm with bundle branch block from a single lead, one must look at additional leads to be sure that QRS morphology is not different from baseline elsewhere. For example, in the present study, it was not unusual for VT to have identical QRS morphology as in baseline rhythm with bundle branch block in one or two leads. Moreover, lead placement between the conventional 12-lead baseline ECG and the bedside monitoring lead is often slightly different so that a change in QRS morphology may be just a product of a change in electrode placement rather than a true change in morphology. Thus, this criterion requires careful scrutiny of baseline and tachycardia 12-lead ECGs.

Several recommendations for manufacturers of bedside cardiac monitors are evident from the present study. First, it is difficult to teach novice nurses how to attach MCL_1 or MCL_6 with current single-channel bipolar systems when the lead wires are labelled right arm, left arm, and right leg. To obtain MCL_1, the left arm electrode must be placed in the fourth intercostal space and the right arm electrode at the left shoulder. The nurse has to remember that when the selector dial on the monitor is turned to lead I, the left arm electrode is the positive electrode, while when the dial is turned to lead II, the leg electrode becomes the positive electrode. Current labels on bipolar monitors are based on the assumption that the leads of choice are lead I, II, or III. In fact, the manufacturer handbooks recommend lead II for optimum monitoring, especially for use with arrhythmia computers. This recommendation is based upon the fact that in the majority of patients who have a normal axis, the QRS complex in lead II has the greatest amplitude, and thus, the greatest signal-to-noise ratio. Therefore, the recommendation comes from monitoring manufacturer engineers whose goal it is to provide a clean signal for the arrhythmia computer to analyze. While

it is true that lead II often has a good quality, high amplitude signal, lead II is unhelpful in diagnosing bundle branch block which is mandatory for diagnosing aberrant ventricular conduction. It would be better if bipolar monitors were labelled just positive, negative, and ground.

Second, the capability for simultaneous display of two precordial leads (i.e., V_1 and V_6) is imperative. Although the current trend in the monitoring industry is to provide "hard-wire" bedside and telemetry systems with the capability of displaying multiple channels, no system is currently available that will display two precordial leads simultaneously. For diagnosis of isolated premature beats and non-sustained VT (which are rarely captured on a conventional 12-lead ECG), dual V_1 plus V_6 leads are superior to a single precordial lead in making the diagnosis. Moreover, if, in the four-channel monitors currently available on the market, it were possible to select two precordial leads, the combination of V_1, V_6, I and AVF would increase diagnostic accuracy of bedside monitoring to that of recording a full 12-lead ECG. Although it is possible with current dual-channel monitors to display simultaneous modified precordial leads MCL_1 and MCL_6 (see Figure 2), such lead placement has disadvantages. For example, it requires placing electrodes designated for the extremities on the chest which causes confusion. In addition, it means that one cannot "scroll through" leads I, II, III, aVR, aVL, and aVF as indicated on the bedside monitor, because the limb leads are no longer in their proper position. Thus, the lead one sees identified on the monitor selector dial is not the tracing gets on the bedside oscilloscope.

A third suggestion for monitoring manufacturers' research and development is to investigate an alternative approach to the traditional monitoring leads by deriving the ECG from vectorcardiographic leads. One such system currently employed in ambulatory Holter monitoring allows for

the recording of all twelve ECG leads from just five electrodes placed in convenient locations on the chest (Dower, Yakush, Nazzal, Jutzy & Ruiz, 1988).

Fourth, it would be advantageous to have rhythm strip recorders with the capability of recording tracings at paper speeds other than the traditional 25 mm/sec. For example, the Kindwall criteria and the new criterion presented in the current study require accurate measurement of short intervals such as 30, 50, 60, and 70 ms. Rhythm strip recorders that have the capability of recording tracings at faster paper speeds (e.g., 100 mm/sec) would make it possible to measure such short intervals with better precision. Moreover, because most bedside monitor signals are digitized, it should be feasible for monitors to determine accurately various QRS intervals and widths by real time computer analysis. Incorporation of such measurements into the arrhythmia computer algorithm would increase the accuracy of computer diagnoses of arrhythmias.

Recommendations which can be made from the present study and incorporated into practice with currently available monitoring systems include the following. For single-channel bipolar hard-wire and telemetry monitoring, leads MCL_1 and MCL_6 have the highest diagnostic accuracy and lowest number of false positives and negatives in distinguishing aberrant SVT from VT. MCL_6 is as accurate as MCL_1 only if the new criterion reported here (i.e., measurement of QRS onset to predominant peak or nadir) is used to make the diagnosis. For dual-channel, combination bipolar/unipolar monitors, MCL_1/MCL_6 leads are superior to other lead sets. If the ability to scroll through the limb leads is important, a second choice would be V_1 plus lead II; however, there will be some loss in diagnostic accuracy in diagnosing wide QRS complex tachycardias compared to a MCL_1 plus MCL_6 lead set. With the current inability to display two precordial leads simultaneously, there is no advantage in monitoring three or four channels over and above that of

monitoring two channels. However, for triple-channel monitors, a combination of V_1, I, and aVF, or V_6, I, and aVF are superior to other three-lead options. Such a triple-lead combination provides information not only on QRS morphology but also on QRS axis. Another option is to monitor dual MCL_1 plus MCL_6 leads and to turn off the third channel since it would not truly display the lead that was indicated on the selector dial. Moreover, triple V_1 plus I plus aVF leads are not superior to dual MCL_1 plus MCL_6 leads in distinguishing aberrant SVT from VT. As already mentioned, without the ability to display V_1 plus V_6 leads, there is no value in monitoring more than two channels. However, because lead II provides a clean signal as well as information about whether atrial depolarization occurs in a retrograde manner (i.e., P waves are inverted during retrograde ventriculo-atrial conduction), a combined V_1 plus I plus II plus aVF or a combined V_6 plus I plus II plus aVF appears to be a reasonable choice.

Conclusion

In Kansas City in the spring of 1962, Dr. Hughes Day opened the first coronary care unit, ushering in an era of special care units. Continuous ECG monitoring of patients has become routine in numerous units besides coronary care, including medical and surgical intensive care units, telemetry units, surgical suites, and emergency rooms. The goals of ECG monitoring have grown from simple heart rate monitoring to sophisticated arrhythmia detection, diagnosis, and treatment. The clinical environment, including the increase in patients' severity and acuteness of illness, and the shortage of experienced critical care nurses, calls for practice standards to be established for ECG monitoring. As the thirtieth birthday of coronary care approaches, there are still no specific guidelines as to which leads to monitor or how many leads to monitor. In addition, there is no universally accepted standardization of electrode placement for obtaining various leads. For

example, some critical care units contain diagrams showing "proper" placement of lead II's positive electrode on the left precordium, while others specify the lower left abdomen. Patients are monitored on a variety of leads within the same hospital and between hospitals. Although a plethora of ECG rhythm strips are included in the patient's permanent hospital record, there is little, if any, documentation as to what lead is recorded. For example, if a patient has a known recurrent VT documented by a conventional 12-lead ECG which has precipitated an aborted sudden death, it is often impossible to tell whether salvos of non-sustained VT recorded from the bedside monitor are the same troublesome arrhythmia because there is no documentation or standardization of lead placement.

A major reason for the paucity of specific guidelines to advise industry and to standardize practice has been the lack of research on which to base these standards. To my knowledge, this is the first study to address these issues, and, as such, it provides insight into three areas. First, it provides a critical re-examination of previously-proposed electrocardiographic criteria to distinguish aberrant SVT from VT with confirmation of some criteria<< and invalidation of others. Second, it introduces a useful new criterion with which to make the distinction in MCL_6 or V_6. Third, it assesses the diagnostic accuracy of various single leads and multiple lead sets in making the diagnosis from bedside monitoring leads with recommendations for industry regarding future research and development as well as recommendations for current clinical practice. Incorporation of the findings by both industry and critical care practitioners will result in an improvement in the manner in which patients are monitored in hospital settings, with the potential for decreasing the misdiagnosis and inappropriate treatment of patients with wide QRS complex tachycardia.

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APPENDIX A

COMMITTEE ON HUMAN RESEARCH
OFFICE OF RESEARCH AFFAIRS, Box 0616
UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

TO: Melvin M. Scheinman, M.D.
Box 0214

Barbara J. Drew
7067 Devon Way
Berkeley, CA 94705

RE: Comparison of Bedside and Conventional Electrocardiographic Leads

The UCSF Committee on Human Research (an Institutional Review Board holding Department of Health and Human Services assurance #M-1169) has approved the above request to involve humans as research subjects.

APPROVAL NUMBER: H1083-03429-02. This number is a UCSF CHR number which should be used on all consent forms, correspondence and patient charts.

APPROVAL DATE: May 4, 1989. Expedited Review

EXPIRATION DATE: May 4, 1990. If the project is to continue, it must be renewed *by the expiration date.* See reverse side for details.

ADVERSE REACTIONS/COMPLICATIONS: All problems having to do with subject safety must be reported to the CHR within ten working days.

MODIFICATIONS: All protocol changes involving subjects must have prior CHR approval.

LEGAL NOTICE: The University will defend and indemnify a principal investigator in legal actions arising from research activities involving humans only if the activities had current CHR approval.

QUESTIONS: Please contact the office of the Committee on Human Research at (415) 476-1814 or campus mail stop, Box 0616.

Good luck on your project.

Sincerely,

Reese T. Jones, M.D.
Chairman
Committee on Human Research

HEPC Project # 88003429

APPENDIX B

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

Department of Physiological Nursing

INFORMATION ABOUT PARTICIPATING AS A RESEARCH SUBJECT

PURPOSE: A study is being done by Barbara J. Drew, RN, to see how accurately patients' electrocardiograms are being monitored in intensive care units. Because I am having a test in which electrocardiograms are being recorded, I am being asked to participate in this study.

PROCEDURES: If I agree to be in this study, the following will occur: I will have two extra adhesive-type skin electrodes put on my chest.

RISKS: There are no risks to me if I decide to participate in the study. All information from this study will be handled confidentially. I will be identified with a code number rather than my name.

BENEFITS: There are no direct benefits to me for being in this study. The findings of this study may provide information that will help patients who are being monitored in intensive care units, but this cannot be guaranteed.

ALTERNATIVES: If I do not chose to participate in this study, I will have the electrophysiology test as planned.

PAYMENT: I will not receive any reimbursement for participation in this study. The cost of the electrocardiographic recordings will be paid by the researcher.

MY PARTICIPATION IN THIS STUDY IS VOLUNTARY. I HAVE THE RIGHT TO DECLINE TO PARTICIPATE AND TO WITHDRAW AT ANY TIME DURING THE STUDY WITHOUT ANY JEOPARDY TO MY TREATMENT AND CARE. If I have questions, I can call Barbara J. Drew at (415) 841-8007.

FOR REFERENCE

NOT TO BE TAKEN FROM THE ROOM

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