REVIEW

Current approaches and future developments in automatic tachycardia detection and diagnosis

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Implantable automatic cardioverter/defibrillators are now commonly used to treat malignant ventricular arrhythmias.¹ Early models simply detected a rapid heart rate and delivered a defibrillation shock, but more modern devices can deliver treatment as a tiered strategy of antitachycardia pacing, synchronised low energy cardioversion, and high energy defibrillation.^{2,3} Accurate detection and diagnosis of arrhythmias is essential to avoid inappropriate intervention: unnecessary shocks cause battery depletion, increase patient morbidity, and may cause degeneration from a stable to a poorly tolerated rhythm.^{4,5} An ideal system should treat haemodynamically unstable arrhythmias with a defibrillation shock, attempt pacing termination of stable ventricular tachycardia, and remain silent during stable atrial arrhythmias. Atrial fibrillation is common in patients with ventricular arrhythmias,⁶ and a rapid ventricular response may suggest a ventricular arrhythmia to an unsophisticated detection system. No currently available automatic detection system can reliably distinguish these situations.

Automatic tachycardia recognition by implanted antitachycardia devices depends on correct sensing and interpretation of physiological changes. Ideally, the sensed variable should alter rapidly with changes in rhythm and recover promptly after normal rhythm is restored. We describe the currently available methods for automatic tachycardia recognition and review future trends.

The table shows the variables currently proposed for automatic tachycardia detection. Many are under preliminary investigation only, and few will achieve practical usage in implantable devices.

Electrical sensing in the right ventricle

The simplest method of tachycardia detection is merely to sense an increase in heart rate manifested by a series of short RR intervals. This technique does not discriminate between sinus and atrial tachycardias and ventricular arrhythmias,^{7,8} and various refinements have been added to increase specificity. The onset of arrhythmia is usually more abrupt than that of sinus tachycardia and this may be used to aid detection of a pathological tachycardia.⁹ However, when the sinus response to sudden maximal effort is compared with the onset of

ventricular tachycardia, there may be overlap in individual subjects.¹⁰ Rate stability has been used as an additional marker of a sustained arrhythmia, but may also be unreliable.¹¹ Systems employing rate criteria show long-term reliability and require relatively little computing power but they are not diagnostic and offer no information about haemodynamic state.

ANALYSIS OF THE VENTRICULAR ELECTROGRAM
The ventricular endocardial electrogram recorded from the right ventricular apex represents the majority of ventricular activation.¹² Unipolar and bipolar sensing seem equally successful experimentally¹³ though with unipolar sensing there is a greater risk of inappropriate sensing of extraneous electrical noise. Simple comparison of electrogram amplitude allows discrimination between sinus rhythm and ventricular fibrillation¹⁴ but neither the peak amplitude nor the maximum slew rate reliably detect ventricular tachycardia.¹⁵

PROBABILITY DENSITY FUNCTION

Probability density function was used in the earliest implantable defibrillators¹⁶: the function calculates the time spent by the sensed electrogram close to the baseline. In organised rhythms, electrogram analysis results in a well defined peak representing the baseline. In ventricular fibrillation, however, there will appear a random scatter of points, with no peak around the baseline amplitude.¹⁶ If used in isolation, this sensing system is independent of rate, and will only diagnose arrhythmias similar in configuration to ventricular fibrillation.

Template matching

Template matching involves comparison of electrogram configuration during arrhythmias with a standard template derived during sinus rhythm.

Correlation waveform analysis calculates from beat to beat the mathematical relation between a normal template and the waveform under analysis.^{17,18} Such a system is independent of amplitude and baseline variation, but may be confounded by the development of paroxysmal bundle branch block.¹⁹ Specificity for uniform ventricular tachycardia can be increased by comparison of the suspect wave-

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*Variables used for automatic tachycardia recognition***Electrical sensing**

Rate criteria:

- Heart rate
- Sustained high rate
- Rate stability
- Rate of onset

Activation sequence

Dual chamber sensing

Electrogram configuration:

- Correlation waveform analysis
- Area of difference analysis
- Gradient pattern detection
- Frequency domain analysis

Haemodynamic sensing

Right ventricular pressure:

- Absolute
- Mean
- Pulse pressure
- dP/dt

Right atrial pressure:

- Mean
- Waveform

Right ventricular

volumetry

Mixed venous oxygen saturation

form and a template derived during previous episodes of induced arrhythmia.²⁰ Reliability is not affected by filtering at typical levels for an implantable device.²¹ The "bin area method" reduces the complexity of analysis because it compares corresponding areas or "bins" in the template and the waveform under analysis by a simple error measure, and requires a fraction of the calculations.²² Data compression before correlation analysis reduces computational demands while still reliably detecting ventricular tachycardia.²³

"Area of difference" analysis assesses the difference between templates of sinus rhythm and ventricular arrhythmias by totalling the absolute differences between individual sample points on the waveform under investigation and the normal template.^{15 18} The method is relatively efficient in terms of computing capacity required, but is affected by signal amplitude and baseline fluctuations. Critical statistical comparison suggests that algorithms using correlation waveform and area of difference analysis are similarly adept in distinguishing ventricular tachycardia from sinus rhythm or atrial fibrillation.²⁴

So far the computing demands of rapid analogue-digital conversion and filtering for these complicated algorithms have prevented practical application. There may be additional problems affecting a chronically implanted device; animal work has demonstrated decreases of up to 18% in electrogram voltages on exercise,²⁵ and use of chronic leads may be associated with a reduction in amplitude and slew rate of the recorded signal.^{26 27}

Gradient pattern detection involves calculation of a first time derivative of the endocardial electrogram, in which the amplitude is proportional to the rate of change of the original electrogram. Abnormal rhythms and left bundle branch block may be reliably distinguished from normal sinus rhythm and the results seem unaffected by changes in respiration and in posture of haemodynamically stable patients.²⁸ The system has also been shown to function accurately for the identification of a single arrhythmia configuration in real time,²⁹ and, when a microprocessor of similar power to implantable pacemaker processors is used, can distinguish multiple arrhythmias to which the computer had previously been exposed, provided the configurations are not too similar.³⁰ However, the numbers studied have been small and further research is needed to assess the longer term prospects for this method.

Frequency domain analysis

Comparison of frequency spectra of cardiac electrograms for the detection and diagnosis of arrhythmias has been relatively disappointing. Fast Fourier transformation is used to convert the raw signal to a plot of voltage versus frequency. This requires a large computing capacity. Appropriate filtering can highlight differences between normal and pathological electrograms.^{31 32} Significant differences between the centre frequency of spectra derived during sinus rhythm and ven-

tricular tachycardia have been demonstrated,³³ and fibrillatory and non-fibrillatory rhythms may be discriminated.³⁴ However, in a study of open chest dogs there was overlap between values for sinus rhythm and ventricular beats in almost half the animals studied.²¹

ATRIAL ELECTRICAL SENSING

Sensing the relation between atrial and ventricular activation may aid tachycardia diagnosis. However, sensing of ventricular far-field signals may obscure a low amplitude atrial signal, as in atrial fibrillation,³⁵ and rhythms involving a 1:1 atrioventricular relation (including atrioventricular reentry and atrioventricular nodal tachycardias and ventricular tachycardia with 1:1 retrograde conduction) remain difficult to discriminate. It is possible to distinguish between anterograde and retrograde atrial depolarisation^{36 37} and between atrial fibrillation and regular atrial rhythms both by atrial rate calculation and amplitude distribution and probability density function generation.^{38 39}

Simple ventricular rate criteria in combination with atrial sensing might identify ventricular tachycardia correctly in almost every patient.⁴⁰ An experimental algorithm, which may be suitable for use in implantable devices, incorporates dual chamber sensing and rate criteria and was capable of discriminating atrial intrinsic and reentry tachyarrhythmias and ventricular tachycardia with and without 1:1 retrograde conduction.⁴¹ An attempt to discriminate sinus tachycardia from paroxysmal tachycardias with a 1:1 atrioventricular relation by introducing a late diastolic atrial extrasystole is effective in the laboratory: in sinus rhythm the subsequent ventricular depolarisation will be correspondingly premature, but in paroxysmal tachyarrhythmias the ventricular regularity will not be disturbed.^{41 42} Clearly, the arrhythmia must be absolutely regular for this technique to work.

Activation sequence

The different sequence of myocardial depolarisation during sinus rhythm and ventricular tachycardia may be detected by multiple endocardial electrodes⁴³ and the addition of an atrial lead may allow differentiation of sinus and supraventricular tachycardias.⁴⁴ The number of electrodes required may be reduced by use of an electrode in the coronary sinus.⁴⁵ It is not possible to differentiate between rhythms with 1:1 atrioventricular relation on timing alone.

Haemodynamic sensing

While the electrical sensing algorithms described are effective in detecting a pathological arrhythmia they give no information on haemodynamic instability. In many arrhythmias rapidity of heart rate is not the sole determinant of cardiovascular instability and there is no correlation between electrogram configuration and hypotension.⁴⁶

Therefore, sensing of a haemodynamic variable, in combination with electrical events, is desirable. Because of the thromboembolic risk associated with chronic instrumentation of the left heart, studies have concentrated on the response to arrhythmias in the right heart.

PHYSIOLOGICAL RESPONSES TO TACHYCARDIA

In a thorough evaluation of the haemodynamic consequences of arrhythmias, Nakano showed that both atrial and ventricular tachycardias are associated with a decrease in mean arterial pressure, stroke volume, and cardiac output, and consequently an increase in atrial pressure bilaterally.⁴⁷ The interatrial pressure gradient increases in proportion to heart rate acceleration. Increased pulmonary artery pressure and total pulmonary resistance may largely be due to the increases in left atrial pressure. After onset of tachycardia there is partial haemodynamic recovery and increased myocardial contractility caused by catecholamine activity.⁴⁷ The magnitude of haemodynamic change is greater for ventricular arrhythmias because of abnormal patterns of ventricular contraction and periodic mitral regurgitation, caused by atrioventricular dissociation, that are associated with variable ventricular systolic and end diastolic pressures.⁴⁷ However, echocardiography during arrhythmias has failed to demonstrate an increased incidence or severity of mitral regurgitation during stable ventricular tachycardia in humans, and impaired cardiac performance was believed to reflect reduced ventricular diastolic filling.⁴⁸ Large atrial waves during arrhythmias may also represent cannon waves as the atria contract against closed atrioventricular valves.⁴⁹

In sinus tachycardia in response to exercise, mixed venous oxygen saturation falls promptly, in line with the increase in heart rate, with changes achieved in less than 10 seconds. Stroke volume also responds quickly to exercise but does not reflect the level of exertion. Maximal right ventricular pressure and dP/dt and mean right atrial pressure increase rapidly (over 10 seconds) with exercise, and the changes observed correlate well with workload.⁵⁰

RIGHT VENTRICULAR PRESSURE MEASUREMENT

Right ventricular pressure was sensed by the first laboratory model for the implantable defibrillator. Abolition of the phasic nature of this pressure triggered charging of the capacitor discharge circuit.⁵¹ During rapid right ventricular pacing to simulate ventricular tachycardia, mean right ventricular pressure showed a significant (mean increase 45%) and sustained increase in dogs.⁵² However, in humans rapid ventricular pacing at a comparable rate did not cause significant fluctuation in right ventricular pressure.⁵³ Conversely, a rapid fall in right ventricular systolic and pulse pressures at the onset of ventricular arrhythmias has also been demonstrated.⁵⁴ Changes in right ventricular pressure occurred within two seconds of tachycardia onset. By estimating the ratio of right ventric-

ular pulse pressure during tachycardia and at baseline, it was possible to discriminate between stable and unstable ventricular tachycardia, though in stable cases drift back to baseline over 30 seconds was noted. Evidence of some correlation between the fall in right ventricular pulse pressure and the associated fall in systemic arterial pressure has been noted, but with poor correlation with tachycardia cycle length.⁵⁵ Because there is large intra-group variability changes in right ventricular pulse pressure do not allow sinus tachycardia to be distinguished from haemodynamically stable or unstable ventricular or supraventricular tachycardia.⁵⁶ Changes in dP/dt distinguished between unstable ventricular tachycardia and sinus tachycardia, but again there was extensive overlap between arrhythmia groups.⁵⁶ An algorithm based on time intervals derived from dynamic right ventricular pressures may help to identify arrhythmias, but this was derived from a limited number of animal experiments in which pacing was used to simulate ventricular arrhythmias.⁵⁷

Right ventricular volumetry

Right ventricular volume may be estimated continuously from changes in intracardiac impedance.⁵⁸ This involves passing a constant subthreshold alternating current across the heart, between an intracardiac bipole or between intracardiac and remote surface electrodes, and measuring the varying voltage generated by this current as the intracardiac impedance changes. Errors may arise because of electrode motion and sensing of myopotentials. There is a practical method to reduce battery drain that uses a standard pacing lead, and the technique has been proposed for use in rate adaptive pacing.⁵⁹ Stroke volume may be more directly measured by a Doppler sensor, suitable for chronic implantation, situated in the superior vena cava and orientated towards the ascending aorta.⁶⁰ This approach is in the early stages of development but is promising.

During tachyarrhythmias cardiac output is reduced by reduced ventricular filling and systolic emptying. Right ventricular impedance during unstable ventricular tachycardia shows a significant reduction to less than 30% of that during sinus rhythm, though the changes during stable ventricular tachycardia are less pronounced.⁶¹ The variability in transcardiac impedance measured between epicardial defibrillator patches in dogs proved to be a reliable detector of ventricular fibrillation.⁶² Such a system removes the need for additional implanted sensors and little extra circuitry is required, but it cannot detect other arrhythmias and is unlikely to be of clinical value with increasing use of transvenous defibrillator lead systems.

Right atrial pressure monitoring

Right atrial pressure has been recommended as a physiological variable that varies consistently with changes in myocardial performance.⁶³ Though pressures are small (–1 to

+2 mm Hg at rest, rising to +5 mm Hg with exercise), significant changes have been shown during tachycardia. In anaesthetised dogs, ventricular pacing to simulate ventricular tachycardia resulted in a significant rise in mean right atrial pressure, which conversely remained stable during rapid atrial pacing imitating a supraventricular tachycardia.⁵² These changes have subsequently been confirmed in humans,⁵³ and mean right atrial pressure has been shown to reach a plateau approximately 30 seconds after the onset of ventricular tachycardia, returning to the baseline level within 30 seconds of the restoration of sinus rhythm.⁶⁴ This may be too slow for mean right atrial pressure estimation to be of practical use in an implantable antitachycardia device. In addition, baseline drift affecting a chronically implanted sensor becomes especially important in a low pressure chamber, and (though regular self-zeroing may partly overcome this problem) a system not reliant on absolute pressures would avoid it. The pressure waveform in the right atrium has been proposed as a variable aiding tachycardia recognition. High fidelity pressure recordings clearly demonstrate the relation between atrial and ventricular contraction and characteristic patterns have been demonstrated associated with specific atrial and ventricular arrhythmias.⁶⁵ Mean right atrial pressure showed an increase during all types of arrhythmia, regardless of haemodynamic stability, with a considerable degree of overlap between atrial and ventricular arrhythmias, though there was a preponderance of well tolerated arrhythmias in the study.⁶⁶ Development of a pattern-recognition algorithm and a reliable high fidelity chronic pressure sensing catheter may allow atrial pressure waveforms to become a realistic method for tachycardia diagnosis.

Mixed venous oxygen saturation

Mixed venous oxygen saturation may be measured chronically by reflectance oximetry sensors. During ventricular fibrillation in dogs, the pulsatile intra-beat variation in oxygen saturation decreased significantly and returned to baseline within 30 seconds of the restitution of sinus rhythm.⁶⁷ In humans reduction in central venous oxygen saturation was greater and more rapid during exercise than at the onset of haemodynamically tolerable ventricular tachycardia.⁶⁸ During unstable tachyarrhythmias increased use of peripheral oxygen results in greater desaturation, unless there is circulatory arrest and no admixture in the central veins.⁶⁹ Though these changes may be helpful in the automatic determination of haemodynamic state, the considerable lag before changes occur would delay intervention.

Conclusions

Over a short period the implantable defibrillator has developed from a simple device for tachycardia detection and delivering shocks into a system in which sophisticated tiered

anti-tachycardia pacing and cardioversion is now possible. To avoid spurious shocks and to optimise device performance a detection system giving increased diagnostic accuracy is desirable. This may necessitate a multisensor system, using both electrical and haemodynamic data. Dual chamber defibrillators will soon be available and may help overcome some problems in tachycardia diagnosis, particularly the difficulties in the detection of atrial fibrillation. The addition of a haemodynamic sensor might allow more appropriate application of tiered treatment. With further advances in lead and microprocessor technology a detection system capable of diagnosing all arrhythmias and determining their haemodynamic state should become a practical reality.

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