

# “Torsade de pointes” tachycardia *Re-entry or focal activity?*

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**SUMMARY** Paroxysms of ventricular tachycardia in which the amplitude and the direction of QRS complexes change periodically are defined as “torsade de pointes” tachycardias. The mechanism of this atypical ventricular arrhythmia has not yet been elucidated. The aim of our study was to induce “torsade de pointes” tachycardia experimentally, in order to gain insight into its possible mechanism. The experiments were carried out with isolated porcine hearts, perfused by the Langendorff technique. Epicardial electrocardiograms were recorded by unipolar leads. The specific pattern of “torsade de pointes” tachycardia could be induced by stimulation of the right and left ventricles in phase. From our experimental observations we conclude that a possible cause of “torsade de pointes” tachycardia is the interaction of two ectopic ventricular foci.

Rapid ventricular tachycardias, with periodic change of amplitude and direction of the QRS complexes, are defined as “torsade de pointes” tachycardias.<sup>1</sup> A detailed description of this serious arrhythmia has been given by Dessertenne,<sup>2</sup> who also created the term “torsade de pointes” (twisted tips). The precise mechanism of this atypical ventricular arrhythmia has not been completely clarified. Prolongation of the QT interval and inhomogeneous repolarisation of neighbouring areas of the myocardium are believed to be of major importance for its genesis.<sup>1,3</sup> “Torsade de pointes” has been recognised during hypokalaemia,<sup>4-6</sup> hypomagnesaemia,<sup>7,8</sup> acute ischaemia,<sup>9</sup> myocardial infarction,<sup>10</sup> myocarditis,<sup>1</sup> during the use of various antiarrhythmic agents, for example quinidine,<sup>11</sup> lignocaine,<sup>1</sup> mexiletine,<sup>12</sup> disopyramide,<sup>13,14</sup> and could be initiated by electrical ventricular stimulation.<sup>15</sup> Thus, aetiology of this rhythm disorder is diverse; its characteristic electrocardiographic appearance, however, suggests a specific mechanism.

The aim of our study was to induce “torsade de pointes” tachycardias experimentally in order to gain insight into its possible mechanism.

## Methods

The experiments were carried out in isolated pig hearts (n=7). The animals (weight 20±1 kg) were

anaesthetised by intravenous injection of sodium pentobarbitone (15 mg/kg). After intubation and artificial respiration the thorax was opened by a midsternal incision and the heart was rapidly removed and fixed to a heart lung machine where it was perfused with the blood of the same pig using the Langendorff technique.<sup>16</sup> The temperature was 37°C, and the coronary flow was set at 200 ml/min. The hearts were paced electrically from the epicardium with two independent stimulation generators at one and a half times the diastolic threshold. Unipolar electrocardiograms were recorded by means of non polarisable cotton wick electrodes from the epicardium and were recorded on an Elema ink writer (Fig. 1).

## Results

If paced from the right ventricle the electrocardiogram recorded within the area of the left anterior descending artery showed a positive deflection (R wave); if paced from the left ventricle the main axis of the QRS complex at the same recording point became negative (S wave) (Fig. 2). If the stimulation was performed from both the right and the left ventricles the resulting QRS deflection could be made zero if there was an appropriate interval between both stimuli. With the right ventricle stimulated at a constant rate (245/min) and the interval of the second stimulus (left ventricle) changed stepwise, the amplitude of the QRS could be adjusted between the maximum negative S wave and the maximum positive R wave. By

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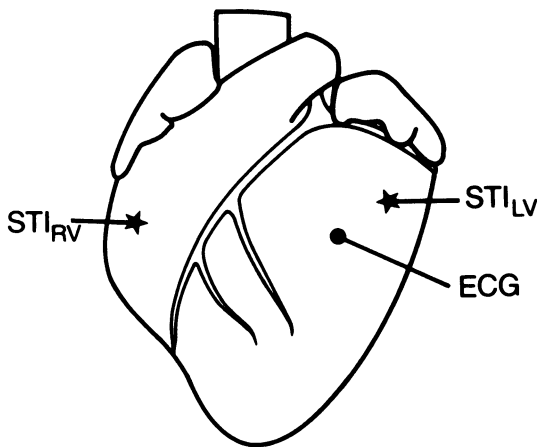


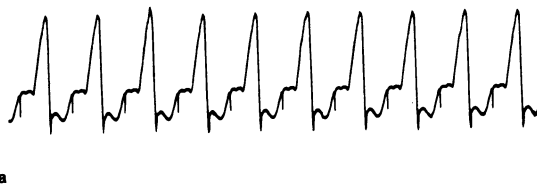
Fig. 1

Fig. 1 Isolated pig heart perfused by the Langendorff method. Arrangement of the stimulation and recording electrodes.  $STI_{RV}$ , site of stimulation on the right ventricle;  $STI_{LV}$ , site of stimulation on the left ventricle; ECG, recording point on the left ventricle.

Fig. 2 Electrocardiograms during stimulation of the right ventricle or the left ventricle. (a) If stimulation is performed to the right ventricle the electrocardiogram shows an R wave; (b) if stimulation is performed to the left ventricle the electrocardiogram shows an S wave.

Fig. 3 Electrocardiogram during stimulation of the right and the left ventricle. If the left ventricle is paced at a constant rate (cycle length CL: 245 ms) and the right ventricle is paced at a similar but periodically changing rate (CL: 230 to 260 ms) the electrocardiographic pattern of "torsade de pointes" occurs.

Fig. 4 After inducing ventricular tachycardia by occlusion of the left anterior descending coronary artery the electrocardiographic pattern of "torsade de pointes" could be generated by pacing the right ventricle at a similar but periodically changing rate. STI, start of stimulation of the right ventricle.



a



b

Fig. 2

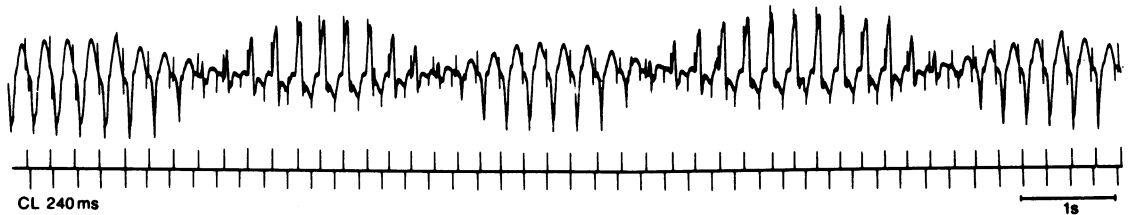


Fig. 3



Fig. 4

continuous prolongation of the coupling interval of the second stimulus from the basic stimulus a gradual decrease of the amplitude of the R wave occurred until it disappeared and then the QRS deflection became negative, increasing until it reached a maximum negative S wave (Fig. 3). If the interval was then shortened the amplitude of the S wave decreased, became zero, and changed again into a positive R wave.

In another series of experiments (n=4) the left anterior descending artery was occluded to produce a stable ventricular tachycardia and then only one pacemaker, stimulating the heart from the right ventricle, was necessary to generate the electrocardiographic pattern of "torsade de pointes". The pacing rate was set at the heart rate and by slowly shortening and lengthening the stimulation interval the electrocardiographic pattern shown in Fig. 4 was recorded.

### Discussion

Our results suggest that "torsade de pointes" tachycardias are based on focal impulse formation and on the interference of two separated foci with two different rates. If these foci are located in the right and in the left ventricle, the electrocardiogram shows either left bundle-branch block or right bundle-branch block. If both foci are active the resulting electrocardiogram represents the sum of both these electrocardiograms. If the impulse spreads from the left ventricle over the heart and the second focus in the right ventricle accelerates increasingly, the electrocardiogram gradually changes from the right bundle-branch block pattern to the left bundle-branch block pattern: "torsade de pointes" occurs.

Others favour the idea of a re-entry mechanism causing "torsade de pointes" tachycardia<sup>17-19</sup> and another possibility is that the tachycardia—beat by beat—originates from a point which slowly moves back and forwards between two regions of the heart, for instance the left and the right ventricle, but no experimental evidence or models have been produced to support these theories.

Our data do not rule out other possibilities but in our opinion there are more arguments for a bifocal genesis than for a re-entrant mechanism. "Torsade de pointes" tachycardias often occur when the excitability of the heart is increased, for example in hypokalaemia, in acute ischaemia, and in myocarditis. Under these conditions abnormal impulse formation could well originate from Purkinje fibres which extend over the whole heart and could set the stage for multifocal activity. The ineffectiveness of lignocaine-like substances in the treatment of "torsade de pointes" fits well with the assumption of abnormal

impulse formation in Purkinje fibres, because this type of activity originates from fibres depolarised into a potential range where the fast channel is already inactivated.<sup>20</sup> Furthermore, the frequent observation of a period of bradycardia before the start of a "torsade de pointes" tachycardia argues in favour of focal activity. The usual occurrence of multifocal extrasystoles—often in the opposite direction to the QRS complexes—just before spontaneous "torsade de pointes" tachycardias start<sup>1 3 21</sup> also suggests a bifocal genesis for this arrhythmia. Lastly, as long as there is no contraindication, overdrive suppression by cardiac pacing appears to be the treatment of choice for "torsade de pointes" tachycardia.<sup>1 13 18</sup> and its effectiveness again supports our hypothesis of bifocal activity, which was originally advanced by Dessertenne in 1966<sup>2</sup> as the cause of this arrhythmia.

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