Association of ventricular extrasystoles and ventricular tachycardia with idioventricular rhythm

S. Talbot and M. Greaves

From Sheffield Royal Infirmary, Sheffield

Patients with acute myocardial infarction were monitored for ventricular arrhythmias in the first 48 hours.

Idioventricular rhythm (rate less than 100/minute) occurred in 35 out of 224 patients (15.6%) during the first day and in 13 out of 192 patients not receiving treatment on the second day (6.8%). This arrhythmia was frequently preceded by late ventricular extrasystoles, which often showed variation of their coupling intervals to the preceding QRS. Double ventricular extrasystoles separated by ≥ 600 ms were also precursors of idioventricular rhythm. Idioventricular rhythm at times could be described as an escape rhythm, but on other occasions it was undoubtedly an accelerated rhythm. Spontaneous changes in the idioventricular cycle length were frequent on single one-minute electrocardiographic recordings. The rate of the dominant rhythm in patients with episodes of idioventricular rhythm was significantly slower than the heart rate of patients without this arrhythmia. Idioventricular rhythm was more frequent in patients with inferior infarction.

Idioventricular rhythm sometimes preceded ventricular tachycardia but there was only a significant association between ventricular tachycardia and idioventricular rhythms with rates of over 75/minute. Irregular idioventricular rhythm frequently accelerated to ventricular tachycardia. It is suggested that the term benign idioventricular rhythm be reserved for those rhythms below 75/minute, and that the term rapid idioventricular rhythm should be used for rhythms between 75 and 120/minute. The rate of idioventricular rhythm is related to the probability of development of life-threatening ventricular arrhythmias during the first 48 hours after myocardial infarction.

Idioventricular rhythm and ventricular tachycardia are not clearly separated in many early analyses of the arrhythmias occurring after acute myocardial infarction (Julian, Valentine, and Miller, 1964; Fluck *et al.*, 1967; Lawrie *et al.*, 1968). However, subsequently it has been found that slow ventricular rhythms are more benign than faster rhythms (Rothfield *et al.*, 1968; Norris, Mercer, and Yeates, 1970). Many idioventricular rhythms are critically dependent on the underlying heart rate, appearing when this slows and disappearing when the sinus rhythm accelerates. The terms 'slow' and 'benign' idioventricular rhythm have become popular. Though the extremes of rate of idioventricular rhythm and ventricular tachycardia do justify such a distinction, it is not easy to provide a definition of these arrhythmias that not only separates their electrocardiographic features but also their clinical significance. Therefore, the dividing line between these arrhythmias has been variously drawn at

100 to 120 beats a minute (Rothfield *et al.*, 1968; Norris *et al.*, 1970; De Soyza *et al.*, 1974a).

In this study the importance of these 'slow' ventricular rhythms after acute myocardial infarction and their relations with ventricular tachycardia and measures of ventricular extrasystolic activity have been examined.

Patients and methods

Between January 1974 and November 1974 269 patients with acute myocardial infarction were admitted to the coronary care unit of the Sheffield Royal Infirmary: 31 patients died in cardiogenic shock during the first 48 hours after myocardial infarction, and 5 patients were paced within 6 hours of admission because of complete heart block. These and a further 9 patients who were on antiarrhythmic therapy on admission were excluded from the study. The remaining 224 patients formed the subject of this study.

The patients were monitored continuously by the nursing staff for at least 48 hours and electrocardio-

graphic recordings were taken of any episodes of idioventricular rhythm or ventricular tachycardia. The nursing staff recorded the number of ventricular extrasystoles at hourly intervals. If at these times the number of ventricular extrasystoles was ≥ 5 /minute a one-minute electrocardiographic recording was taken and this was repeated at 6-hourly intervals for 48 hours. Similar observations were made at other times if ventricular extrasystoles appeared or increased in number.

A clinical assessment of each patient was made on admission and at least daily thereafter throughout the patient's stay in hospital. Daily 12-lead electrocardiograms and at least one chest radiograph were performed in the first 48 hours.

Antiarrhythmic therapy was not used routinely but was given for ventricular tachycardia or after an episode of ventricular fibrillation. An episode of ventricular tachycardia was diagnosed when there were three or more ventricular extrasystoles in a row separated from each other on average by < 600 ms. The cycle length of each arrhythmia was measured and idioventricular rhythm was considered irregular if the range of variation of cycle length was greater than 10 per cent of the mean cycle length.

The diagnosis of ventricular extrasystoles and their differentiation from aberrant conduction were based on the usual electrocardiographic criteria (Lipman and Massie, 1965; Sandler and Marriott, 1965; Talbot, 1973). All the coupling intervals of single uniform ventricular extrasystoles on each record were examined. If there were multiform ventricular extrasystoles the coupling intervals of each form of extrasystole were measured separately. Uniform ventricular extrasystoles which had more than 100 ms variation in their coupling intervals were described as variable coupled extrasystoles. All recordings were examined for parasystole (Scherf and Schott, 1953). Ventricular extrasystoles that occurred on or after the P wave of the succeeding sinus beat have been called late ventricular extrasystoles. Two successive extrasystoles separated from each other by ≥ 600 ms and ventricular bigeminy lasting for at least a minute were recorded separately.

The diagnosis of myocardial infarction was made electrocardiographically and confirmed by serum enzyme tests (Blackburn *et al.*, 1960; Goldberg and Winfield, 1972). The location of infarction was described as anterior, inferior, septal, or lateral or any combination of these (Talbot *et al.*, 1973). The time of onset of infarction was determined from the clinical history and examination in conjunction with the results of three serum enzyme tests on consecutive days. If these were conflicting a further history was taken to elucidate if and when one or more infarctions had occurred.

Results

On the first day after infarction 35 out of 224 patients had one or more episodes of idioventricular rhythm. Thirty-two patients received treatment for ventricular tachycardia or ventricular fibrillation and,

therefore, on the second day only 192 patients could be studied further. Nine of these patients had one or more episodes of idioventricular rhythm in the second 24 hours after infarction. In all, 44 patients had idioventricular rhythm. Seventeen of these patients also had ventricular tachycardia. In 12 patients idioventricular rhythm clearly preceded ventricular tachycardia, whereas in 5 patients idioventricular rhythm was interspersed with episodes of ventricular tachycardia and the initial arrhythmia could not be determined. Forty-two patients developed ventricular tachycardia during the 48-hour period. There was a significant association between idioventricular rhythm and subsequent episodes of ventricular tachycardia (χ^2 17.1, $P < 0.01$). There was also an association between episodes of ventricular tachycardia and idioventricular rhythm in single one-minute electrocardiographic recordings.

It was discovered that the association between idioventricular rhythm and ventricular tachycardia was the result of an association of faster irregular idioventricular rhythms between 75 and 100 per minute and ventricular tachycardia (Table 1). In this table the average cycle length and the variability of idioventricular rhythm with or without ventricular tachycardia are shown. Though arbitrary distinctions between these arrhythmias have been used in this paper, idioventricular rhythms and ventricular tachycardias were difficult to distinguish, since either rhythm could often end with cycles that were longer or shorter than the predominant cycle of the rhythm. In any individual patient the contour of the two rhythms was often similar, and they were often associated with ventricular extrasystoles of similar shape.

The average heart rate of the patients with idioventricular rhythm was 69.9 per minute (SD 13.9) and this was significantly slower than the average heart rate of patients without this arrhythmia on the first day after infarction (78.4 SD 15.2— $t=3.3$, $P < 0.01$). Idioventricular rhythm was associated with inferior infarction. There were 88 patients with inferior infarction (37.7%), and of these, 21 had at least one episode of idioventricular rhythm. However, there were 136 patients with infarction in areas other than the inferior wall of the heart and of these only 14 had idioventricular rhythm ($\chi^2=6.4$, $P < 0.05$). It appeared that the association of idioventricular rhythm and inferior infarction was the result of a parallel association with bradycardia, because there was no association of any ventricular rhythms with the site of infarction if only patients with heart rates between 75 and 100 a minute were considered.

Ventricular extrasystoles which preceded idio-

TABLE 1 Association of idioventricular rhythm and ventricular tachycardia

<i>Idioventricular rhythm associated with ventricular tachycardia</i>		<i>Idioventricular rhythm unassociated with ventricular tachycardia</i>	
<i>Cycle length (ms)</i>	<i>Variation of cycle and form</i>	<i>Cycle length (ms)</i>	<i>Variation of cycle and form</i>
700	—	1000	—
900	Irregular	1000	—
740	Irregular	1080	—
680	—	1200	—
900	Irregular	880	—
640	Irregular	920	Irregular/multiform
720	Multiform	960	Multiform
800	Irregular/multiform	1040	—
800	Irregular/multiform	800	—
800	Irregular/multiform	920	Irregular/multiform
900	Irregular/multiform	1020	—
780	—	960	—
640	Irregular	1200	—
660	—	960	Irregular/multiform
760	Irregular/multiform	1000	—
680	Irregular/multiform	1100	—
880	Irregular	1020	—
		840	Irregular/multiform
		940	—
		820	Irregular/multiform
		880	—
		980	Irregular
		1040	—
		1100	—
		1120	—
		1080	—
		1040	—
Mean cycle 764* (SD 92)		Mean cycle 996* (SD 109)	

*Significant difference ($t=7.25$, $P<0.01$).

ventricular rhythm showed a number of characteristic features (Table 2). Late ventricular extrasystoles were the most frequent precursors of idioventricular rhythm (Fig. 1, 2, and 3). These extrasystoles could have fixed coupling intervals but often they had variable coupling intervals, particularly in association with faster and irregular idioventricular rhythms.

The variation of coupling intervals was often, but not always, restricted to the end of electrical diastole (Fig. 2 and 3). Idioventricular rhythm associated with ventricular tachycardia was more often followed or preceded by ventricular extrasystoles which varied throughout electrical diastole. However, if the rate of the dominant rhythm was rapid, electrical diastole was so reduced that even 'late' ventricular extrasystoles were also close to the T wave. In such cases, though variation of coupling intervals was often obvious, such extrasystoles have to be described on the criteria used earlier as having fixed coupling (Fig. 4). Extrasystoles which occurred after a long sinus pause (escape beats) were also associated with idioventricular rhythm and late

ventricular extrasystoles. The pauses could be the result of sinus arrhythmia, atrial extrasystoles, or sinus slowing after one ventricular extrasystole (Fig. 4).

Double ventricular extrasystoles, separated by more than 600 ms were associated with idioventricular rhythm (Fig. 2 and 3). Ventricular bigeminy occurred both before and after idioventricular rhythm. After lignocaine therapy for ventricular tachycardia, the rhythm was sometimes converted to idioventricular rhythm before it finally disappeared or changed to ventricular bigeminy.

Fusion beats were obvious in 15 patients with idioventricular rhythm (Fig. 2), but were also found alone or in association with variable coupling, double ventricular extrasystoles, and late ventricular extrasystoles. Parasystole was detected in two one-minute electrocardiographic recordings, but only one of the two patients had idioventricular rhythm. However, short episodes which could have been interpreted as parasystole or parasystolic ventricular tachycardia were often preceded by or followed unprotected idioventricular rhythm.

FIG. 1 Top: One ventricular extrasystole towards end of electrical diastole. One episode of irregular idioventricular rhythm (mean cycle 630 ms). Bottom: Sustained ventricular tachycardia (cycle 340 ms).

Discussion

This study has shown that idioventricular rhythm and ventricular tachycardia are associated. A clear distinction between these arrhythmias is impossible and many ventricular rhythms vary above and below 100/minute. Idioventricular rhythm sometimes follows paroxysms of ventricular tachycardia if these are not treated. By not treating idioventricular rhythm routinely, this arrhythmia was found to be a significant prelude to ventricular tachycardia. Idioventricular rhythm was associated with sinus rates below 75 a minute, and it appeared that this was the reason for an association with inferior infarction which is more often accompanied by sinus bradycardia than infarction in other areas of the heart. Idioventricular rhythms under such conditions were usually slower and not significantly associated with ventricular tachycardia. However, faster and irregular idioventricular rhythms of

between 75 and 100 a minute were associated with ventricular tachycardia.

Idioventricular rhythm has been described as a distinct arrhythmia after myocardial infarction (Rothfield *et al.*, 1968; Norris *et al.*, 1970). It was soon appreciated that it was common and occurred in up to 30 per cent of patients. It did not appear as sinister as ventricular tachycardia, and was called benign. This is a relative term and recently it has been suggested that idioventricular rhythm and late ventricular extrasystoles can be a prelude to ventricular tachycardia (De Soyza *et al.*, 1974a, b). This study supports this view.

It is generally accepted that idioventricular rhythm is caused by increased ventricular automaticity. A number of features support this.

- (1) It is associated with escape beats.
- (2) Often the first ventricular extrasystole follows the preceding sinus beat by an interval that is

similar to or longer than the cycle of the idioventricular rhythm.

- (3) It may be suppressed by acceleration of the dominant rhythm.
- (4) It may be associated with fusion beats.
- (5) It is usually unifocal and is sometimes associated with parasystole (Chung, Walsh, and Massie, 1965; Roelandt and Schamroth, 1971).

The original description of idioventricular rhythm only included accelerated ventricular rhythms that became manifest when the dominant rhythm slowed and could be abolished by acceleration of the sinus rate. Such an automatic focus cannot be protected and the rhythm is comparable to an escape rhythm in a patient with heart block.

However, the lack of protection may not be absolute even in a one-minute recording, so that parts of the rhythm may resemble parasystolic ventricular tachycardia (Chung *et al.*, 1965). If an exit block occurs intermittently, parasystole may be associated

with idioventricular rhythm. Unequivocal parasystole is uncommon after acute myocardial infarction and appears to be benign. Protection of an ectopic focus does not appear to be related to the probability of ventricular tachycardia (Baxter and McGuinness, 1974), and in this study there was no evidence that episodes of idioventricular rhythm were a result of a regular exit block of ventricular tachycardia.

Parasystole and extrasystoles have features in common; they may not be easily distinguished and could be caused by similar local electrophysiological events (Watanabe, Pamintuan, and Dreifus, 1973; Watanabe, 1971). However, they could still be the result of enhanced automaticity and re-entry, respectively. Likewise, the relation between idioventricular rhythm and ventricular tachycardia does not prove that these arrhythmias are the result of the same mechanism. Variable coupled extrasystoles may be non-parasystolic and are associated

FIG. 2 Top: Double ventricular extrasystoles (separated by 1080 ms). First extrasystole is late ventricular extrasystole. Middle: Two late ventricular extrasystoles associated with variable idioventricular rhythm. Bottom: Late ventricular extrasystole and possible fusion beat. One early ventricular extrasystole (variable coupling).

TABLE 2 Features of ventricular extrasystoles associated with idioventricular rhythm

	No. of patients with idioventricular rhythm	No. of patients without idioventricular rhythm	Total no. of patients
Late ventricular extrasystoles	25*	16*	41
Variable coupling of ventricular extrasystoles	23*	31*	54
(a) <80% of electrical diastole	14*	24*	38
(b) ≥80% of electrical diastole	9**	7**	16
Double ventricular extrasystoles (≥600 ms)	10**	5**	15
Ventricular bigeminy	12**	19**	31
Total no.	44	180	224

*Significant differences between patients with and without idioventricular rhythm (χ^2 test $P < 0.01$).

**Significant differences between patients with and without idioventricular rhythm (χ^2 test $P < 0.05$).

with ventricular tachycardia both in patients with chronic ischaemic heart disease and patients with myocardial infarction (Talbot, 1973; Talbot and Dreifus, 1975). The variable coupling associated with idioventricular rhythm is mainly related to late ventricular extrasystoles, but variable coupling related to ventricular tachycardia often extends closer

to the T wave. If ventricular tachycardia is caused by re-entry, re-entry pathways may vary and this may be the explanation for the variable coupling of associated ventricular extrasystoles. There is also evidence that ventricular tachycardias may not be a homogeneous group of arrhythmias. Immediately after infarction they may be brought about by

FIG. 3 One-minute electrocardiographic recording showing variable coupling, double ventricular extrasystoles, late ventricular extrasystoles, and irregular idioventricular rhythm. The second ventricular extrasystole is slightly deformed because of prematurity.

FIG. 4 Top: Ventricular extrasystoles with apparent variable coupling, but because of limits of measurement these were described as having fixed coupling. Electrical diastole is abbreviated because of short cycle length of dominant rhythm. Middle: Two episodes of variable idioventricular rhythm with retrograde atrial excitation. Subsequent sinus slowing facilitates the next ventricular beat. Bottom: Extrasystoles with limited variation of coupling intervals as above, but following an extrasystole of different form and shorter coupling, ventricular fibrillation occurs.

enhanced automaticity but later and in other conditions they may be the result of re-entry (Wellens, Lie, and Durrer, 1974).

The association of idioventricular rhythm and ventricular escape beats with late ventricular extrasystoles and double ventricular extrasystoles separated by ≥ 600 ms suggests that such extrasystoles are the result of increased automaticity. Since the coupling intervals of these extrasystoles often resemble that of the idioventricular rhythm, the ventricular focus is probably discharged by each sinus beat; however, perhaps because of varying automaticity or exit block, these extrasystoles are not always manifest.

Facilitation of idioventricular rhythms by slow sinus rates may explain why the slower idioventricular rhythms are of less significance. If automaticity is increased to such an extent that the rhythm is manifest at faster heart rates it must be a faster rhythm. Therefore, late ventricular extrasystoles at faster heart rates may be of more significance than those at slower heart rates (Fig. 4). In addition, ventricular extrasystoles which appear at variable times towards the end of electrical diastole will more often be manifest and not dependent on sinus slowing.

It may be helpful to distinguish those rhythms that only become manifest with sinus slowing as

benign or slow idioventricular rhythms (<75/minute), and use the term rapid idioventricular rhythm for rhythms above this. The term ventricular tachycardia is well established for rhythms of 120/minute or over. Treatment may differ for slow and rapid idioventricular rhythms. Atropine may eliminate those arrhythmias associated with sinus slowing, but lignocaine may be a better treatment for those arrhythmias associated with a sinus rate of over 75/minute.

References

- Baxter, R. H., and McGuinness, J. B. (1974). Comparison of ventricular parasystole with other dysrhythmias after acute myocardial infarction. *American Heart Journal*, **88**, 443.
- Blackburn, H., Keys, A., Simonson, E., Rautaharju, P., and Punsar, S. (1960). The electrocardiogram in population studies: a classification system. *Circulation*, **21**, 1160.
- Chung, K.-Y., Walsh, T. J., and Massie, E. (1965). Ventricular parasystolic tachycardia. *British Heart Journal*, **27**, 392.
- De Soyza, N., Bissett, J. K., Kane, J. J., Murphy, M. L., and Doherty, J. E. (1974a). Association of accelerated idioventricular rhythm and paroxysmal ventricular tachycardia in acute myocardial infarction. *American Journal of Cardiology*, **34**, 667.
- De Soyza, N., Bissett, J. K., Kane, J. J., Murphy, M. L., and Doherty, J. E. (1974b). Ectopic ventricular prematurity and its relationship to ventricular tachycardia in acute myocardial infarction in man. *Circulation*, **50**, 529.
- Fluck, D. C., Olsen, E., Pentecost, B. L., Thomas, M., Fillimore, S. J., Shillingford, J. P., and Mounsey, J. P. D. (1967). Natural history and clinical significance of arrhythmias after acute cardiac infarction. *British Heart Journal*, **29**, 170.
- Goldberg, D. M., and Winfield, D. A. (1972). Diagnostic accuracy of serum enzyme assays of myocardial infarction in a general hospital population. *British Heart Journal*, **34**, 597.
- Julian, D. G., Valentine, P. A., and Miller, G. G. (1964). Disturbance of rate, rhythm and conduction in acute myocardial infarction. *American Journal of Medicine*, **37**, 915.
- Lawrie, D. M., Higgins, M. R., Godman, M. J., Oliver, M. F., Julian, D. G., and Donald, K. W. (1968). Ventricular fibrillation complicating acute myocardial infarction. *Lancet*, **2**, 523.
- Lipman, B. S., and Massie, E. (1965). *Clinical Scalar Electrocardiography*, 5th ed. Year Book Medical Publishers, Chicago.
- Norris, R. M., Mercer, C. J., and Yeates, S. E. (1970). Idioventricular rhythm complicating acute myocardial infarction. *British Heart Journal*, **32**, 617.
- Roelandt, J., and Schamroth, L. (1971). Parasystolic ventricular tachycardia. *British Heart Journal*, **33**, 505.
- Rothfield, E. L., Zucker, I. R., Parsonnet, V., and Alinsorin, C. A. (1968). Idioventricular rhythm in acute myocardial infarction. *Circulation*, **37**, 203.
- Sandler, I. A., and Marriott, H. J. L. (1965). The differential morphology of anomalous ventricular complexes of RBBB-type in lead V₁. Ventricular ectopy versus aberration. *Circulation*, **31**, 551.
- Scherf, D., and Schott, A. (1953). *Extrasystoles and Allied Arrhythmias*. Heinemann, London.
- Talbot, S. (1973). Fixed and variable coupling of ventricular extrasystoles. *Cardiology*, **58**, 118.
- Talbot, S., and Dreifus, L. S. (1975). Characteristics of ventricular extrasystoles and their prognostic importance. *Chest*, **67**, 665.
- Talbot, S., Dreifus, L. S., Watanabe, Y., Reich, M., Chiang, R., and Morris, K. (1973). Diagnostic accuracy of a 15-lead hybrid computer-aided electrocardiographic system. *European Journal of Cardiology*, **1**, 29.
- Watanabe, Y. (1971). Re-assessment of parasytostole. *American Heart Journal*, **81**, 451.
- Watanabe, Y., Pamintuan, J. C., and Dreifus, L. S. (1973). Role of intraventricular conduction disturbances in ventricular premature systoles. *American Journal of Cardiology*, **32**, 188.
- Wellens, H. J. J., Lie, K. J., and Durrer, D. (1974). Further observations on ventricular tachycardia as studied by electrical stimulation of the heart. Chronic recurrent ventricular tachycardia and ventricular tachycardia during acute myocardial infarction. *Circulation*, **49**, 647.

Requests for reprints to Dr. S. Talbot, Department of Cardiology, Hammersmith Hospital, Du Cane Road, London W12 0HS.