The Nature of Ventricular Ectopic Beats in Chronic Ischaemic Heart Disease

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SUMMARY

Periodic 6- to 7-hour dynamic electrocardiographic studies were performed on 84 active postmyocardial infarction patients to evaluate frequency, persistency, configuration and coupling periods of ventricular ectopic beats (VEB). It was found that VEB were present in 73% on first evaluation, rising to 95% with subsequent studies. A method of classification of frequency of VEB is presented, and a comparison between the occurrence of VEB on routine resting ECGs and monitoring of ECG on magnetic tape, is made. The frequency of VEB of particular patients tends to remain remarkably constant on subsequent study. Also a particular configuration of VEB tends to remain constant even over prolonged periods of time. Considering the observation that the longer the VEB are monitored the wider the variation in coupling periods, attempts at the differentiation of true ventricular extrasystole from ventricular parasystole met with little success. Considerable study needs to be done to elucidate the factors causing VEB to become manifest or to change in incidence.

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The association of ventricular ectopic beats (VEB) and ischaemic heart disease is well known. However, excess mortality has been reported in men exhibiting frequent VEB after myocardial infarction.[1-3] Identification of a risk factor must ideally lead to modification thereof. Unfortunately it is not yet known whether successful suppression of VEB in these patients would have a beneficial effect on the associated risk. Conceivably, successful suppression of electrical instability, without causing any other adverse effects, would lead to a decrease in the incidence of sudden death in these high-risk patients. Conversely, if successful elimination of other risk factors leads to the disappearance of VEB, their continued absence could be of prognostic value.

If VEB are not detected during routine and casual examination of a patient, it does not mean that they do not exist, nor can the frequency or persistence of any detected VEB be accurately judged. Also, VEB are traditionally designated as either true ventricular extrasystoles or as ventricular parasystoles. This dual manifestation presumably denotes different mechanisms, and would therefore be of different significance. This study was designed to determine the pattern and consistency of VEB in active postmyocardial infarction patients.

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PATIENTS AND METHODS

The study was conducted on 80 males and 4 females who had been hospitalised at 1 Military Hospital with the clinical picture of acute myocardial infarction, substantiated by serial electrocardiographic and serum enzyme changes. After discharge from hospital the patients were periodically evaluated at the Military Medical Institute. The average age at the first evaluation was 51 years (range 29 - 63 years).

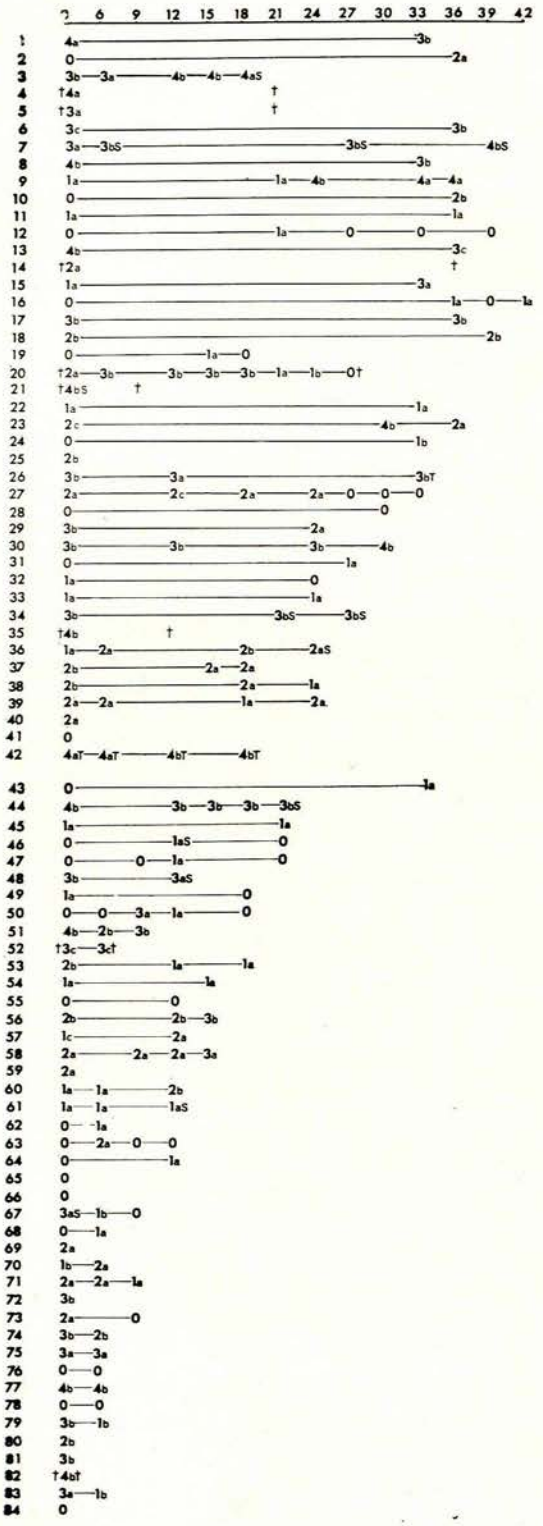
Evaluation included a resting multichannel 12-lead electrocardiogram (ECG) (average recording time 55 seconds) and a 6- to 7-hour continuous ECG on magnetic tape. The patients were required to wear the recorder during a normal working day, and to log activity and any symptoms experienced. A bipolar lead was utilised in all patients. The negative electrode was placed in the second right intercostal space on a vertical line through the junction of the middle and lateral thirds of the clavicle. The positive electrode was placed in the V5 position.

The tapes were subsequently scanned on an Avionics electrocardioscanner. The presence, number, configuration and the coupling period with the preceding normal sinus beat were noted. The absolute number of VEB were graded from 0 to 4 (Table I). Unifocal ectopic beats are indicated by *a*, bifocal VEB by *b* and trifocal VEB by *c*. Two consecutive VEB of the same configuration were designated a salvo (S), whereas 3 or more consecutive VEB were designated as ventricular tachycardia (T) (Table II).

TABLE I. GRADING OF NUMBER OF VENTRICULAR ECTOPIC BEATS

Grade	No. of VEB per hour	Total No. of VEB over 7 hours	Approximate No. of VEB per normal sinus beat
0	0	0	0
1	<1	1 - 6	<1/3 600
2	1 - 7	7 - 49	1/3 600 - 1/500
3	8 - 50	50 - 350	1/500 - 1/70
4	>50	>350	>1/70

TABLE II. NATURE OF VEB IN 84 PATIENTS FOLLOWED OVER A PERIOD OF MONTHS



Grade 0 - 4 = no. of VEB; a, b, and c = uni-, bi- or trifocal origin respectively of VEB; S = salvos; T= ventricular tachycardia; and † + death after last tape recording

At least two months were allowed to elapse between the acute episode and the first dynamic electrocardiograph. Care was taken to exclude possible anti-arrhythmic or dysrhythmic effects of various antihypertensive agents, beta-blockers, sedatives, etc., which some of the patients were taking at various times. During the period of evaluation conclusions regarding the VEB were taken into consideration only when a patient was stabilised on the prescribed medication.

RESULTS

A total of 237 pairs of resting and dynamic ECGs were studied. On the resting ECGs one or more VEB were noted in 45 instances, whereas 180 of the dynamic studies exhibited VEB, i.e. 25% of positive dynamic studies exhibited VEB on the corresponding routine ECG. Fig. 1 depicts this incidence according to the applied grading system.

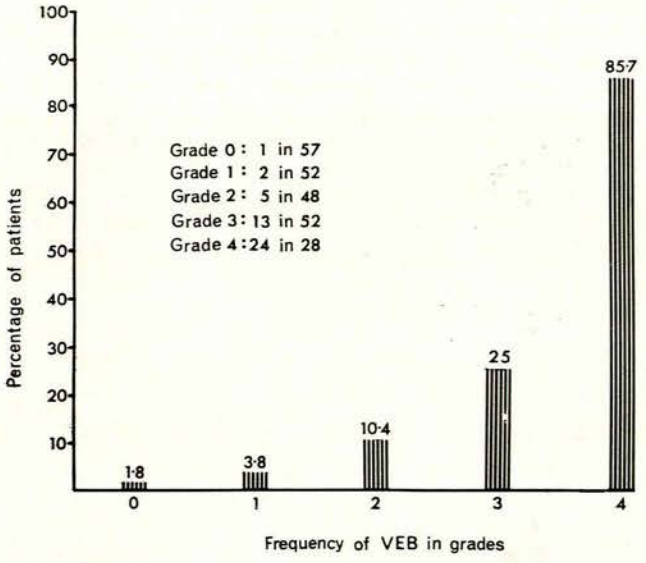


Fig. 1. Graph of percentage of patients showing VEB on dynamic study also exhibiting VEB on routine ECG.

A previous Holter monitoring study from this Institute reported a VEB incidence of 78% in these patients.[4] In this larger group an incidence of 73% was noted on the first dynamic study, but only 4 patients remained free of ectopics on subsequent studies, resulting in an incidence of 95%.

Results of 209 of the 237 dynamic studies done are summarised in Table II (28 were eliminated on grounds of possible anti-arrhythmic effects of treatment at the time). In studying Table II it is evident that in individual cases the total number of VEB tends to remain remarkably constant, even over prolonged periods of time. Exceptions to this general behaviour were noted in a few cases. In 2 patients VEB were present on their resting 12-lead ECGs, while on the subsequent tape recordings less than one ectopic beat per hour was noted. In 3 patients with 1 - 6

ectopics/7 hours, ectopics occurred in salvos. Three patients with 7 - 50 VEB/hour experienced transient episodes of bigeminy. One patient with approximately 10 VEB/hour revealed one episode of ventricular tachycardia with complexes of different configuration to his regularly occurring ectopics.

By utilising a constant lead system the configuration of the recorded complexes stayed unchanged in individual patients. A comparison was therefore made between the configurations of VEB in subsequent tapes. The histogram depicts the number of patients with VEB of identical configuration in repeat recordings (Fig. 2).

A wide variation in coupling intervals of VEB of identical configuration in individual cases was observed (Table III). It was clear that the longer the period of observation or the longer the period of monitoring, the greater the variation in coupling intervals of VEB. No attempt was made to represent this graphically. Examples of this phenomenon are shown in Figs 3 and 4.

TABLE III. VARIATION OF COUPLING INTERVALS IN SECONDS OF VEB OF IDENTICAL CONFIGURATION IN INDIVIDUAL CASES

	<0,04	0,04 - 0,07	0,08 - 0,12	>0,12
No. of instances	16	14	21	31

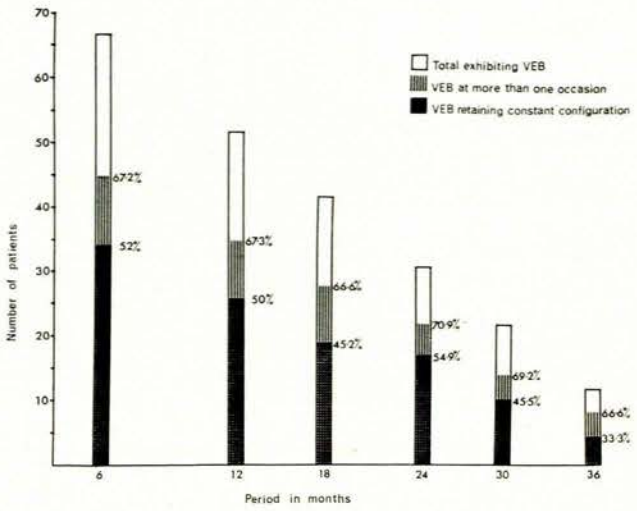


Fig. 2. The cumulative histogram depicts the total number of patients exhibiting VEB, the number of patients exhibiting VEB on more than one occasion, and the number in whom the VEB retained the same configuration.

There were 8 deaths during the period of follow-up. In 7 instances death occurred suddenly, i.e. within 24 hours of onset of symptoms, and was presumably of

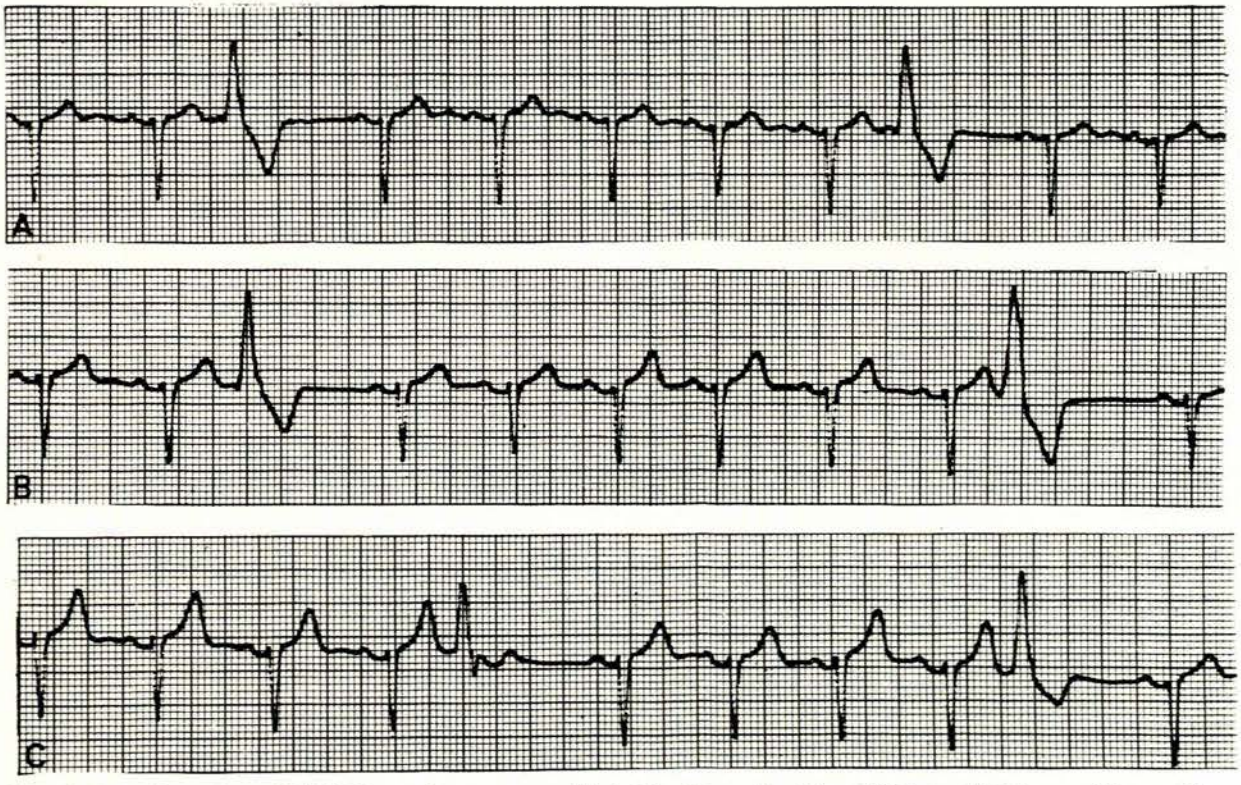


Fig. 3. A total number of 60 VEB per hour was manifested in this patient from 0820 to 1322 hours. The coupling interval during this period of time was constant at 0,44 seconds (A). At 1322 hours an ectopic beat with a coupling interval of 0,32 seconds appeared (B). From 1322 hours until completion of the tape at 1520 hours the frequency of VEB remained the same, but the coupling intervals varied between 0,40 and 0,44 seconds (C).

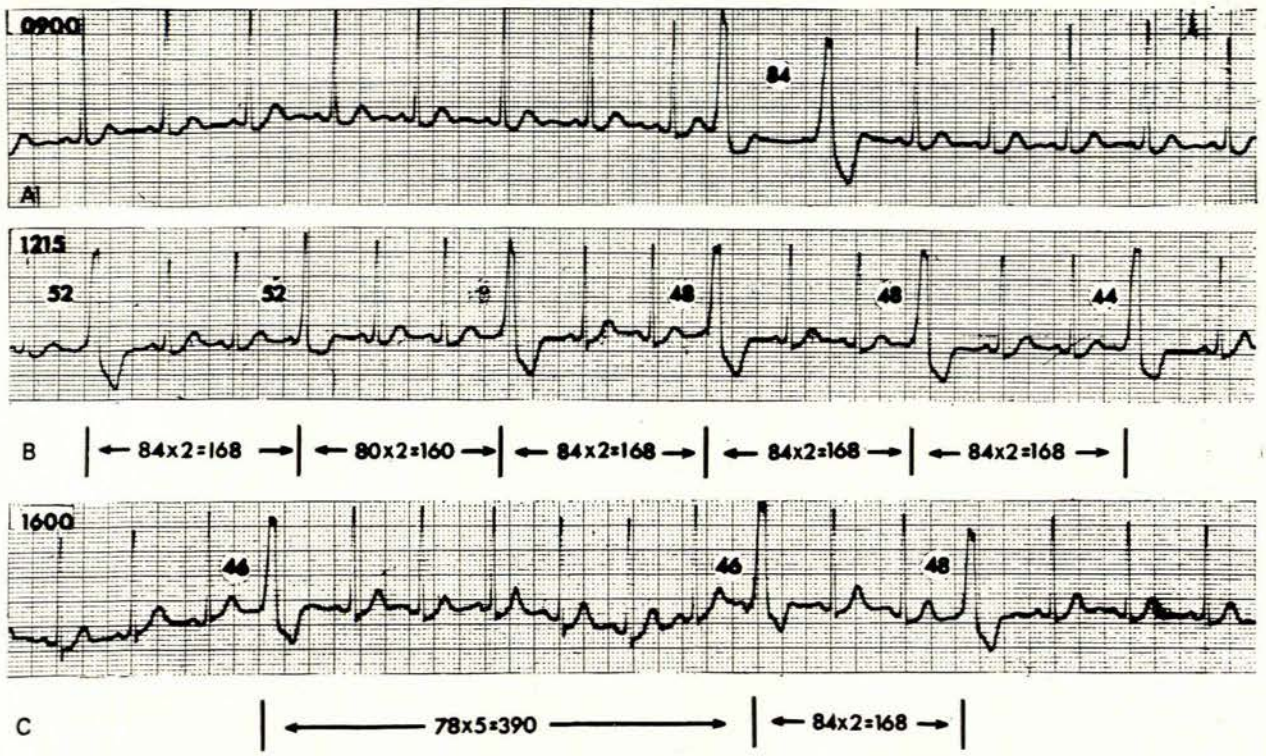


Fig. 4. Representative tracings of patient No. 75 who exhibited a total of 150 VEB over the 7-hour recording period. In A at 0900 hours the interectopic interval of 0,84 sec could represent a ventricular parasystolic discharge rate of 72/min. In B the variation in coupling intervals, (coupling intervals in hundredths of a second), the presence of a fusion beat and plausible parasystolic rate fulfils the criteria for the diagnosis of ventricular parasystole. In C, however, the presumably instantaneous variation of ectopic discharge rate between 77 and 72/min renders the diagnosis equivocal, unless an entirely new discharge rate or some other manifestation of the same focus is presumed. Conversely, if C represented the predominant pattern during the recording on this patient, parasystole would not have been considered.

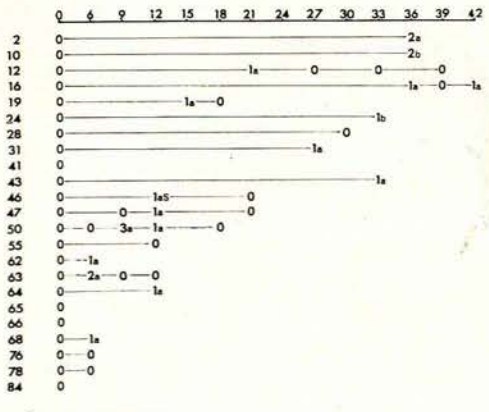
cardiac origin in all 7 cases. Patient No. 14 died of bronchial carcinoma.

Patient No. 20 is of interest. Since he was on 0,25 mg digoxin *b.i.d.* and 40 mg oxprenolol *t.d.s.* for no apparent reason, the drugs were discontinued 1 week before his sudden death. No definite conclusions can be drawn, but it is apparent from Table II that all the patients who died exhibited a high frequency of VEB.

DISCUSSION

From this study it appears that in patients with ischaemic heart disease who exhibit infrequent VEB, ectopic activity can be detected with confidence only when use is made of dynamic electrocardiography, i.e. by monitoring a patient for several hours while performing routine activities. In our group of 84 patients 80 revealed VEB on dynamic study, whereas only 23 patients showed evidence of ectopic activity on routine ECG. It is noteworthy, however, that those with no VEB on initial dynamic study tend to have either no VEB, or very few, on subsequent studies (Table IV). It should be noted that there is very little difference between no VEB, graded 0, and less than one ectopic beat per hour, graded 1.

TABLE IV. NATURE OF ECTOPIC ACTIVITY IN 23 PATIENTS WITH 0 VEB ON INITIAL STUDY



Abbreviations the same as in Table II.

It is remarkable that the total number of VEB tends to remain constant in a particular patient over a period of months or even years. The factors leading towards the occurrence of VEB in patients with ischaemic heart disease remain largely unknown. From the logs the

patients kept it would seem as though the majority were unaware of the arrhythmia or of factors which constantly correlate to the emergence of VEB. No constant increase or decrease in the number of VEB was noted with the onset of either a bradycardia or a tachycardia. This confirms the observation of Vedin *et al.*[5] that the proportion of men having premature beats did not change between the resting state and various levels of workload, but is in contrast to the increasing frequency of VEB noted with progressive stress testing in middle-aged men free of manifest heart disease.[6] Goldschlager *et al.*[7] noted both an increase and a decrease of VEB with exercise in patients with coronary artery disease. Considerable study needs to be done to identify the factors which cause VEB to become manifest. Nevertheless it seems justified to postulate that, if in fact there is a causal relationship between ischaemia and VEB, an increase in the number. or the emergence of new foci, would denote worsening of the underlying condition. Conversely, therapy which is not directly anti-arrhythmic in nature and which leads to a decrease in the number of VEB, may denote objective evidence of its efficacy.

An additional interesting observation was that a particular configuration of VEB in a patient tended to recur in subsequent studies of the particular patient (Fig. 2). It is realised that the presumed identity of VEB is based on the configuration in one lead only and conceivably a multichannel recording might reveal an entirely different configuration of a manifest ectopic beat in some leads. A recent study also demonstrated that, at least in the dog heart, the ventricular wall *per se* was the determinant factor in the configuration of VEB rather than the exact site of origin.[8] However, it would be reasonable to accept that the absolutely identical configuration indicates that either the emanating focus remains the same or that foci in close proximity are responsible for the VEB over the period of observation. Assuming a localised area of irritability or ischaemia responsible for VEB over a period as long as 3 years, it is confusing that only one or two areas are active and remain active in extensive coronary artery disease with presumably multiple areas of ischaemia.

As is the case in healthy individuals, it is apparent that the differentiation between true ventricular extrasystole and ventricular parasystole is in fact often unrealistic.[9] It was observed that for hours the coupling interval would vary less than 0,04 seconds, only to suddenly change by more than 0,12 seconds, with no change in configuration or heart rate. It can be stated categorically that the longer a ventricular ectopic beat of a particular configuration is monitored, the greater are the chances of finding a variation in coupling period. We therefore feel that in chronic myocardial ischaemia caution should be applied in attaching too much prognostic significance to the degree of prematurity of ectopic beats.

In conclusion, knowledge of the consistency in both total number and a tendency for the configuration of VEB in a particular patient to remain constant, might serve as a useful parameter in judging efficacy of therapy for the underlying cardiac condition.

REFERENCES

1. Coronary Drug Project Research Group (1972): Ann. Intern. Med., **77**, 677.
2. *Idem* (1973): J. Amer. Med. Assoc., **223**, 1116.
3. Kotler, M. N., Tabatznik, B., Mower, M. M. and Tominaga, S. (1973): Circulation, **47**, 959.
4. Myburgh, D. P. (1972): S. Afr. Med. J., **46**, 1162.
5. Vedin, J. A., Wilhelmsson, O. E., Wilhelmsen, L., Bjure, J. and Ekströmjodal, B. (1972): Amer. J. Cardiol., **30**, 25.
6. Blackburn, H., Taylor, H. L., Hamrell, B., Buskirk, E., Nicholas, W. C. and Thorsen, R. D. (1973): *Ibid.*, **31**, 441.
7. Goldschlager, N., Cake, D. and Cohn, K. (1973): *Ibid.*, **31**, 434.
8. Mautner, B. and Girotti, A. L. (1973): Amer. Heart J., **85**, 389.
9. Myburgh, D. P. and Lewis, B. S. (1971): *Ibid.*, **82**, 307.