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Sinus bradycardia

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This paper presents the features of 46 patients with unexplained bradycardia. Patients were admitted to the study if their resting atrial rate was below 56 a minute on two consecutive occasions. Previous electrocardiograms and the response to exercise, atropine, and isoprenaline were studied.

The ages of the patients varied from 13 to 88 years. Only 8 had a past history of cardiovascular disease other than bradycardia, but 36 had syncopal or dizzy attacks. Of the 46 patients, 35 had another arrhythmia in addition to bradycardia; at some stage, 16 had sinus arrest, 15 had junctional rhythm, 12 had fast atrial arrhythmia, 16 had frequent extrasystoles, and 6 had atrioventricular block. None had the classical features of sinoatrial block. Arrhythmias were often produced by exercise, atropine, or isoprenaline. Drug treatment was rarely satisfactory, but only 1 patient needed a permanent pacemaker.

It is suggested that the majority of the patients were suffering from a pathological form of sinus bradycardia. The aetiology remains unproven, but the most likely explanation is a loss of the inherent rhythmicity of the sinoatrial node due to a primary degenerative disease. The descriptive title of 'the lazy sinus syndrome' is suggested.

Bradycardia with a slow atrial rate is usually regarded as an innocent condition common in certain types of well-trained athlete, but occasionally it may occur in patients with symptoms of heart disease. Laslett in 1909 described a 40-year-old woman who had a slow pulse and complained of fainting attacks. Using a MacKenzie Polygraph, he showed that the attacks were associated with arrest of the whole heart. Subsequently, there have been a number of reports of patients who tended to have a slow atrial rate and were subject to other disorders of cardiac rhythm. In some instances the bradycardia was considered to be sinus (Campbell, 1943; Pearson, 1945; Short, 1954; Birchfield, Menefee, and Bryant, 1957), while in others sinoatrial block was suggested as the mechanism (Levine, 1916; Cowan, 1939; Stock, 1969; Rokseth *et al.*, 1970). The clinical details have varied from case to case, but commonly reported features both in those described as having sinus bradycardia and those with sinoatrial block are syncope and episodes of tachycardia. The cardiograms have on occasions shown sinus arrhythmia, wandering pacemaker, sinus arrest, with and without junctional (nodal) escape, paroxysmal atrial tachycardia, atrial flutter, and atrial fibrillation. The study presented in this paper was undertaken in an attempt to define the clinical syndrome of bradycardia with a pathologically slow atrial rate and to clarify the nature of the arrhythmia.

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Plan of study

Patients were admitted to the study if their resting atrial rate was below 56 a minute on two consecutive visits, providing they were not taking drugs of the digitalis group, or propranolol, quinidine, procainamide, methyldopa, or bethanidine. Patients with untreated myxoedema, jaundice, or known raised intracranial tension were excluded as were those with temporary slowing of the heart in association with recent myocardial infarction (within 28 days), or acute carditis.

Forty-six patients fulfilled the criteria of the study. Most of these were found after a direct approach was made to the family doctors in the clinical area. Two hundred and ninety doctors were asked to give details of patients with heart block or pulse rates below 56 a minute, and replies were obtained from all but 8. A full report of this survey has been given by Shaw and Eraut (1970). Nineteen patients were seen in routine hospital practice over a period of 6 years and 2 were reported to us by colleagues who knew of our interest in the condition. The patients have been followed up at intervals for periods up to 6 years. Previous electrocardiograms were obtained in 23 of the 46 patients, the average time between the first and last cardiogram being approximately 5 years.

Most of the patients were seen in the Cardiac

Department at the Royal Devon and Exeter Hospital. Six were unable to attend and were visited at their homes only. One patient (Case 20) was not seen by us; Dr. Cosh of Bath has kindly sent serial reports. The patients were asked if they suffered from syncopal attacks, dizziness, dyspnoea, or chest pain on effort, and inquiry was made for a past history of diphtheria, rheumatic fever, or myocardial infarction. After a general physical examination, a standard 12-lead cardiogram was recorded and the resting heart rate was taken from a 3-foot strip of lead II. In 32 patients a continuous recording was made for 3 minutes at a slow speed with a 3-channel mingograph recorder. Records were repeated after exercise in 42 patients, and in 27 patients records were taken throughout the standard exercise test used in the department. This consists of walking up and down steps at a fixed rate for 3 minutes, followed by a 3-minute rest with continuous cardiogram recording. The effect of 0·6–1 mg atropine was studied by serial cardiograms in 22 patients, the drug being given by intravenous injection in 3 and orally in the others. All experienced dryness of the mouth. Isoprenaline was given by intravenous injection in 3 patients and sublingual tablets (20 mg) in 24 patients.

The cardiogram was examined to check the rhythm and atrial rate at rest and during exercise. A search was made for junctional (nodal) rhythm, periods of sinus arrest with or without escape beats, fast atrial arrhythmia, atrial tachycardia, flutter or fibrillation, coupled and frequent extrasystoles (more than 1 for 10 sinus beats), and for any evidence of abnormal atrioventricular conduction. The PR interval and the duration and height of the P waves were measured in lead II. Where atrial arrest occurred, the duration of the arrest and the preceding and following PP intervals were measured to assess the presence or absence of single blocked complexes of sinoatrial block or of Wenckebach conduction in sinoatrial block. First and third degree sinoatrial block cannot be recognized from the cardiogram (McGarry, 1966). The criteria for the diagnosis of second degree sinoatrial block used in this study are based on the work of Winton (1948), McGarry (1966), and Stock (1969), and are as follows.

1 Isolated single dropped beats with a prolonged PP cycle, length twice the length of the predominant PP interval.

2 Several consecutive dropped beats with a prolonged PP length 3, 4, or 5 times the predominant PP interval.

3 Sudden doubling or trebling of the atrial rate, often in response to exercise or atropine and implying a relatively fixed 2:1 or 3:1 sinoatrial block.

4 Wenckebach conduction in sinoatrial block with beats dropped more or less regularly. In this instance there should be progressive shortening of the PP interval up to the pause, the PP interval including a blocked sinoatrial impulse is shorter than twice the length of the PP interval preceding it (provided that only one impulse is blocked), and the PP interval following the dropped sinoatrial impulse is longer than the PP interval preceding it. In cases of doubt the duration of the sinus cycle can be calculated and the presence or absence of sinoatrial block tested by formulae suggested by Schamroth and Dove (1966).

Results

Basic clinical data are given in Table 1. The age range of the patients with bradycardia was wide (13 to 88), but over half were elderly, being within the range 55 to 75. Only 8 patients had a history of heart disease other than the bradycardia. Four patients had had a myocardial infarction. Two patients had congenital heart disease: one of these had an atrial septal defect, and one had dextrocardia and situs inversus. One patient was considered to have an idiopathic cardiomyopathy. One patient had rheumatic heart disease and a further 4 gave a history of childhood rheumatic fever, but had no clinical history of heart disease. Neurological disease was also rare. Three had had cerebrovascular accidents, and 3 had been considered to have epilepsy, though on review this diagnosis was subsequently abandoned in one who had mild presenile cerebral degeneration, and was unsubstantiated in another. Two patients had suffered from myxoedema and were on thyroxine, and one had had a partial thyroidectomy but was euthyroid at the time of the study.

Thirty-six of the 46 patients had symptoms of circulatory impairment. Twenty-three experienced disturbance of consciousness, in 14 there were episodes of complete loss of consciousness, and 15 had attacks of dizziness; 6 patients had both. Central chest pain on exertion was complained of by 17 patients; only 3 of these had a past history or cardiographic evidence of myocardial infarction. Twenty-nine patients complained of breathlessness on exercise, and 10 patients complained of swollen ankles.

The mean blood pressure for the group was 150/77 mmHg, the range being 100/60 to 240/100 mmHg. Only 1 had a diastolic blood pressure above 100 mmHg.

The resting atrial rate at the time of the last assessment when sinus rhythm was dominant ranged from 28 to 54 a minute, the mean for the group being 44 (see Table 1). The rate increased with exercise but not to the same extent as observed in normal subjects. The mean maximal exercise atrial rate in the patients fully studied was some 20 a minute below the mean normal value for this department. Eleven of the patients developed an arrhythmia during or just after exercise, which

TABLE I *Clinical data on 46 patients studied*

Case No.	*Age (yr)*	*Sex*	*Minimum length of history (yr)*	*Symptoms*	*Atrial rate/min*	*Arrhythmia*		
						Sinus arrest	*Junctional rhythm*	*Fast atrial arrhythmia*
36	13	M	1	Breathlessness	54			
43	15	M	1	Disturbance of consciousness	38			
6	17	M	1	—	43			
34	18	M	18	Disturbance of consciousness	35	*	*	
46	26	M	19	Breathlessness, disturbance of consciousness	40		*	
27	38	M	29	—	36			*
13	39	M	1	Angina, breathlessness	52			
20	40	F	13	Disturbance of consciousness	35	*	*	*
40	42	M	3	Angina, breathlessness, disturbance of consciousness	36			
38	43	M	2	—	50			
32	43	F	35	Disturbance of consciousness	52			*
42	44	M	1	Angina	48			
41	47	M	4	Breathlessness	48	*		
35	47	M	33	Angina, breathlessness, disturbance of consciousness	38			
4	49	M	5	Angina, breathlessness	44			
3	54	M	1	Breathlessness, disturbance of consciousness	50			
31	54	F	1	—	50			
19	59	F	3	Breathlessness, disturbance of consciousness, oedema	47			*
25	60	M	5	Breathlessness, disturbance of consciousness	50	*	*	*
10	62	M	15	Breathlessness, disturbance of consciousness	43	*	*	
26	62	M	32	Breathlessness, disturbance of consciousness	32	*	*	*
33	62	M	15	Breathlessness	40	*	*	
5	62	F	3	Disturbance of consciousness, oedema	30	*	*	
7	63	M	2	Breathlessness, disturbance of consciousness	50			
15	64	F	1	Angina, disturbance of consciousness	50	*		
1	65	M	1	—	52			
2	69	M	1	—	50			
17	69	M	1	Angina, breathlessness	45			
8	70	F	5	Angina, breathlessness, oedema	50			
22	70	F	18	Angina, breathlessness, disturbance of consciousness, oedema	40	*		*
16	71	M	8	—	42		*	
18	71	M	1	Angina	48			
44	71	M	50	Angina, breathlessness, disturbance of consciousness, oedema	40			
45	71	M	61	Breathlessness, disturbance of consciousness	30	*	*	
9	73	F	14	Breathlessness, oedema	54			
11	74	M	38	Breathlessness, disturbance of consciousness	40	*	*	
12	75	M	14	Angina, breathlessness	52			
14	75	F	9	Angina, breathlessness, oedema	36	*	*	
21	75	F	3	Angina, breathlessness, disturbance of consciousness	45		*	*
23	76	M	3	Angina, breathlessness	40	*		*
28	77	M	6	Angina	54			*
24	79	M	18	Angina, breathlessness, disturbance of consciousness	28			*
39	80	M	8	Breathlessness, oedema	42	*	*	
37	81	M	1	Breathlessness	50			
29	84	M	18	Breathlessness, disturbance of consciousness, oedema	36	*	*	*
30	88	M	5	Disturbance of consciousness, oedema	54			

was not present at rest at some time. Bradycardia tended to persist despite atropine. In the 22 patients given this drug the mean atrial rate only increased from 44 to 60 a minute, while 6 patients developed an arrhythmia. The mean atrial rate in the 26 patients given isoprenaline rose to 54 a minute, an increase of 10 a minute, and arrhythmias developed in 3 patients.

The cardiograms showed that the P waves tended to be of low voltage, being difficult to distinguish in lead I and in 4 patients were hardly discernible in lead II. In those in whom serial cardiograms were available, the amplitude of the P waves decreased over the years. The P waves were broad (0·10 sec or more in lead II) in 26 of the 46 patients, and 10 had P waves of 0·11 sec or more. The PR interval was prolonged in excess of 0·2 sec in 4 patients, the time intervals being 0·28, 0·3, 0·34, and 0·52 sec. One patient was in partial atrioventricular block on some occasions and complete block on others, and one (Case 40) had episodes of complete block.

In 16 patients there were periods when the atrium failed to depolarize at the expected time and there would be a pause in atrial electrical activity for from 1·4 to 3·5 sec (Fig. 1 and 2). Their occurrence was quite unrelated to respiration. The duration of atrial standstill bore no relation to the preceding PP interval, nor was there a tendency for the PP intervals to shorten before atrial standstill. The pauses in atrial activity in these patients, therefore, were considered to be associated with sinus arrest. These were usually followed by a junctional escape beat (Fig. 1), though in several the sinoatrial node restarted spontaneously on occasions (Fig. 2). Sinus arrhythmia was present in 7 of the 46 patients but coincided with sinus arrest in only 1. In 4 the PP interval varied by over 0·75 sec over the respiratory cycle; even in these cases analysis of the cycle lengths using the formula of Schamroth and Dove (1966) failed to conform to sinoatrial block.

Junctional rhythm was observed at one time or another in 15 patients. In 12 the sinus rate was so slow at rest that on occasion the atrioventricular junctional tissue took over as pacemaker, and in 3 additional patients junctional rhythm was precipitated by drugs or exercise. In 2 patients a ventricular centre at times competed with the sinoatrial node (Fig. 3). Five patients showed atrioventricular dissociation. In 4 of these the rates of the atrium and the lower pacemaking centre were so similar that atrioventricular synchrony (accrochage) was suspected.

Fast atrial arrhythmia was recorded in 12 patients, in 4 atrial tachycardia occurred, and in 3 there was atrial flutter. One patient with atrial tachycardia and one with flutter later developed atrial fibrillation. Seven patients had episodes of atrial fibrillation. Atrial flutter or fibrillation became established in 9 patients. One patient (Case 27) was defibrillated by DC shock and settled down to an asymptomatic sinus bradycardia at a rate of 36. Subsequent questions revealed that he had a bradycardia dating back to his student days when he was found to have a pulse in the 40's and that this had been present up to the onset of fibrillation.

Thirty-five of the patients had one or more arrhythmia in addition to the bradycardia, and 24 had a major disturbance of rhythm such as sinus arrest, junctional rhythm, or fast atrial arrhythmia (see Table 2).

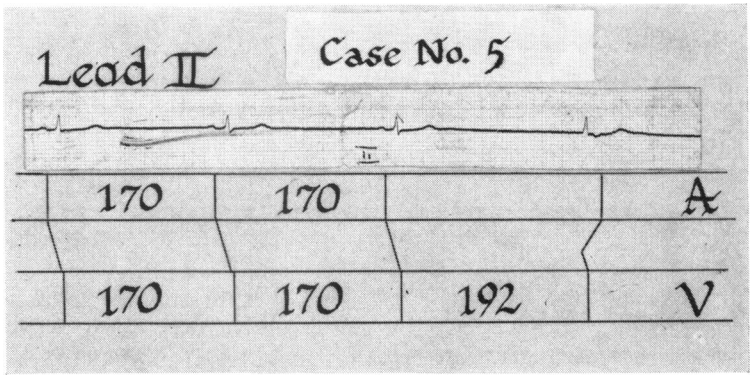
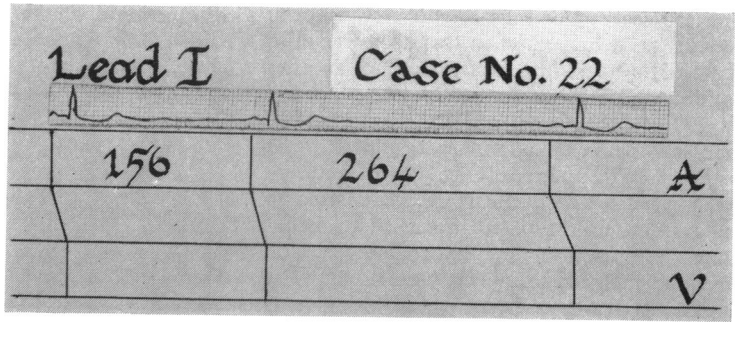


FIG. 1 *Cardiogram from Case 5 showing 3 sinus beats followed by sinus arrest and a junctional escape. The figures for the time intervals are in hundredths of a second.*

FIG. 2 *This recording from Case 22 shows 2 sinus beats followed by sinus arrest. The sinus node restarts spontaneously after a 2½-second pause. The figures for the time intervals are in hundredths of a second.*



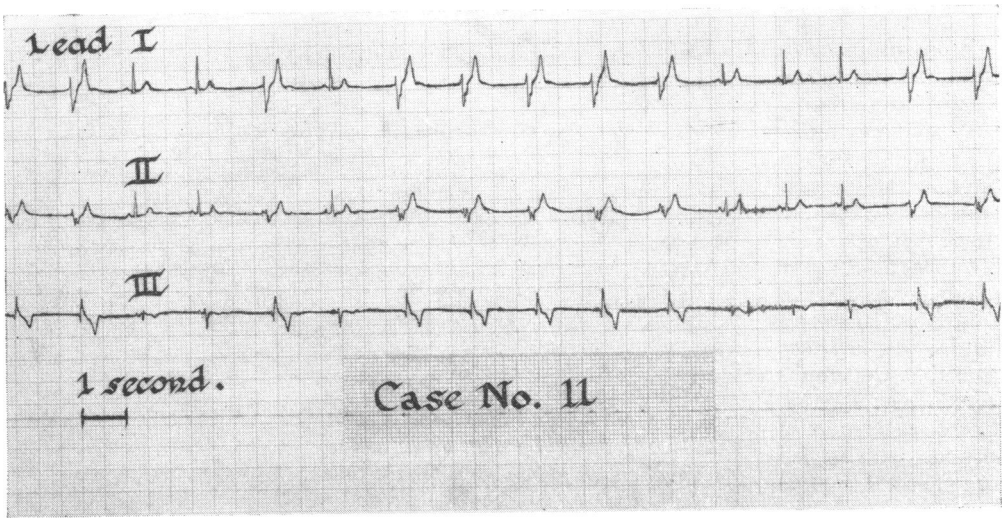


FIG. 3 *Slow speed recording of the 3 standard leads in Case 11. The trace shows runs of sinus beats at a rate of 42 a minute alternating with beats from a ventricular pacemaker at 40 a minute and 2 fusion beats.*

Cardiograms have been obtained antedating the study in 18 patients, and these show that bradycardia had been present for up to 14 years, the average being 7 years. The average length of the history of bradycardia in the 35 patients with one or more arrhythmia was 14 years, and in 11 patients the history suggested that bradycardia was noted in childhood or early adult life. The length of the history of bradycardia in the 11 patients who showed no arrhythmia was 1 year or less, apart from one patient with a 2-year history.

Long-term treatment was undertaken in 11 patients in the attempt to increase the heart rate. Nine patients were given 'saventrine' (isoprenaline hydrochloride), but the dose required to increase the sinoatrial rate also tended to provoke fast atrial arrhythmia or multiple atrial or ventricular extrasystoles, and only 4 patients are still taking the drug. Tincture of belladonna was used in 3 patients. Again, it tended to produce abnormal rhythms and unpleasant side effects, and no long-term benefit was achieved when this drug was used alone. However, the heart rate in another patient who required propranolol to control attacks of paroxysmal atrial tachycardia was increased when belladonna was added to the regimen. In one patient (Case 5) with frequent syncopal attacks, the heart rate decreased to 30 a minute despite treatment with belladonna and isoprenaline hydrochloride, and an on-demand pacemaker was fitted. The pacemaker has now been in place for over 2 years and there have been no further syncopal attacks.

Discussion

Chronic bradycardia is usually regarded as physiological, so that it might be expected that the majority of patients in the present study would have a slow atrial rate for physiological reasons. However, most had symptoms of cardiovascular disease, and some abnormality of rhythm, in addition to bradycardia, was found in all but 11 of the 46 patients. It seems, therefore, that in these 35 patients the slow atrial rate was a manifestation of heart disease and was in fact a pathological atrial bradycardia. The mechanism could be sinoatrial block or sinus bradycardia. Since depolarization of the sinoatrial node cannot be

TABLE 2 *Cross reference of types of disturbance in rhythm or conduction in 35 patients who had arrhythmia in addition to bradycardia*

	Sinus arrest	*Junctional rhythm*	*Fast atrial arrhythmia*	*Frequent extrasystoles*	*AV block*
Sinus arrest	*16*	12	6	5	1
Junctional rhythm	12	*15*	5	5	2
Fast atrial arrhythmia	6	5	*12*	4	2
Frequent extrasystoles	4	5	4	*16*	1
AV block	1	2	2	1	*6*

recorded (McGarry, 1966), it is impossible to disprove the existence of sinoatrial block. However, none of the patients in the present study had the classical features of this condition, nor could a change in block be produced by measures that release the parasympathetic drive on the sinoatrial node, such as exercise or the administration of atropine. Indeed, in the majority of the patients the increase in the rate of depolarization of the sinus node in response to exercise and other agents was abnormally small, while in 7 cases the response was so poor that the atrioventricular junctional tissue took over as pacemaker. A further point of distinction between the patients of the present study and those of cases of sinoatrial block is the amplitude of the P wave in the cardiogram. The very low wave, often indiscernible in lead I, seen in the patients with bradycardia, is in marked contrast to the sharply demarcated P waves illustrated by Levine (1916) in his classic description of sinoatrial block. It seems reasonable, therefore, to attribute the slow atrial rate to sinus bradycardia rather than to sinoatrial block.

Most of the arrhythmias seen in this study have been reported to occur in patients with sinus bradycardia by previous authors, though the frequency of junctional rhythm does not appear to have been emphasized. Periods of sinus standstill were described by Pearson (1945), Short (1954), and Birchfield *et al.* (1957), and alternating episodes of bradycardia with atrial tachycardia, atrial flutter, or atrial fibrillation were observed by Short (1954) and Birchfield *et al.* (1957). These workers also recorded junctional rhythm after the administration of atropine. The spontaneous onset of junctional rhythm which alternated with atrial tachycardia and atrial flutter was found in a patient with sinus bradycardia by Cohen, Kahn, and Donoso (1967), and a similar case with permanent junctional rhythm and 'periods of standstill of the whole heart' and obvious bradycardia was described by Wedd and Wilson (1930).

There have been reports of patients with bradycardia ascribed to sinoatrial block who have developed other arrhythmias or responded abnormally to exercise or atropine (Cowan, 1939; Stock, 1969; Rokseth *et al.*, 1970). Cowan (1939) reported a group of patients showing periods of atrial standstill which he attributed to sinoatrial block. In at least 2 of these cases the periods of standstill were followed by escape beats and at times junctional rhythm alternated with sinus rhythm in a way similar to that seen in sinus bradycardia. Stock (1969) describes 3 patients with bradycardia and syncopal attacks, 2 of whom subsequently developed atrial fibrillation. In 1 of these cases intravenous atropine was followed by junctional rhythm. As the author implies, this is not the response to be expected had the patient been suffering from sinoatrial block, and it seems probable that one or more of these were cases of sinus bradycardia. Rokseth *et al.* (1970) described 14 patients, 12 with symptoms of dizziness and syncopal attacks that they attributed to sinoatrial block. Six of their patients had paroxysmal supraventricular tachycardia and 7 patients were described as having sinus arrest, the pause being terminated by a junctional escape beat. From the data available it seems that 10 of the 14 patients present features very similar to those seen in sinus bradycardia, and it is tempting to consider that some had this condition. It is possible that sinoatrial block and sinus bradycardia may coexist in the same patient; Laake (1946) described a patient with sinus bradycardia who developed sinoatrial block after digitalis and quinidine. Again, Lown (1966) described a group of patients who after electrical defibrillation failed to develop a sustained sinus rhythm, but had erratically recurring atrial complexes of varying morphology associated with both sinoatrial standstill and sinoatrial block.

The cause of the sinus bradycardia remains unexplained in the majority of cases. The case reported by Pearson (1945) later was found to have carcinomatous infiltration of the cardiac plexus (Pearson, 1950). Theoretically, this might have increased the parasympathetic drive on the sinus node, though the author considered it unlikely. Laslett (1909) suggested that excessive vagal tone was a possible cause for the bradycardia and episodes of cardiac standstill in the patient he described, and Wedd and Wilson (1930) also attributed the cardiac standstill in their case to vagal activity. Short (1954), however, did not consider that this mechanism was likely to have been responsible for the bradycardia in his 4 patients. As already stated, in the present study atropine tended to precipitate rather than correct the rhythm disturbances. It seems likely, therefore, that at least in the majority of patients with sinus bradycardia there is a primary loss of the automatic depolarizing activity in the sinoatrial node, rather than suppression of the node by abnormal parasympathetic drive.

Kirk and Kvorning (1952) found bradycardia in 3 patients with diseases of the central nervous system, and the patient with bradycardia described by Wedd and Wilson (1930) also had a hemiplegia from a lesion of the internal capsule. Limitation of the normal re-

sponse of the pulse rate to exercise has been described in 2 patients with brainstem lesions by Frick, Härtel, and Punsar (1964) and Frick, Heinonen, and Heikkilä (1966), but, unlike patients with sinus bradycardia, their resting pulse rates were normal. Clinical evidence of nervous system lesions was rare in the patients of the present study. Only 3 had had cerebrovascular accidents, 1 was thought to have some unspecified brain atrophy, and none was known to have had encephalitis. In 3 a diagnosis of epilepsy had been made on account of the recurrent blackouts, though this was only substantiated by electroencephalogram in 1.

It is possible that in sinus bradycardia sinoatrial function is disturbed by a faulty nodal blood supply. Sinus node arrest and bradycardia are well recognized after myocardial infarction (James, 1961; Lippestad and Marton, 1967), but usually these arrhythmias persist for a few days only (Fluck *et al.*, 1967). It seems unlikely that such a mechanism played a part in many of the patients of the present study, since only 4 had any evidence of past cardiac infarction. Rokseth *et al.* (1970) commented that 5 of their 14 patients with bradycardia had had rheumatic fever or diphtheria implying a causal relation. However, in the present study this seems unlikely since these illnesses had occurred in only a minority of the patients. Fowler *et al.* (1969) suggested that the bradycardia might be due to primary degenerative disease affecting the sinoatrial node, and likened the aetiology to that of idiopathic heart block. Certainly, the frequency of possible aetiological features such as a past history of coronary artery disease or rheumatic fever or diphtheria is remarkably similar to that of patients with atrioventricular block. This is illustrated in Table 3 which compares the past illnesses of the patients with sinus bradycardia with those recorded in 100 consecutive patients with 2nd and 3rd degree heart block seen in this department (Shaw and Eraut, 1970). Another similarity is the frequency of fast atrial arrhythmias in the two conditions. Thus paroxysmal atrial tachycardia or atrial flutter or fibrillation occurred in 12 of the 46 patients with bradycardia and 15 of the 100 with heart block. Further, 6 of the 46 patients with sinus bradycardia had evidence of disturbed atrioventricular conduction, and in view of the rarity of the two conditions this association is likely to be significant.

The common finding of broad low voltage P waves in the patients with sinus bradycardia implies that the pathological process, whatever it may be, often involves the atria in addition to the sinoatrial node. The frequency of atrial fibrillation in patients with sinus bradycardia is further evidence of this, since though pathological changes in the sinoatrial node are usual in patients 'dying' in atrial fibrillation (Hudson, 1960), ischaemia or injury to the node alone is inadequate to produce the arrhythmia (James, 1961).

TABLE 3 *Comparison of possible aetiological factors in past history of patients with heart block and those with sinus bradycardia*

Past history	*Heart block (100 patients)* (%)	*Sinus bradycardia (46 patients)* (%)
Cardiac infarction	13	9
Rheumatic fever	11	11
Diphtheria	12	11
Congenital heart disease	2	4
Miscellaneous	1	2
None of these	65	67

From the data presented it is suggested that sinus bradycardia exists as a pathological entity distinct from sinoatrial block and that patients with the condition tend to present the following features: (1) a long history of a slow pulse rate; (2) a tendency to syncopal or dizzy attacks; (3) evidence of failure of sinoatrial node rhythmicity (sinus arrest with junctional or ventricular escape); (4) abnormally broad and low amplitude P waves in the cardiogram; (5) excessive irritability of the atria leading to fast atrial arrhythmias or frequent atrial ectopic beats; and (6) an abnormal response to the stimulus of exercise, atropine, or isoprenaline.

The aetiology remains unproven but the most probable seems to be the loss of the inherent rhythmicity of the sinoatrial node associated with a primary degenerative disease. While awaiting further evidence to establish the mechanism of the condition, the descriptive title of 'the lazy sinus syndrome' is suggested.

We would like to thank the family doctors in the Exeter clinical area for their help and co-operation on which this study depended. We are grateful to Dr. J. A. Cosh of Bath and Dr. M. G. Thorne of Torbay for supplying the details of two of the patients and for the support of other physicians in the Devon clinical area who kindly allowed us to borrow case notes. Part of this work was financed by a grant from the Department of Health and Social Security.

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