Athlete's bradycardia as an embolising disorder?

Symptomatic arrhythmias in patients aged less than 50 years

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SUMMARY One hundred and sixty consecutive patients < 50 years of age (mean 38 years) referred for long term electrocardiographic recording were evaluated retrospectively. Significant cardiac arrhythmias were detected in 51 of 107 (48%) patients examined because of syncope or dizzy spells or both. Of 39 patients examined for cardiac complaints or presumed complex arrhythmias, 15 (38%) had significant arrhythmias. Of 14 patients examined because of otherwise unexplained strokes, nine had slow sinus rates. Of these, one patient had recently undertaken moderately intensive athletic activity and four had been undertaking vigorous athletic activities for several years.

All of the 12 active athletes who were followed up on account of syncope or dizzy spells were free of symptoms after reducing their athletic activities. The cardiac rhythm returned to normal in four out of five who underwent repeat long term electrocardiographic recording.

It is suggested that vigorous athletic activity in subjects of 30-50 years of age may transform the adaptative bradycardia of the athlete into a condition similar to the embolising sick sinus syndrome.

Using long term electrocardiographic recording several investigators have found in patients with suspected Adams-Stokes syndrome high incidences of episodic cardiac arrhythmias capable of causing general ischaemic cerebral attacks with dizzy spells or syncope.¹⁻⁷ Furthermore, focal neurological signs may be caused by cerebral embolisation in patients having permanent or paroxysmal atrial arrhythmias,⁸⁻¹²—that is, atrial fibrillation or flutter and the sick sinus syndrome. Recently, it was found that one out of two patients with non-haemorrhagic stroke had atrial arrhythmias when undergoing long term electrocardiographic recording.¹³

Although most patients with the Adams-Stokes syndrome are elderly, it may also be found in younger patients.¹⁴ Nevertheless, when sinus bradyarrhythmias or even high degree atrioventricular block are found in athletes^{15 16} this is believed to be an adaptation to physical fitness rather than a pathological bradycardia.

During the past few years we have noted that patients with strokes aged < 50 years also have atrial arrhythmias of possible aetiological importance; this is especially true of regular participants in strenuous athletic activities. For this reason we reviewed retrospectively all long term electrocardiographic recordings in patients < 50 years old seen during a three year period. Our aim was to identify the significant arrhythmias in symptomatic patients aged < 50 years with special reference to atrial arrhythmias and neurological symptoms including stroke in subjects undergoing physical training.

Patients and methods

This hospital incorporates the only cardiac unit available to the 235,000 inhabitants of Malmö. During the study period some 1,400 patients were referred for long term electrocardiographic recording each year. The overwhelming majority was suspected of having the Adams-Stokes syndrome, but patients with known complex arrhythmias or atypical cardiac complaints were also evaluated. Patients with otherwise unexplained strokes were increasingly referred for the detection of possible atrial arrhythmias known to cause cerebral embolisation.

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This study included all patients aged < 50 years undergoing long term electrocardiographic recording during the study period. Before recording was carried out examinations were performed to detect any non-cardiac causes of symptoms. For comparison we also studied the case records and routine resting electrocardiograms of all patients aged < 50 years not examined by long term electrocardiographic recording who were admitted to this department during the study period with a final diagnosis of cerebrovascular accident.

LONG TERM ELECTROCARDIOGRAPHIC RECORDING

Using portable electrocardiographs type SRA/HRB-3 (Helcomed Norden/Hellige) all patients were examined for a routine period of about 24 hours. Technically poor recordings were repeated until a satisfactory tracing covering 24 hours was obtained. All recordings were screened by either of two automatic units. Both were programmed to produce a print-out for each arrhythmic event detected. One unit (prototype, Svenska Radio AB) produced printouts for each single RR interval ≤ 0.5 s or ≥ 1.5 s (that is, corresponding to rates ≥ 120 and ≤ 40 beats/min respectively). The other unit (Multiscriptor EK 27, Helcomed Norden/Hellige) produced printouts for each single RR interval 30% shorter or longer than the mean for the last minute of recording. In addition, both units were programmed to produce printouts for every tenth ventricular extrasystole as recognised by QRS configuration criteria and occurring within one minute of recording.

CLASSIFICATION OF ARRHYTHMIAS

Arrhythmias known to or presumed to correlate with cerebral symptoms were termed significant. Minor arrhythmias expected to occur in asymptomatic individuals were considered insignificant and are not included in this report. The criteria for significant arrhythmias used routinely in this department are derived from the findings of 103 elderly subjects selected randomly from the population of Malmö and undergoing long term electrocardiography for approximately 24 hours using the same equipment and screening procedure as in the present study.¹⁷

Significant arrhythmias

Slow sinus rates were identified when one or more of the following criteria were fulfilled: (a) sinus bradycardia ≤ 50 beats/min with a variation of consecutive PP intervals of $\geq 20\%$; (b) regular sinus bradycardia ≤ 45 beats/min when awake and (c) sinoatrial block. The lowest rates were calculated from a sequence of three or more beats of the ordinary rhythm. This screening did not yield printouts for a

regular or almost regular sinus bradycardia above 40 beats/min. Thus all detected sinus bradycardias of 41–50 beats/min had to occur in connection with either an early extrasystole, frequent ventricular extrasystoles, or an RR interval of ≥ 1.5 s. The criteria used for slow sinus rates are identical to those applied by us in the diagnosis of the sick sinus syndrome in symptomatic (and mostly elderly) patients. In elderly patients repeated sinus arrests ≥ 1.5 s also qualify for the diagnosis of the sick sinus syndrome.¹⁷

Atrioventricular block (second and third degree)

Ventricular arrhythmias included frequent ventricular extrasystoles—that is, one ventricular extrasystole followed by a second ventricular extrasystole within no more than five complexes and this second ventricular extrasystole followed by a third ventricular extrasystole within no more than five complexes—and potentially dangerous ventricular extrasystoles according to the Lown grading system.¹⁸ A ventricular tachycardia consisted of three consecutive impulses at a rate of ≥ 100 beats/min.

Atrial tachyarrhythmias included rates ≥ 120 beats/min and lasting > 8 s.

Main arrhythmia category

Patients with significant arrhythmias were categorised according to one main arrhythmia whereas additional significant arrhythmias were analysed separately.

Results

A review of all long term electrocardiographic recordings from the study period yielded 160 patients aged 3 weeks to 49 years. Eighty one were men (mean (SD) age 38 (10) years) and 79 were women (37 (11) years).

PREVIOUS CARDIAC DISEASE

There was no clinical evidence of organic heart disease in 125 patients who all had normal QRS-T electrocardiographic patterns at rest. In 29 of these 125 patients one or more types of significant arrhythmia was previously documented by electrocardiography: 11 had slow sinus rates, eight had atrial tachyarrhythmias, 11 had ventricular arrhythmias, and two had previously had third degree atrioventricular block.

Of the 35 patients with clinically diagnosed heart disease, eight were treated for cardiac failure, 11 had had a definite or suspected myocardial infarction, two had alcoholic cardiopathy, and one had dilated cardiomyopathy, while 13 had other conditions.

SIGNIFICANT ARRHYTHMIAS

Table 1 summarises the main categories of arrhythmia in relation to the type of symptoms which was the

immediate reason for long term electrocardiographic recording. Of the 160 patients, 75 (47%) had one or more types of significant arrhythmia.

Of 55 patients with slow sinus rates, two also had episodes of atrioventricular block (second and third degree) and nine had ventricular arrhythmias. Six patients had bursts of atrial tachyarrhythmias—that is, the bradycardia-tachycardia syndrome.

Table 1 *Relation of symptoms to different categories of arrhythmias detected by long term electrocardiographic monitoring. Figures are number of patients*

	Symptoms		
	Syncopal dizziness (n=107)	Stroke* (n=14)	Cardiac symptoms (n=39)
Significant arrhythmias:			
With	51	9	15
Without	56	5	24
Category of arrhythmias:			
Slow sinus rates	35	9	11
Atrioventricular block (second/third degree)	1	0	0
Ventricular arrhythmia	12	0	4
Atrial fibrillation	3	0	0

*Includes eight patients who also had syncope/dizziness before stroke.

SLOW SINUS RATES

Table 2 shows the range of bradycardias of the 55 patients who had slow sinus rates. All patients with pulse rates <45 beats/min had pronounced sinus arrhythmias except one who had sinoatrial block and frequent ventricular extrasystoles. Of the 10 patients with pulse rates of 46–50 beats/min, seven had sinus bradyarrhythmias during daytime activities. Of the remaining three patients who had regular sinus bradycardias of 46–50 beats/min, sinoatrial block was found in one, episodes of atrial fibrillation in one, and ventricular tachycardias in one.

DRUGS

Fourteen patients with significant arrhythmias were taking some cardiac medication whereas 61 were not.

Table 2 *Range of bradycardias in 55 patients with slow sinus rates*

	Bradycardia rate (beats/min)		
	≤40	41–45	46–50
Total No of patients	21	24	10
Sinus bradyarrhythmias	18	23	7
Sinoatrial block	6	6	3
Atrioventricular block (second/third degree)	1	1	0
Significant ventricular arrhythmias	4	3	2
Atrial fibrillation/flutter	1	2	3

Of the patients with slow sinus rates, four were taking digitalis—two of them in combination with a beta blocker—seven beta blockers, and one verapamil. One patient with atrial tachyarrhythmia and one with ventricular arrhythmia were taking digitalis.

ATHLETIC ACTIVITIES

The records of 26 patients stated that the patient was regularly participating in athletic activities. No such statement was made in the records of the remaining 134 patients. Of the 26 athletic patients, 12 were actively competing or were ex-competitors. Twenty one of these had slow sinus rates and two significant ventricular arrhythmias.

TREATMENT FOR ARRHYTHMIAS

In 21 patients both arrhythmias and symptoms were severe enough to cause withdrawal or institution of cardiac medication. Two patients underwent pacing. Patients undertaking vigorous athletic training who had symptomatic slow sinus rates were urged to reduce the intensity of their physical activities. Twelve patients who did so were available for follow up; all were free of symptoms. Long term electrocardiographic recording was repeated in five of them. In one patient with sinoatrial block and in two with sinus bradyarrhythmias of 38 and 41 beats/min respectively their rhythms had completely returned to normal. One patient who had sinoatrial block at a rate of 38 beats/min later had a sinus bradyarrhythmia of 42 beats/min but without sinoatrial block. One patient with a sinus bradyarrhythmia of 35 beats/min showed a temporary improvement but then developed permanent atrial fibrillation which was resistant to cardioversion.

STROKES

Strokes (all non-haemorrhagic) were experienced by 14 patients aged 29 to 49 years. Of these, nine had slow sinus rates, of whom four (out of 34) had not and five (out of 21) had participated in athletic activity.

Table 3 shows the details of the strokes suffered by five patients with a history of recent physical activity and slow sinus rates. In all instances the onset was sudden and without prodromal signs. In the four patients who underwent angiography there were no abnormalities of the vessels except abrupt occlusions corresponding to the site of neurological symptoms. Hence, all strokes were found to be consistent with cerebral embolism. There was no clinical or echocardiographic evidence of valvar disease of the heart in any of these patients, and none showed arrhythmias on their routine electrocardiograms. Four patients had undergone intensive and vigorous endurance training for several years and one was a jogger who had earlier had a possible embolisation of the retinal

Table 3 Details of stroke in athletes fulfilling criteria for slow sinus rates

Case	Sex/age (yr)	Athletic activity	Stroke	Electrocardiography
1	F/29	Ex-competitive athlete, continued vigorous training	Sudden hemiparesis/hemianaesthesia with incomplete recovery; CT showed non-haemorrhagic infarction; four vessel angiography normal except for abrupt occlusion	Sinus bradyarrhythmia (45 beats/min) during daytime; sinoatrial block
2	M/33	Ordinary jogger	Sudden hemiparesis with complete recovery; CT normal; four vessel angiography normal except for abrupt occlusion	Sinus bradyarrhythmia (45 beats/min) during daytime activity
3	M/38	Ex-competitive athlete, continued vigorous training	Sudden onset of cerebellar symptoms with incomplete recovery; brain scan showed abnormal uptake; four vessel angiography normal except for abrupt occlusion	Sinoatrial block; episodes of atrial fibrillation
4	M/38	Vigorous athletic activity several times weekly	Sudden nausea; Babinski's sign present at left side; complete regression	Sinus bradyarrhythmia (47 beats/min) during daytime activity
5	F/46	Vigorous daily athletic activities	Sudden hemiparesis with incomplete recovery; CT showed non-haemorrhagic infarction; four vessel angiography normal except for abrupt occlusion	Sinus bradyarrhythmia (33 beats/min); sinoatrial block

CT, computed tomography.

artery. Three patients made an incomplete recovery although the remaining symptoms were mild.

Risk factors

One of the five patients with slow sinus rates, stroke, and a history of physical activity was a smoker and one was taking a beta blocker for hypertension. Of the four patients with slow sinus rates and stroke but no history of physical activity, three had an alternative explanation for their strokes; one may have had cerebral sarcoidosis, one had generalised vasculitis, and one had a recent myocardial infarction.

All five stroke patients without atrial arrhythmias had possible causes for the stroke other than an arrhythmia; one was an alcoholic, two had hypertension, one had a previous myocardial infarction, and one may have had cerebral sarcoidosis.

Four of the 21 patients with slow sinus rates and a history of physical activity and two patients without significant arrhythmias during long term electrocardiographic recording had previously had non-cerebral arterial embolisations. One of the latter had a history of earlier vigorous activity.

COMPARATIVE STUDY GROUP

During the study period 36 additional patients were admitted with a final diagnosis of non-haemorrhagic stroke. Twenty three were men aged 25–49 (mean 42 (8)) years and 13 were women aged 26–49 (mean 38 (12)) years. Slow sinus rates were detected in the resting electrocardiograms of four and atrial fibrillation in one. Later another patient developed slow sinus rates when undergoing long term electrocardiographic recording. Thus six (17%) of the patients with non-haemorrhagic stroke who were not studied by long

term electrocardiographic recording had possible atrial arrhythmias consistent with embolism. One of the patients with slow sinus rates and stroke had a history of previous athletic activity.

Discussion

Several studies have shown that severe paroxysmal arrhythmias are common among patients with cerebral symptoms,^{1–7} but the patients studied so far have generally been elderly. Goldreyer *et al* reported a series of middle aged patients who had pronounced haemodynamic deterioration when atrial tachycardia was induced.¹⁹ Recently, Schott *et al* pointed out that cardiac arrhythmias may “masquerade as epilepsy” in young adults.¹⁴ The present study shows that long term electrocardiographic recording may identify arrhythmias in patients < 50 years old with more diffuse cerebral symptoms such as dizziness and syncope.

Cardiac arrhythmias may cause stroke in two different ways. Firstly, a haemodynamic crisis may follow serious arrhythmias, though at necropsy brain infarcts are seldom found in patients who have previously survived a cardiac arrest.²⁰ Secondly, a common group of atrial arrhythmias—for example, the sick sinus syndrome and atrial tachyarrhythmias including fibrillation—may reflect atrial disease, which is also linked with atrial thrombus formation and subsequent systemic embolisation. The risk of stroke is high among patients with atrial fibrillation, even when valvar disease is not present⁹; this arrhythmia may occur in one out of five patients admitted to hospital because of a stroke.²¹ The incidence of demonstrable left atrial

thrombi is also high in postmortem studies of atrial fibrillation.^{8 22 23}

In clinical studies of the sick sinus syndrome the risk of cerebral embolisation is high.^{24–26} During follow up of 254 symptomatic patients with sick sinus syndrome as shown by long term electrocardiographic recording the annual risk of stroke was found to be more than 7% per patient.¹² The criteria and equipment used in this study¹² were identical to those used for slow sinus rates in the present study. Patients with episodes of mild bradycardia (46–50 beats/min) had the same risk of embolisation as patients with severe bradycardias. Among patients aged 20–49 years the annual risk of systemic embolism was still 5% per patient. In 30 patients who died, necropsy showed that 12 had encephalomalacias and 11 had had one or more non-cerebral arterial embolisms. Atrial thrombi were detected in six of the 30 patients. Another recent study has shown that sick sinus syndrome was present in 21 of 86 consecutive patients with non-haemorrhagic stroke when long term electrocardiographic recording was used.¹³

Although the relation between the sick sinus syndrome and cerebral embolism and the Adams-Stokes syndrome is well established, generally accepted electrocardiographic criteria for the disorder are still lacking. Mackintosh defined the symptomatic sick sinus syndrome as “syncope or dizziness suggestive of transient asystole together with demonstrated sinus node dysfunction on a resting electrocardiogram or during ambulatory monitoring.”²⁷ All patients with slow sinus rates in this series were symptomatic. The definition by Mackintosh is probably acceptable to most cardiologists, but, nevertheless, it does not include the initial phase which may be asymptomatic although already linked with embolism. Ferrer widened and popularised the concept of the sick sinus syndrome.²⁸ In her indirect diagnostic criteria she included “the inappropriately slow sinus rate or the relatively slow sinus rate.” She also states that “the sick sinus syndrome often starts with sustained or periodic episodes of slow sinus rate. This may start with sinus bradycardia between 45 and 55/min.”

Nevertheless, even with the widened concept of the sick sinus syndrome its definition by electrocardiographic criteria remains difficult. Our criteria of the sick sinus syndrome used elsewhere¹³ do not include the perfectly regular sinus bradycardia of 41–50 beats/min during night sleep, which is a common and physiological phenomenon. The type of electrocardiography applied may also be crucial. The methods used in this study were that an RR interval ≥ 1.5 s or ≤ 0.5 s or an RR interval variation of 30% was required for the detection of a bradycardia. Frequent ventricular extrasystoles also produced a printout. This means that sinus bradycardias of 41–50 beats/

min remained undetected unless accompanied by a more complex type of arrhythmia.

In the age group of 30–50 years, seven studies have shown that sinus bradyarrhythmias are rare.^{29–35} On the other hand, some studies have shown that slow sinus rates may be more common at and below the age of 30. In the study by Zapfe and Hatano of normal subjects aged about 30 years, 2% had sinoatrial block.³⁶ Engel and Burckhardt noted that one in two subjects (mean age 24 years) had sinus pauses or arrests averaging 1.2 s but that an escape rhythm of normal frequency always emerged within that time.³⁷ Brodsky *et al* studied male medical students, of whom none “was a trained athlete” and found a sinus bradycardia of < 50 beats/min during waking periods in 26% and that 24% had a sinus bradycardia of < 40 beats/min at some time during the 24 hour period.³⁸ Later Sobotka *et al* from the same centre (Chicago), examining female subjects aged < 30 years, found that 8% had sinus bradycardias of < 40 beats/min during sleep and 18% of < 50 beats/min during waking periods.³⁹ Sobotka *et al* explained the higher heart rates among women as a sex difference, but none of their female subjects were allowed to have participated in “formal or informal physical training during the previous year.” Furthermore, when endurance runners were examined by Talan *et al*, at the same Chicago centre, only slightly lower heart rates were found among them than among the male medical students.⁴⁰ This is in contrast to Viitasalo *et al*, who monitored endurance runners and compared them with sedentary control subjects aged < 30 .⁴¹ Although bradycardia was frequently found among the physically active subjects only 6% of the control subjects had sinus pauses of ≥ 2 s, and the lowest heart rates in control subjects during night sleep was 45(6) beats/min. In the study by Mackintosh 10 out of 19 healthy volunteers had sinus rates < 40 beats/min, but again the lowest heart rate “was always during the night.”²⁷ Mackintosh concluded that among young adults a lowest heart rate of 35 beats/min would be a suitable criterion for the sick sinus syndrome.

It is still possible that there is no definite borderline between abnormal and normal sinus rates in young adults. Our criteria for the sick sinus syndrome may in fact be similar to those of Mackintosh since we did not include moderate and perfectly regular sinus bradycardia during night sleep. The various principles for detecting bradycardias with different equipment may also explain some of the different opinions on normal compared with abnormal slow sinus rates. Furthermore, the extent of bradycardia tolerable to an asymptomatic subject may be different from that of a symptomatic patient.

Among our patients with slow sinus rates almost all had a non-physiological bradycardia during activity,

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and 47 out of the 55 patients had a sinus bradyarrhythmia which may be a more serious arrhythmia than regular sinus bradycardia. In addition, two of them had second or third degree atrioventricular block, 15 had sinoatrial block, six had atrial tachyarrhythmias, and nine had significant ventricular arrhythmias. Our finding that athletically active patients may develop symptomatic sinus bradyarrhythmias accords with the report by Rasmussen *et al.*⁴²

Because of the different opinions on the lower limit of the normal sinus rhythm in patients < 50 years of age we also studied eight patients aged < 50 with the pre-excitation syndrome. None had a slow sinus rate according to the criteria currently applied when the same equipment and screening procedure as in this study were used.

Kerr and Strauss recently introduced the measurement of the sinus node refractoriness as a means of diagnosing the sick sinus syndrome.⁴³ Twelve patients were compared with control subjects. All patients had prolonged sinus node effective refractory periods. Of these, eight had only sinus bradycardia and four had more complex types of sinus bradyarrhythmias, two of whom were aged 13 and 33 years. Hence, the method of Strauss and Kerr has shown that various types of sinus bradyarrhythmias may be a pathological finding in young adults.

The sinus bradyarrhythmias induced by physical activity seem to have several features in common with the genuine sick sinus syndrome: a tendency to the Adams-Stokes syndrome, an ability to cause systemic embolism, and a broad spectrum of various but significant arrhythmias including atrial fibrillation. Although athlete's bradycardia may be regarded as an adaptation to physical fitness, it may have the propensity to become a condition similar to the sick sinus syndrome. Our results are therefore consistent with the hypothesis that athlete's bradycardia after vigorous physical training in lower middle age may develop into a condition similar to the symptomatic sick sinus syndrome with a tendency to cerebral embolisation. The reasons for this remain obscure. Nevertheless, it cannot be excluded that athletic activity may cause the premature development of an inherent disorder in susceptible subjects who would anyway have developed it in later life. Further and prospective studies are needed to confirm or reject our hypothesis. Surprisingly, there are no follow up studies of patients with athlete's bradycardia despite the presumed normality of the condition.⁴¹ If it can be confirmed that excessive and vigorous physical activity may trigger an embolising sick sinus syndrome in lower middle age a new cardiovascular risk factor has been found, which possibly reduces the beneficial effects of physical overactivity.

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