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Carotid sinus hypersensitivity in patients presenting with syncope

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SUMMARY In 23 patients (ages 44 to 81) presenting with syncope, vertigo, or transient amnesia, carotid sinus massage produced a significant bradycardia in association with symptoms. The 10 most severely symptomatic patients were studied electrophysiologically, including measurement of intracardiac conduction times and corrected sinus node recovery times, as well as with carotid sinus massage before and after atropine. The only detectable abnormality in five of this group was asystole produced by carotid sinus massage; the other five had, in addition, evidence of either sinuatrial disease or an intracardiac conduction defect. Cardiac pacing in these 10 patients completely abolished their symptoms. In a control group of 52 asymptomatic patients (ages 36 to 87), an abnormal response to carotid sinus massage was uncommon (2%).

Syncope, vertigo, and transient amnesia are common clinical problems and may result from many different conditions. Recently, it has been recognised that in some patients presenting with these symptoms an underlying arrythmia may be the primary cause (Abdon and Malmcrona, 1975). The carotid sinus syndrome is a rare cause of symptoms in association with transient bradycardias (Weiss and Baker, 1933; Ferris *et al.*, 1935).

In patients presenting with idiopathic syncope, carotid sinus massage may induce severe bradycardia. We report on a study of such a group of patients, in whom intracardiac electrophysiological tests were performed during carotid sinus massage.

Patients and methods

(1) CONTROL GROUP

The response of heart rate to carotid sinus massage was assessed in a control group of 52 men in hospital (ages 36 to 87). They were in sinus rhythm, with no evidence of cardiac or neurological disease and had normal resting electrocardiograms. None had suffered from syncope or disturbance of consciousness or other symptoms related to the central nervous or cardiovascular systems.

(2) SYMPTOMATIC GROUP

Over an 18-month period, 23 patients (15 men, eight women) referred for evaluation of syncope, near syncope, or transient amnesia (Table 1) had an abnormal response to carotid sinus massage, and they have been followed up over a period of 18 months to three years. All patients had a full history taken, clinical examination, and resting electrocardiogram. Carotid sinus massage was performed by the same observer in all cases, with the patient in a semirecumbent position of 45 degrees. Continuous electrocardiographic monitoring was carried out while the carotid sinus was massaged for five seconds unless asystole with symptoms intervened. All drug treatment had been withdrawn for at least 48 hours before this manoeuvre. None had been receiving digitalis or beta-blocking agents before study. An abnormal response was considered to be present if asystole occurred for more than three seconds (Thormann and Schwartz, 1975; Scarpa, 1976).

Intraventricular conduction disturbances were diagnosed from the electrocardiogram, using the criteria of Rosenbaum (1970). Ten severely symptomatic patients were submitted to electrophysiological studies which included measurement of intraventricular conduction times and measurement of corrected sinus node recovery times after atrial pacing at various rates (Narula *et al.*, 1972). Carotid sinus massage was also performed and all measurements were then repeated after the intravenous administration of 1·2 mg atropine.

Results

(1) CONTROL GROUP

In these 52 patients carotid sinus massage produced slowing of the sinus rate. The maximal RR

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Table 1 *Patients with abnormal response to carotid sinus massage; cases 1 to 10 studied electrophysiologically and paced permanently*

Case no.	Age (y)	Symptoms	Associated diseases	Electrocardiogram
1	67	Syncope 1 month	Hypertension	Normal
2	73	Syncope 6 months	Nil	Sinus bradycardia/ SA block
3	49	Syncope/amnesia 8 years	Coronary artery disease	Normal
4	52	Syncope/amnesia 3 months	Coronary artery disease	Normal
5	55	Syncope 2 years	Hypertension	Left axis
6	64	Syncope/light-headedness 6 months	Coronary artery disease	Normal
7	69	Syncope/amnesia 2 years	Nil	Normal
8	66	Syncope 1 year	Coronary artery disease; diabetes	Intermittent lt axis SA block
9	71	Amnesia 2 years	Nil	Intermittent LBBB
10	66	Amnesia 10 years	Hypertension	Left axis
11	60	Syncope 30 years	Hypertension	Left axis
12	54	Syncope 18 months	Coronary artery disease	Normal
13	47	Light-headedness	Nil	Normal
14	64	Amnesic episodes 1 year	Nil	Normal
15	67	Syncope/vertigo 1 year	Nil	Normal
16	67	Vertigo	Hypertension	Normal
17	66	Vertigo	Coronary artery disease	Intermittent RBBB
18	82	Syncope 2 years	Hypertension	Normal
19	44	Syncope/vertigo 3 years	Nil	RAD
20	63	Syncope 1 year	Cerebrovascular disease	Intermittent atrial fib
21	74	Syncope 6 months	Cerebrovascular disease	LBBB
22	73	Syncope	Nil	Normal
23	72	Syncope 3 years	Coronary artery disease	Left axis

prolongation obtained, expressed as a percentage of the resting RR interval, was 127 ±30 per cent. In one patient, asystole of 4·6 seconds was produced on one occasion only.

(2) SYMPTOMATIC GROUP

In the 23 symptomatic patients (Table 1), carotid sinus massage produced asystole lasting longer than three seconds in association with cerebral symptoms. The standard electrocardiograms were normal in 12 patients; in the remaining 11, two showed periods of sinuatrial block: one of these patients also had intermittent atrial fibrillation and one had persistent left bundle-branch block.

Electrophysiological investigation of 10 patients (cases 1 to 10, Table 1) showed that five patients with normal resting electrocardiograms had normal sinus node function with conduction times, the only abnormality being an abnormal response to carotid sinus massage which was totally abolished by intravenous atropine.

The two patients (cases 2 and 8) with electrocardiographic signs of sinuatrial block showed evidence of gross sinuatrial node dysfunction with post-pacing asystole for 3 and 5·3 seconds followed by a slow junctional escape rhythm. After intravenous atropine, the junctional escape rhythm was preceded by asystole for 2 and 4·9 seconds, but their abnormal response to carotid sinus massage was totally abolished. Three cases with left axis deviation had prolongation of HV intervals (Table 2).

The 10 patients who underwent electrophysiological tests had cardiac pacemakers implanted; on follow-up (18 months to three years) their symptoms have completely disappeared. The remaining 13 patients (cases 11 to 23), whose symptoms were

Table 2 *Results during electrophysiological study: intervals in milliseconds*

	Before atropine				After atropine			
Case no.	RR	HV	CSNRT	CSM rr	RR	HV	CSNRT	CSM rr
1	850	50	10	4700	720	50	−5	810
2	950	50	5300 JER	9330	660	50	4900 JER	660
3	930	45	180	7950	850	45	160	1130
4	990	55	0	3460	610	55	50	610
5	1005	60	150	3050	910	60	130	920
6	950	55	320	4260	890	55	300	950
7	910	45	35	7850	850	45	55	850
8	1020	35	3050 JER	3100	810	50	2030 JER	820
9	830	75	270	3490	590	75	205	590
10	770	70	50	3150	620	75	10	640

HV, conduction time His-Purkinje system (normal 35 to 55 ms); CSNRT, corrected sinus node recovery time (normal < 525 ms); CSM, carotid sinus massage (normal < 3000 ms); JER, junctional escape rhythm.

mild and infrequent, did not receive pacemakers and their symptoms persisted.

Discussion

Hypersensitivity of the carotid sinus reflex has long been recognised to be associated with spontaneous syncope. The carotid sinus syndrome is a term reserved for symptomatic patients who have an abnormal response to carotid sinus massage (Weiss and Baker, 1933; Ferris *et al.*, 1935). Several investigators have shown that, in an asymptomatic population, a significant number of people will show severe bradycardia on carotid sinus massage (Purks, 1939; Heidorn and McNamara, 1956; Sigler, 1963). The prevalence of an abnormal response appears to vary according to the duration of the pressure of stimulus applied to the carotid sinus. Thus, in 25 per cent of normal subjects, massage for 30 seconds produced asystole, with or without symptoms (Heidorn and McNamara, 1956).

Massage for 12 seconds in patients with ischaemic heart disease often produced severe bradycardia (Purks, 1939). Because of this high incidence, we have used a shorter period of massage, believing this to be perhaps more akin to the clinical problem and knowing that prolonged massage may be hazardous (Goldenberger, 1963). It is impossible to get a truly standardised stimulus. However, carotid sinus massage was performed by one person only and, using this method, an abnormal response in the control group of patients in hospital was rare (2%). The 23 patients gathered from a general medical outpatient clinic complained of syncope, transient amnesia, light headedness, or vertigo. Several patients had been severely symptomatic for many years and five had been misdiagnosed as having epilepsy. Six patients volunteered that they had suffered from transient attacks of amnesia: case 7 could not remember playing a golf shot, and case 3, a garage mechanic, was frequently unable to recall what he had done after servicing cars in the garage pit. The other patients presented with syncope and less specific complaints, such as dizziness, light headedness, or vertigo. In cases 3 and 7, rotation or extension of the neck invariably produced symptomatic bradycardia.

In case 2, symptomatic bradycardia could be produced repeatedly when swallowing a glass of cold water. In other patients, such a clear association was not seen. However, carotid sinus massage reproduced symptomatic bradycardia in all patients and a high degree of vagal tone or responsiveness may have been responsible for their spontaneous symptoms.

Monitoring was performed in three ambulant patients, but episodic profound sinus bradycardia was observed in all cases. In the two patients (cases 2 and 8) who showed both carotid hypersensitivity and impaired sinus node automaticity it was possible to show that these were entirely separate phenomena by the use of atropine, though in the clinical situation these abnormalities may well have had an additive effect in the production of symptomatic bradycardia. It can be argued that by only estimating sinus node recovery time we are not excluding the presence of sinus node disease in the other eight patients studied (Gupta *et al.*, 1974) and that estimation of sinuatrial conduction time, as described by Strauss *et al.* (1973) or Narula *et al.* (1978), would have led to the identification of lesser degrees of sinus node disease. We do not believe that this is so or even relevant in patients with such profound symptomatic bradycardia related to excessive vagal responsiveness.

The estimation of sinuatrial recovery and conduction by electrophysiological methods has limited value, and we agree with Evans *et al.* (1978) that from the practical point of view the diagnosis of sick sinus syndrome is best made from the resting electrocardiogram together with ambulatory monitoring.

Before the widespread use of cardiac pacemakers for the treatment of bradycardia, the carotid sinus syndrome was treated by drugs such as atropine, amphetamines, ephedrine, or by irradiation or denervation of the carotid sinus (Thomas, 1969). Clearly, the most reliable method of treatment in such patients should be cardiac pacing. Until recently there have been isolated reports concerning the role of pacing in this condition.

More recent studies have reported the effect of pacing on carotid hypersensitivity often in association with episodes of complete atrioventricular dissociation (Voss and Magnin, 1970; Bahl *et al.*, 1971; Peretz *et al.*, 1973; Walter *et al.*, 1978).

In conclusion, our findings show that hypersensitivity of the carotid sinus reflex is present in a group of patients presenting with transient cerebral disturbances. There may, in addition, be intraventricular conduction disturbance or sinus node disease. However, some patients with severe symptoms may have a grossly abnormal response to carotid sinus massage in the presence of normal electrophysiology. Increased vagal tone or responsiveness as shown by our group of patients seems to be a relatively common mechanism in the production of symptoms in patients presenting to a general medical clinic. Further study is needed to determine the true prevalence of syncope in association with carotid sinus hypersensitivity. The

technique of carotid sinus massage, as described, is simple and safe; we consider it worth while to include this procedure in the routine examination of patients who have a history of syncope.

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