du diagnostic précoce et du traitement chirurgical de la cryptorchidie et de corriger certaines idées fausses.

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Permanent demand pacing for hypersensitive carotid sinus syndrome

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Summary: Ten patients with proved hypersensitivity of one or both carotid sinuses and with symptoms of recurrent lightheaded spells and syncope had implanted a permanent transvenous demand pacemaker. In a follow-up course ranging from 6 to 55 months there has been no recurrence of lightheadedness or syncope in any of the patients. Six of the ten have had their battery packs replaced owing to routine battery exhaustion.

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The carotid sinus is a major vagal centre for regulation of heart rate and peripheral vascular tone. In normal humans, stimulation of the carotid sinus area at the bifurcation of the internal and external carotid arteries produces a mild slowing of heart rate and insignificant fall in blood pressure.[1,2] In some individuals, more commonly in the elderly, there is a hypersensitivity of the carotid sinus reflex so that stimulation by pressure over the carotid sinus or increase in pressure within the carotid sinus will cause a marked slowing of cardiac rate, or even asystole, with lightheadedness or syncope.

Hypersensitivity of the carotid sinus with hemodynamically significant bradycardia or syncope is not as uncommon as one would suspect from the small number of cases reported in the literature over the years. The carotid sinus reflex, hypersensitivity of the carotid sinus and carotid sinus syncope have been well described and little can be added to the description provided in 1933 by Weiss and Baker.[1] The syndrome consists of sudden episodes of fainting or lightheadedness which occur spontaneously, often in circumstances recognized by the patient, such as while looking upward or stroking the side of the neck, and the whole symptom complex is often reproducible by carotid sinus massage. The symptomatic episodes are usually extremely short, measured in seconds, and are only occasionally preceded by an aura which may be visual, auditory, vertiginous or hallucinatory. In our experience, seizure phenomena have not been displayed. The patients rarely have symptoms unless in the vertical posture, contrary to idiopathic Stokes-Adams syncope and epileptic attacks.

Recovery from the syncope is usually extremely prompt and there is no post-ictal phase. Diagnosis is dependent on reproduction or partial reproduction of the symptoms by carotid sinus massage, often with the patient sitting in a chair, and simultaneous electrocardiographic evidence of asystole or marked bradycardia. In our patients we have reproduced syncope in only three cases, whereas in the other seven there was asystole in excess of five seconds or a marked sinus bradycardia with a feeling of "faintness". In our opinion reproduction of the actual syncope is not necessary provided the clinical story is present and hypersensitivity of the carotid sinus vagal output as described can be demonstrated.

The descriptions of the treatment of carotid sinus syncope in the past, including a detailed and exhaustive review of the subject in 1970,[3] allude to surgical denervation of the carotid sinus area, radiotherapy, parasympatholytic agents and sympathomimetic agents, but there was no mention in the literature up to that time of cardiac pacing on demand to prevent the syncopal attacks associated with hypersensitivity of the cardioinhibitory reflex. In the last year there have been two single case reports[4,5] on the treatment of carotid sinus syncope with demand transvenous cardiac pacing, and it was listed as an indication for demand pacing by one of the authors in 1971.[6]

Materials and methods

Ten patients without demonstrable

episodes of atrioventricular block were seen for episodes of lightheadedness and/or syncope in an office and consultative cardiological practice between January 1968 and August 1972. All were found to have hypersensitivity of their right and/or left carotid sinus as the cause of their symptoms. Nine were in sinus rhythm, one having a wandering pacemaker with frequent sinus arrest. Nine were males, the age range for the group being 47 to 88 years, with an average age of 64 years. They all complained of recurrent episodes of lightheadedness and/or one or more episodes of syncope. During the initial examination auscultation of the heart was carried out in order to determine cardiac rate during carotid sinus stimulation. If the patient went into bradycardia or asystole, then this was reconfirmed with the patient supine and attached to an electrocardiograph. Palpation and auscultation of each carotid artery were done in order to detect any obstruction. The bifurcation of the carotid artery was palpated to determine whether or not it was prominent. The right and then the left carotid sinus areas were compressed against the transverse process of the adjacent vertebrae with the head turned to the contralateral side and the examiner's other hand palpating the ipsilateral superficial temporal artery. Pressure was placed upon the carotid sinus firmly but without occluding the pulsation in the ipsilateral superficial temporal artery. If marked bradycardia resulted (less than 30 per minute) massage was maintained with the patient first lying and then sitting to determine if symptoms would become manifest (Fig. 1). If asystole of over five seconds' duration developed during massage, then pressure was removed and the patient asked to cough; if syncope occurred during the asystole, the patient was struck on the chest (Fig. 2).

If bradycardia was the sole cardiac arrhythmia produced by carotid sinus massage we demanded reproduction of the presenting complaint before accepting the patient as a case of "hypersensitive carotid sinus syndrome". If asystole of over five seconds was induced by light carotid sinus massage we did not require reproduction of syncope.

Atropine in sufficient dosage should abort the sensitivity of the carotid sinus. The patients were all given 0.6 mg. atropine sulfate intravenously at five-minute intervals up to a maximum of 1.8 mg. or until their sinus rate increased to more than 100 per minute. In each of our cases this prevented the appearance of significant bradycardia or asystole.

Results

Table I summarizes the important data from these patients. Nine were in sinus rhythm, one having a wandering pacemaker and frequent episodes of sinus arrest, with his usual rate often as low as 30 per minute. This patient apparently has the "sick sinus syndrome" but none the less, vagal stimulation with carotid sinus massage produced asystole consistently. One patient had sustained an inferior myocardial infarction in the remote past, and another had a left bundle branch block, with no history of previous myocardial disease. A third patient had a rate-dependent left bundle branch block which appeared at rates of less than 40 per minute, with no aberrant conduction when his rate was more than 40 per minute. In this patient (H.M.) the right carotid sinus alone was sensitive, and upon stroking it his rate would decrease to 20 per minute and a left bundle branch block would immediately develop with a fall in blood pressure, increase in premature ventricular contractions and syncope. Coughing would initiate exactly the same response. In a fourth patient (H.McR.) a

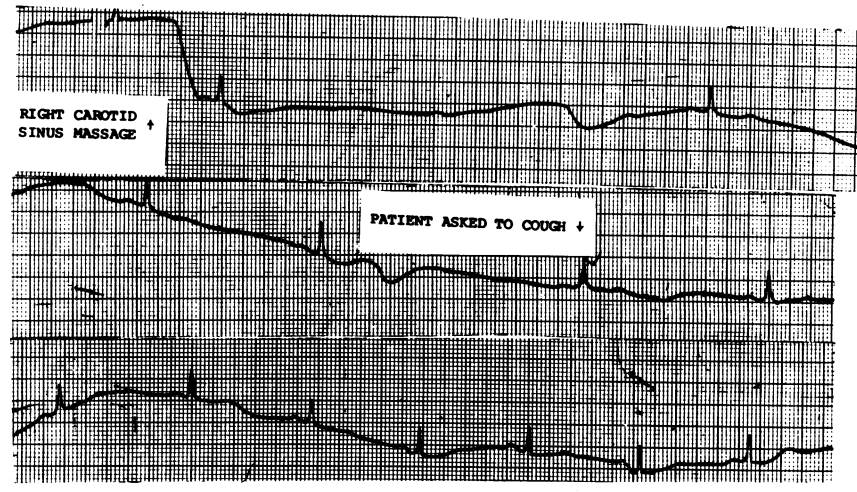

FIG. 1—Slowing of cardiac rate due to carotid sinus hypersensitivity.

Table I
Data of 10 patients with hypersensitive carotid sinus syndrome

Patient	Age	Sex	Pacemaker inserted	Symptoms	Carotid sinus sensitivity
S.P.	75	Male	January 1968	Recurrent lightheadedness and syncope, 3 yrs.	R
H.M.	47	Male	June 1969	Syncope x 1	R
J. McD.	63	Male	September 1969	Fainted 9 times	R = L
J.P.	54	Male	October 1969	Dizziness and syncope, 2 yrs.	L > R
H.H.	66	Male	March 1970	Syncope x 6	R = L
J.N.	61	Male	November 1970	Lightheadedness 1 yr., fainted x 2	R > L
W.G.	88	Male	March 1971	Lightheadedness several times weekly	R = L
H.McR.	65	Male	March 1971	Fainted many times after coughing	R > L
R.M.	54	Male	October 1971	Wandering pacemaker, sinus arrest, syncope	R > L
G.R.	67	Female	February 1972	Syncope x 7, many lightheaded episodes	R

marked sinus bradycardia of 28 per minute was induced by massage over the right or left carotid sinus, and by coughing several episodes of documented asystole lasting up to nine seconds were initiated.

The right carotid sinus was sensitive in all cases, the right being more sensitive than the left in three, equal in sensitivity to the left in three, and the only sensitive sinus in three. In one case only was the left carotid sinus more sensitive than the right on initial examination. One patient (H.H.) was in congestive cardiac failure and was initially seen while taking digitalis. Upon discontinuation of his cardiac glycosides hypersensitivity of the carotid sinus could no longer be elicited. He, however, required digitalis to remain free of congestive cardiac failure, and subsequently had a permanent transvenous demand pacemaker inserted and digitalis once again added to his therapeutic regimen.

All 10 patients had permanent transvenous endocardial pacemakers inserted in the demand mode at approximately 62 electronic impulses per minute (Fig. 3). All are alive and none have had any recurrence of lightheadedness or syncope since insertion of the pacemaker. These results are very much superior to those reported in the literature from any other form of therapy. There was no mortality and no significant morbidity in any patient due to implantation of the pacemaker. One patient had a thrombosis of the axillary vein following insertion, but this cleared with anticoagulant and anti-inflammatory therapy.

Discussion

The hypersensitivity reaction to external or internal pressure upon the carotid sinus is customarily classified into:

1. Cardioinhibitory reflex characterized by a sharp drop in blood pressure associated with asystole or marked bradycardia. This reflex is inhibited with atropine.
2. Depressor or vasomotor reflex characterized by a sharp drop in blood pressure but no slowing of the heart rate. This reflex is inhibited with epinephrine.
3. "Cerebral" reflex with loss of consciousness but no change in blood pressure or pulse rate.

Drugs are said to have no effect on the third or cerebral type of reflex. Many doubt its existence and there is evidence that it is probably a result of occlusion of cerebral flow rather than a carotid sinus reflex.[3,7] In our experience we have only seen patients with the cardioinhibitory type of syndrome.

Certain drugs are known to increase the vagal tone and may unmask hypersensitivity of the carotid sinus. Of particular importance in this respect are cardiac glycosides, morphine and meperidine. One of our patients demonstrated this with digitalis.

The "sick sinus syndrome" may present in a fashion similar to the hypersensitive carotid sinus syndrome but the manifestations of wandering pacemaker with changing configuration of the P waves and length of PR interval, junctional escape beats and sinus arrest are apparent.

It is interesting to note that in two recent papers[2,3] the statement that "milder forms of the disease respond to reassurance and drug therapy" is, in our opinion, a dangerous concept. Presumably by "milder forms" is meant infrequent syncopal attacks and those which last but a short period of time. However, in everyday life, many potentially extremely serious consequences could occur from even a few seconds' loss of consciousness, particularly when driving an automobile or working in certain situations. For this reason and because of the low risk of implantation of permanent transvenous demand pacing units,[6] it is our opinion that all patients with hypersensitive carotid sinuses who have demonstrated episodes of lightheadedness or syncope should have such a pacemaker implanted.

We have found that reproducibility of hypersensitivity of the carotid sinus is not consistent. On several occasions a drop in cardiac rate produced by initial stimulation and documented by auscultation could not be reconfirmed a few minutes later by the time the patient had been connected to an electrocardiograph. Occasionally several days elapsed with the patient on the cardiac monitor and carotid sinus massage performed several times daily, before hypersensitivity could be reconfirmed. One would suspect, therefore, that a number of patients with carotid sinus hypersensitivity will escape detection because they fail to show the cardioinhibitory response on the initial testing. If the clinical story is suggestive and no other etiological possibilities such as carotid or vertebral artery insufficiency, or primary cerebral disease is found, then repeated examination over several days is needed before the examiner can be satisfied that carotid

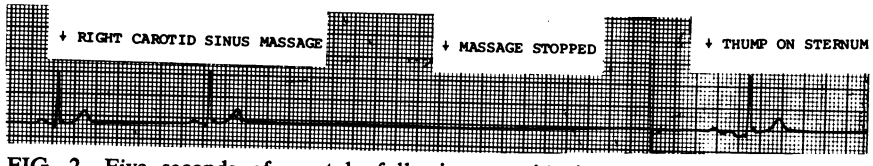

FIG. 2—Five seconds of asystole following carotid sinus massage with response to striking sternum. The patient had lost consciousness.

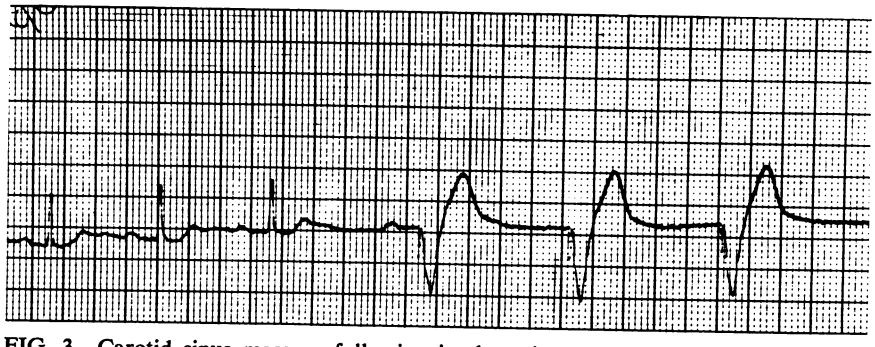

FIG. 3—Carotid sinus massage following implantation of permanent demand pacemaker; electronic pacemaker can cut in, aborting syncope or lightheaded episodes.

sinus hypersensitivity does not exist.

Résumé

Stimulateur cardiaque automatique à demeure pour les cas de syndrome du sinus carotidien

Chez dix malades souffrant d'hyperréflectivité de l'un ou des deux sinus carotidiens et présentant des symptômes de vertige et de syncope, on a implanté par voie intraveineuse un stimulateur cardiaque à demeure opérant automatiquement. L'évolution a été suivie pendant une période variant de 6 à 55 mois. On n'a noté chez aucun malade de récidive des étourdissements ou des syncopes. Chez six des 10 malades, il a fallu, à un moment donné, remplacer la pile épuisée.

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