A modified technique to locate the interscalene groove for brachial plexus anaesthesia is described. Palpation of the groove is easier when the muscles are firm. During a slow deep inspiration the scalene muscles contract, facilitating location of the groove and successful injection of local anesthetic.

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Sick-sinus Syndrome Manifested during Anesthesia

Margaret G. Pratila, M.D.,* and Vasilios Pratilas, M.D.†

Sick-sinus syndrome may be defined as an unexplained and marked sinus bradycardia or sinus arrest with or without associated supraventricular arrhythmias. The following is a case of sick-sinus syndrome first manifested following induction of anesthesia.

Report of a Case

A 59-year-old woman was admitted with a chief complaint of cold lower extremities. The patient had originally complained of "stiff" calves for three years; this had progressed to intermittent claudication.

Past medical history included hypertension for 12 years, treated with hydralazine HCl, 25 mg, orally, twice daily. Her only previous operation was a hemorrhoidectomy 13 years previously.

On physical examination, the patient weighed 66 kg; height was 165 cm. Cardiovascular findings were a grade II/VI systolic ejection murmur at the left sternal border. Pulse rates ranged from 96 to 60/min. Blood pressure was 210/120 mm Hg. Femoral, popliteal and tibial pulses were absent. Auscultation of the chest revealed bilateral rhonchi. Laboratory findings were within normal limits. EKG showed left ventricular hypertrophy and strain, and left atrial hypertrophy. Chest x-ray disclosed no abnormality.

Aortography was performed using local anesthesia and showed Leriche syndrome with complete occlusion of the origin of the right common iliac artery and the left common iliac artery at its bifurcation. The remaining vessels in the lower limbs were extremely atherosclerotic, the left more than the right. Both renal arteries were atherosclerotic at their origins. The abdominal aorta was hypoplastic. Stenosis was severe at the origin of the left vertebral artery and moderate at the origins of both internal carotid arteries.

An aorto-bifemoral bypass was scheduled. Premedication consisted of atropine 0.4 mg, meperidine, 50 mg, and secobarbital, 100 mg, im, 1½ hours before operation. Anesthesia was induced with thiopental 250, mg, and succinyldicholine, 80 mg, iv, followed by orotracheal intubation without difficulty. Maintenance was with enflurane (1–1.5 per cent), N_2O (3 l/min), O_2 (3 l/min), and pancuronium (total dose 9 mg). Following induction, the pulse rate slowed from 84 to 52/min, and atropine, 0.4 mg, given twice, resulted in a short-lived improvement to 64/min, with a decline to 40–42/min (see fig. 1). Atropine to a total of 2 mg, iv, was given, without response. The blood pressure was well maintained. Arterial blood-gas values obtained at this time were: P_{O_2} 152 mm Hg, P_{CO_2} 34 mm Hg, *p*H 7.46, Hb_{O_2} 98.8 per cent. Serum potassium was 4.3 mEq/l.

A temporary demand pacemaker was inserted via the right subclavian vein. The heart rate was maintained at 72–75/min with continuous pacing. Anesthesia and operation for the remainder of the 8 hours were uneventful except for a short period (5–10 min) of severe hypotension following transection of an aberrant renal vein and a period of decreased urinary output, treated with furosemide, 20 mg, iv.

* Associate Attending Anesthesiologist, Beth Israel Medical Center, and Clinical Associate, Mount Sinai School of Medicine, City University of New York, New York, New York.

† Assistant Clinical Professor, Department of Anesthesiology, Mount Sinai School of Medicine, City University of New York, New York, New York.

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Address reprint requests to Margaret G. Pratila, M.D., 145 East 15th Street, New York, New York 10003.

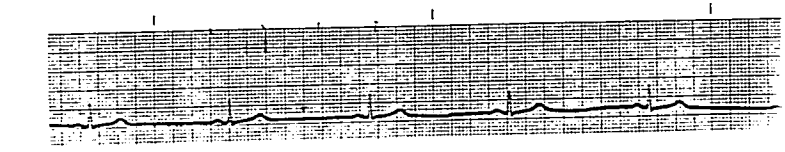

FIG. 1. ECG showing bradycardia at a rate of 40/min.

At the end of operation the patient was transferred to the recovery room with the endotracheal tube in place and connected to a T-piece delivering 40 per cent O_2 and a cardiac monitor. Blood pressure was stable at 160/100 mm Hg, paced rate 72/min. Extubation was performed after 2½ hours and a face mask with a F_{IO_2} of 0.40 applied. At this time, V_T was 350–450 ml, pacing rate 76/min, P_{O_2} 120 mm Hg, P_{CO_2} 35 mm Hg, *p*H 7.48, Hb_{O_2} 98.5 per cent.

The pacemaker was removed on the second postoperative day, pacing not being evident after the first postoperative day, and the patient transferred from the intensive care unit to the floor. No bradycardic episode was seen after pacemaker removal, and the pulse rate remained 80–90/min, with regular rhythm. EKG's were unchanged except for an occasional ventricular premature contraction in the first postoperative day.

The postoperative course was uncomplicated, and the patient was discharged on the fourteenth postoperative day.

DISCUSSION

The name "sick-sinus syndrome" was first used by Ferrer[1] in 1968. Much has since been published on the various clinical entities making up this syndrome. Although almost 70 years have passed since Flack and Keith first identified the sinoatrial node,[2] knowledge of its function is still incomplete. Recent work has clarified its exact location, its size, and its blood supply via a single artery, a branch of the right coronary artery in 55 per cent of the population and the left circumflex artery in 45 per cent,[3] which makes it very vulnerable to vascular damage.[4] Its structure consists of pacemaker, transitional and working myocardial cells.[5] Since more than one pacemaker cell exists, major changes in sinus rate are the result of suppression of an area and dominance of another, and not an alteration of rate or rhythmicity of a single cell.[6]

Diseases affecting the sinoatrial node have assumed increasing importance, and those affecting the pacemaker have been grouped together as the sick-sinus syndrome. The syndrome occurs in all ages and probably is responsible for a certain number of sudden deaths occurring in children and young athletes.[7] Recognition has caused reconsideration of the belief that sinus bradycardia is a benign condition.

The mechanism of bradycardia in sick-sinus syndrome may be either disordered impulse generation within the sinus node or impaired conduction of impulses from the sinus node into the atrium. Underlying conditions include coronary-artery disease, hypertensive heart disease, rheumatic heart disease, congenital heart disease, cardiomyopathies, luetic aortic insufficiency, diphtheria, pericarditis, Friedreich's ataxia, progressive muscular dystrophy, metastatic disease,[8] amyloidosis, hemochromatosis, and an idiopathic isolated fibrotic local lesion. A familial sinus node disease has also been described.[9]

The commonest associated ECG abnormalities are 1-degree A-V block and left axis deviation.[10] The cardiogram of our patient on admission showed left ventricular hypertrophy, left ventricular strain, and left atrial hypertrophy.

Pharmacologic treatment of sick-sinus syndrome has been usually unsuccessful.[11] Atropine is ineffective and has produced tachyarrhythmias on occasion. This was true in our patient, in whom a 2-mg total dose, iv, failed to produce cardiac acceleration. Beta-adrenergic receptor agonists have also proved ineffective, as a rule, and although we considered isoproterenol infusion, it was not used for this reason, and because of the possibility that serious ventricular arrhythmias or hypotension[12] might occur, especially in combination with enflurane anesthesia. An additional factor in our choice of treatment was the prolonged period that would be necessary for operation. Of interest is that although slow heart rates

are known to promote ventricular ectopic activity, this is not common in sick-sinus syndrome.

The sick-sinus syndrome in its chronic form runs an erratic course, with periods of normal sinoatrial node function and periods of abnormal function. Our knowledge of the course and prognosis of the disease is incomplete, but it is believed that 5 to 10 years elapse from onset to complete sinus arrest. If episodes of sinus bradycardia are frequent or symptoms such as dizzy spells, unexplained congestive heart failure, syncope, or cardiac arrest occur, permanent pacing should be instituted.[13]

Effects of Anesthetics on the Mechanism of the Sinoatrial Node

The rate of firing of an automatic cell depends on three factors: the slope of phase-4 depolarization, the level of the threshold potential, and the maximum diastolic potential. Decreases in the first two and/or an increase in the last factor will slow the rate.

Anesthetic agents affect the sinoatrial node by their effect on the rate of phase-4 depolarization or the threshold potential of the cells. Decreasing the rate favors emergence of pacemaker function in lower automatic cells unless they are similarly affected. Similarly, factors that tend selectively to increase the rate of spontaneous phase-4 depolarization, with the upper cells maintaining the same rate, also favor the appearance of extrasystoles generated by lower pacemaker activity.

Studies of electrophysiologic effects of anesthetic agents have elucidated some points, but have raised many questions. Davis *et al.*[15–16] investigated the effects of cyclopropane on the Purkinje fibers. Cyclopropane enhanced the slope of phase-4 depolarization and potentiated the increased slope and magnitude of phase-4 depolarization produced by epinephrine. Arrhythmias were common. Propranolol antagonized this potentiation. The effects of cyclopropane were markedly influenced by the concentration of calcium in the medium.[17] Reynolds *et al.*[18] investigated the effects of halothane and methoxyflurane on pacemaker fibers. The two agents had some very similar and some very different effects. Halothane had a negative chronotropic action on the sinoatrial node that was not prevented by atropine. Methoxyflurane had a biphasic effect, with an initial brief acceleration preceding the negative chronotropic action. Both compounds cause complete cessation of electrical activity of nodal fibers *in vitro*. This may occur with low concentrations of methoxyflurane. It was not preceded by a progressive slowing, but was associated with a marked loss of maximum diastolic potential, increase in threshold potential, and, at the end, loss of excitability of the fiber when there was only a slight reduction in rate. Halothane antagonized the positive chronotropic action of epinephrine, while methoxyflurane had little effect. The major difference between the effects of the two on Purkinje fibers was that halothane depressed phase-4 depolarization and antagonized the stimulatory action of epinephrine and ouabain, actions characteristic of an antiarrhythmic agent, while methoxyflurane increased the rate of phase-4 depolarization and potentiated the action of epinephrine. In this it resembles cyclopropane.

Krishna *et al.*[19] investigated the action of diethyl ether on the atrium. They found it had a direct positive chronotropic action on isolated atria independent of central innervation. Their results indicated this positive chronotropic effect was not mediated via catecholamine release, by direct beta-adrenergic receptor stimulation, or by cholinergic block. They concluded that diethyl ether has a direct stimulant effect on rat atrial pacemakers.

Heart rate appears to be stable during enflurane anesthesia. This is attributed to the maintenance of normal cardiovascular response to variations in carbon dioxide.[20] McDowell *et al.*[21] have shown that the heart rate remains constant with increasing concentrations of enflurane to the point of cardiac arrest. During periods of induced hypoxia during enflurane anesthesia in dogs, heart rate was reduced without added arrhythmias. The normal tachycardia seen during hypoxia is believed to be due to a reflex stimulation from lungs to heart mediated by the vagus. With enflurane, either a marked vagotonic effect or a sympatholytic effect overrides the response of the heart to hypoxemia.[22]

In summary, management of a patient with sick-sinus syndrome has been described. The

effects of general anesthetics on sinoatrial nodal mechanisms have been reviewed.

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