The correct diagnosis with a good definition of the tumor limits is thus mandatory. Careful study of the clinical picture may be of considerable help in this respect. The symptoms indeed depend upon the level and extension of the mass. The different clinical symptoms are generally related to progressive occlusion of the inferior vena cava followed by development of collateral circulation.

In previous reports, when the tumor was located in the upper third of the inferior vena cava, Chiari's syndrome was observed with hepatomegaly, ascites and jaundice. The origin of the hepatic vein was invaded by the tumor or occluded by antemortem thrombosis. Hepatic failure was sometimes more dramatic when the occlusion of the hepatic vein was abrupt. Tumors located in the middle third of the inferior vena cava give rise to a nephrotic syndrome and renal failure.

Leiomyosarcoma located in the lower third of the inferior vena cava are extremely difficult to diagnose. Very often, they extend to the peritoneum with no obstruction in the vena cava, but produce severe lumbar pain. It is only when the tumor grows inside the vessel that swelling of the legs may appear with a palpable abdominal mass.

Cavography seems to be the most suitable method to reach the diagnosis and to obtain information as regards the tumor extent and location. This technique was used in two previous instances by Roussak and Heppleston and Deutsch and colleagues. In our patient inferior cavography revealed a defect of filling with many anastomotic vessels. The superior cavography showed extension of the tumor into the right atrium. Filling defects were also observed in peripheral branches of the pulmonary artery. Propagation into the right atrium was found in cases so far. In all of them, the tumor arose in the upper third of the inferior vena cava.

Resection of the tumor was reported in six cases. The first operation was carried out by Melchior in 1921. Three patients survived the surgical procedure and were still alive between nine months and one year after surgery.

REFERENCES

Rhythmic Shoulder Girdle Muscle Contractions as a Complication in Pacemaker Treatment

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A 69-year-old man had been treated for 5½ years with permanent transvenous pacemaker for Adams-Stokes syndrome. On the day following vigorous arm exercise he had muscle contractions in the right shoulder synchronous with the heart activity. Local damage to the electrode cable with current leakage to the upper brachial plexus was verified by neurophysiologic examination and at operation. A simple method of repairing the insulation defect is reported.

A pacemaker-treated patient presented some peculiar neurologic symptoms—rhythmic muscle contractions arising in the right shoulder girdle on abduction of the arm. Neurologic examination led to suspicion of damage to the electrode cable close to the upper brachial plexus. After operation, the patient made an uneventful recovery and became free of the muscle jerks. No similar case is known to have been reported previously.

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CASE REPORT

A 69-year-old man reported in March 1972 with painful jerks of his right arm. He had received a pacemaker in 1966 at age 63 because of a brief attack of syncope. Two years earlier he had begun to experience occasional attacks of bradycardia.

At the time of his only Adams-Stokes attack, in September, 1966, he was examined in the Department of Medicine, Serafimerlasarettet, where an ECG showed second degree A-V block and left bundle branch block. A transvenous pacemaker electrode was implanted through the right external jugular vein by the method of Lagergren1 because of his reduced physical working capacity and at least one Adams-Stokes attack.

He had no syncopal episodes during course of pacemaker therapy, (impulse generator Siemens-Elema EM 152, stimulation frequency 70/min, output voltage 6.2, impulse duration 1.8 msec). On routine replacement of the impulse generator in May 1967 an exostosis on the right clavicle was found in intimate relation to the brachial plexus. The exostosis was excirpated.

Present Neurologic Symptoms

On February 1, 1972, one day after scraping a wall for three hours during which time he constantly had his right arm elevated at the shoulder-joint and bent at the elbow-joint, he had pains in the whole of his right arm. On elevating the arm he noticed rhythmic jerks in the shoulder. The symptoms recurred every time he abducted or elevated his arm. A sudden turning of his head to the left could also provoke the jerks. The strength of the right arm and hand was not reduced, nor had he any local pain or reduction of sensibility in the arm.

Physical examination on March 7, 1972, revealed a man in good general condition without sign of heart failure at rest. Heart rhythm, (66/min) was regular. The first heart sound was generally loud, the second normal. A systolic murmur of degree 1/6 (AHA) was heard over the fourth intercostal space. His blood pressure was 165/80 mm Hg.

On 10-15° abduction of the right arm, rhythmic muscle jerks occurred in the right shoulder girdle. The jerks were observed in the deltoid, supraspinatus and infraspinatus muscles and a small part of the pectoralis major. They gave rise to slight abduction, forward elevation and outward rotation in the shoulder-joint. On continued abduction of the shoulder, the jerks successively increased in amplitude. They were maximal at 45° abduction and diminished again at 90°. When the arm was elevated above the horizontal plane, the jerks successively disappeared and ceased entirely at 30° elevation, above the horizontal plane. When the arm was lowered again, the jerks recurred.

The muscular jerks were found to be synchronous with the heart activity. They were therefore assumed to be generated from the pacemaker impulses and to be caused by an insulation defect in the electrode cable close to the upper brachial plexus. Fluoroscopy of the area around the right clavicle showed that movements of the right arm did not produce any abnormal displacement of the electrode cable. No skeletal changes on the clavicle were observed.

Electromyographic examination. Two silver plate electrodes were applied to the skin over the following muscles: deltoid, supraspinatus, infraspinatus, pectoralis major, biceps and triceps on the right side. The electromyographic activity was recorded together with an ECG on an inkwriting Grass electroencephalograph.

When the seated patient let his arm hang down in complete relaxation, no electromyographic activity was recorded from the muscles studied (Fig 1A). A directly conducted stimulation artefact from the pacemaker and ECG activity were registered. When the patient voluntarily raised his right arm and held it 45° abducted, a continuous activity was recorded in the deltoid, supraspinatus and infraspinatus muscles. On every pacemaker impulse, furthermore, a twitch contraction was recorded in these three muscles (Fig 1B). No twitch activity was recorded from the pectoralis major, biceps or triceps muscles. When the patient raised his arm above the horizontal plane, the jerks disappeared (Fig 1C).

The pacemaker-induced contractions occurred also if the patient's arm was passively raised to the corresponding positions. Simultaneous recording of an ECG showed no distinct relation in time between cerebral activity and muscle jerks.

In order to study in detail the time relation between the pacemaker stimulation artefact and the muscle jerks, an ECG was recorded simultaneously with the EMG activity from the deltoid or infraspinatus muscle on a double-beam cathode ray oscilloscope. These recordings showed that the time from the start of the pacemaker impulse to the first part of the muscle contraction was approximately 3 msec which corresponds to a conduction velocity in the motor nerve fibers of about 60-75 m/sec. With continuous film recording (Fig 2) it was evident that, after every induced twitch, a roughly 50 msec pause occurred in the voluntary activity of the muscle.

![Figure 1. Electromyographic recordings from the right shoulder girdle in different arm positions related to pacemaker-controlled electrocardiographic activity. A) The arm hangs relaxed: the pacemaker stimulation artefact and ECG activity are recorded. B) At 45° abduction twitches occur (tall spikes) which are synchronous with the pacemaker activity. C) Elevation of the arm above the horizontal plane causes the twitches to disappear. Time bar 0.5 sec. Vertical bar 1 mV.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/20958/)
and the patient. The amount by which the stimulation effect of the heart electrode decreases depends on the relative impedance between the heart electrode and cable defect, respectively, and the patient. In this case, there was probably only a small current leak in the region of the clavicle, as cardiac pacing was unaffected. A larger insulation defect might have caused a current distribution which would have resulted in sub-threshold heart stimulation and asystole. This mechanism was probably the cause of sudden death in a case described by Edhag.4

The insulation defect most probably resulted from minor damage to the electrode cable during surgical excision of the exostosis on the right clavicle 1½ years following pacemaker implantation. The exostosis, in turn, may have resulted from mechanical trauma to the periosteum in conjunction with implantation of the electrode.

In our patient a long time elapsed between extirpation of the exostosis and the onset of symptoms. It is therefore likely that the intensive arm movements which preceded the arm jerks added to a partial insulation defect not extending down to the steel core of the cable. That the insulation material had undergone such changes because of 5½ years’ use, or that it did not withstand the stresses in conjunction with the intense arm work, appears improbable. Before this type of electrode came into clinical use, it had been subjected in the laboratory to mechanical strains corresponding to several decades of clinical use.4 It should perhaps be added here that in the Department of Thoracic Surgery, Karolinska Sjukhuset in Stockholm, up to January 1973, 700 transvenous electrodes had been implanted, some 500 of which more than three years previously (in fact, in 1972 some electrodes had been in use for ten years), without sign of limited life of the cable insulation in a single case.

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Pulmonary Monosporosis: Report of a Case with Precipitating Antibody*

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