Letters

but is also encountered in long standing sympathetic denervation of the pupil,⁶ and can be found in either Horner's or Raeder's syndrome.

This case illustrates the need for thorough investigation of Raeder's and Horner's syndromes.

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Thrombosis of cerebral veins following intravenous application of clomipramine

Sir: Thromboembolic complications, mostly within the veins of the legs, following treatment with antidepressants are well known. We report a patient, who developed a lethal thrombosis of the cerebral veins shortly after the beginning of a treatment with intravenous clomipramine.

A 61 year old woman suffering from an agitated depressed state did not respond to oral applications of different antidepressants and electroconvulsive therapy. She had a history of thrombosis of the veins of the right leg and of cardiac failure treated adequately with digitalis. Physical examination revealed a systolic apical murmur and a resonant chest percussion. A chest radiograph showed an enlargement of the left ventricle. Further investigations (ECG, EEG, laboratory reports) were unremarkable. On the 273rd day after admission to the hospital, treatment with clomipramine infusions (50 mg in 500 ml Ringer's solution) was started. The next day

the patient vomited, then had a seizure, afterwards she became comatose and died at the 275th day of treatment.

CT on the day she died showed haemorrhage in the left hemisphere. Autopsy revealed complete occlusion of the superficial cerebral veins, the superior longitudinal sinus, the right lateral sinus, the right sigmoid sinus, partial occlusion of the left sigmoid sinus and the left lateral sinus, and intracerebral haemorrhage of the left hemisphere with oedema.

Thrombosis of cerebral veins are rare compared with other diseases of the cerebral vessels. We found only one other case report concerning thrombosis of the Galenic system veins after the start of intramuscular injections with amitriptylinepamoate.¹ Besides the slowdown of the blood flow by prolonged bed rest we assume that changes of the blood coagulation and of the vessel endothelium played a part in the pathogenesis of thrombosis in our patient.² This case draws attention to the importance of antithrombotic prophylaxes during treatment with infusions of antidepressants, such as low-dose heparin (5,000 units three times a day), bandaging of the legs, and exercise.³

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Myotonic dystrophy with unilateral bulbar involvement

Sir: Myotonic dystrophy is a hereditary autosomal dominant multisystem disease

characteristically affecting skeletal muscle, but also involving the heart, gastrointestinal smooth muscle, respiratory system, skull, brain, eyes, and skin.¹

Among the bulbar signs, weakness of the pharyngeal muscles is quite common and not as a late manifestation of the disease.² On the other hand, laryngeal abnormalities have been reported late in the course of the disease but are not common, and are mild and bilateral.³⁻⁵

We present here an unusual case of myotonic dystrophy in which the laryngeal symptoms were the presenting signs combined with unilateral atrophy of the neck and shoulder. A 29 year old unmarried man was admitted to the Ear-Nose-Throat department, because of progressive dysphonia in the past year and dysphagia for 4 months. On examination a unilateral left vocal cord paralysis together with severe weakness of the left pharyngeal muscles were observed. The rest of the examination was normal. Thyroid scan, plain radiographs of the foramen jugularis, and computed tomography of the brain stem and the base of the skull were negative.

He was transferred to the neurological department for further investigation. On examination myopathic facial appearance together with unilateral atrophy of the left sternocleidomastoid and trapezius muscles were observed. Myotonic reaction of both hands, decreased deep tendon reflexes in the arms without any decrement of strength or atrophy of the arms and hands were found. Bilateral posterior polar mild cataract, small testicles and first degree A-V block in an ECG were the additional findings. Chest radiographs were normal and lung function tests showed mild proximal airway obstruction. A barium swallow radiograph showed bilateral pooling of barium in the valleculae, more pronounced on the left side, with normal oesophageal peristalsis. Electromyographic study showed the typical myotonic discharges sounding like a "dive bomber" with normal conduction velocities in peripheral nerves of the limbs.

The diagnosis of jugular foramen syndrome was eliminated by normal CT of the base of the skull, and local disease in the larynx was excluded by 2 years of surveillance without any new finding in an ENT examination.

We have checked nine more siblings and found another brother 40 years old with cardiac pacemaker for 7 years, with advanced myotonic dystrophy. Five more siblings had partial syndrome, with cataract and diabetes mellitus.

The laryngeal involvement in myotonic dystrophy is well accepted even though it is