takes several months to change in response to thyrotoxicosis, the value remains normal in patients with transient thyrotoxicosis. Clinical distinction between these two conditions is important for management, since transient thyrotoxicosis with hyperemesis gravidarum only occasionally needs treatment with antithyroid drugs, whereas such treatment usually is required when there is pre-existing thyrotoxicosis in association with hyperemesis gravidarum.

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References

- 1 Lao TTH, Chin RKH, Cockram CS, Panesar NS. Transient hyperthyroidism in hyperemesis gravidarum. J R Soc Med 1986;79:613-5
- 2 Chin RKH, Lao TTH, Cockram CS, Swaminathan R, Panesar NS. Transient hyperthyroidism in pregnancy. A case report. Br J Obstet Gynaecol 1987;94:483-4
- 3 Lao TTH, Chin RKH, Swaminathan R, Panesar NS, Cockram CS. Erythrocyte zinc in differential diagnosis of hyperthyroidism in pregnancy: Preliminary report. Br Med J 1987;294:1064-5

'Dry tap' in glue ears

Further to Mr Steel's letter (August 1987 JRSM, p 537), a well inflated middle ear does make myringotomy easier, and certainly with a grossly retracted tympanic membrane any inflation is a help.

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Diaphragm weakness and syringomyelia

Sir, We were interested in the case of diaphragm weakness reported by Mier et al. (May 1987 JRSM, p 315). We have recently studied a patient with severe bilateral diaphragmatic weakness without gross orthopnoea, secondary to a Chiari malformation and a cervical syrinx.

This 55-year-old man had noticed progressive numbness, weakness and wasting of the hands, and unsteadiness of gait since the age of 52, and during the year before presentation he developed moderate breathlessness on exertion and while lying supine. On examination he had lower motor neurone signs in the upper limbs and spasticity in the legs; he also had a short neck. Cranial nerves were normal, and there was an area of suspended sensory loss for pain and temperature modalities from C1 to T10 bilaterally.

Plain X-rays showed marked basilar impression and fusion of cervical vertebrae. Nuclear magnetic resonance scans showed protrusion of the cerebellar tonsils through the foramen magnum, and an extensive syrinx involving the cervical and thoracic spinal cord. Radioscopy was suggestive of an almost complete bilateral diaphragmatic palsy, and he was unable to generate an active transdiaphragmatic pressure. After $\rm CO_2$ rebreathing for 3 minutes, arterial $\rm PCO_2$ rose from 46.6 mmHg (breathing air) to 76 mmHg, and pH dropped from 7.39 to 7.25 without a significant increment of ventilation. A polysomnographic study

showed 12 apnoeas during the night of central type, and the sleep efficiency was reduced to 0.68. In view of the abnormalities found, surgical treatment was ruled out.

Respiratory abnormalities have been described previously in syringomyelia¹⁻³ but, as far as we know, there has been no report on the coexistence of bilateral diaphragmatic palsy, which may be a contributing factor in the occurrence of CO₂ retention and sudden death during sleep in these patients.

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References

- 1 Bokinsky GE, Hudson LD, Weil JV. Impaired peripheral sensitivity and acute respiratory failure in Arnold-Chiari malformation and syringomyelia. N Engl J Med 1973;288:947-8
- 2 Rodman T, Resnick M, Berkowitz RD, Fennelly JF, Olivia J. Alveolar hypoventilation due to involvement of the respiratory center by obscure disease of the central nervous system. Am J Med 1962;32:208-17
- 3 Haponik EF, Givens D, Angelo J. Syringobulbia-myelia with obstructive sleep apnea. Neurology 1983;33: 1046-9

Antiphospholipid antibody syndrome

Sir, We read with interest the timely and comprehensive review on this subject by Bingley and Hoffbrand (July 1987 JRSM, p 445). However, we would take issue with the unqualified suggestion that the kaolin partial thromboplastin time (KPTT) can be used satisfactorily as a screening procedure for antiphospholipid antibody. Our own studies have demonstrated the need for a comprehensive range of coagulation and immunological investigations in order to exclude the presence of lupus anticoagulant/ anticardiolipin antibody1. In particular, in a large series of subjects with unexplained thrombosis, poor obstetric history, or collagen vascular disease, lupus anticoagulant would have remained undetected in 22% of cases had the KPTT alone been used for screening purposes. In our hands, the dilute Russell's viper venom time is a more sensitive test and is well within the capabilities of a routine haematology laboratory. It is also noteworthy that samples positive for IgG or IgM anticardiolipin antibody were occasionally negative for lupus anticoagulant in all coagulation tests.

It is thus clear that reliance cannot be placed on any single screening test, and detection of these antibodies, which we agree are of diagnostic and therapeutic importance, necessitates a comprehensive clinical and laboratory approach.

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Reference

1 Cooper SM, Malia RG, Preston FE, Duncan SLB, Smith ARB, Greaves M. Clinical and laboratory features associated with lupus anticoagulant. Thromb Haemost 1987;58:1432