well to treatment with metronidazole. We were able, however, to make an immediate diagnosis of infection with *Trichomonas vaginalis* by microscopical examination of wet preparations of the tracheal aspirate and gastric contents.

We cannot be certain that our patient's disease was caused by this organism. Aspiration of amniotic fluid results in a similar picture, and the organism may have been just a contaminant. Bacterial pneumonia was also a possibility, despite the negative cultures, as the patient improved with antibacterial treatment without receiving specific treatment for *Trichomonas vaginalis*. Nevertheless, the findings in our patient, including his slow recovery, and the report of McLaren *et al* suggest that *Trichomonas vaginalis* may be an unrecognised cause of neonatal pneumonia.

Determining the cause of respiratory distress in an ill baby is often difficult, and infection with *Trichomonas vaginalis* should be considered when the aetiology is not clear. In these circumstances direct microscopical examination of wet preparations of gastric content and tracheal aspirate is a useful screening test. Further work is needed on the incidence of contamination of the newborn with this organism, which commonly infects mothers during pregnancy,³ and consequent respiratory infection.

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² Swischuck LE. Radiology of pulmonary insufficiency. In: Thibeault DW, Gregory GA, eds. *Neonatal pulmonary care*. Menlo Park, California: Addison-Wesley, 1979:126-52.

³ McLaren LC, Davis LE, Healy GR, James CG. Isolation of *Trichomonas vaginalis* from the respiratory tract of infants with respiratory disease. *Pediatrics* 1983;71:888-90.

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Argyll Robertson pupils due to neurosarcoidosis: evidence for site of lesion

In 1869 Argyll Robertson described the syndrome of miosis accompanied by absent pupillary light reaction and intact near response but the location of the responsible lesion has been in doubt.¹ This report describes a patient with Argyll Robertson pupils in whom magnetic resonance imaging located a lesion in the periaqueductal region of the mid brain.

Case report

A 28 year old white housewife had presented in 1974 with weight loss, bilateral facial palsies, uveitis, retinopathy, and numbness and pain affecting her limbs and trunk. She had bilateral hilar adenopathy and a Kveim-Siltzbach skin test had proved positive. Sarcoidosis had been diagnosed, and she had responded slowly to steroid treatment. Her cerebrospinal fluid had contained 12 lymphocytes/ml and a total protein concentration of 0.95 g/l.

Ten years later she became lethargic with a return of pain and numbness to her limbs and trunk. On examination she had bilateral ptosis and small pupils. They did not react to bright torch light directly or consensually. The pupils constricted to the near response and accommodation was normal. Sensation was reduced bilaterally in the three divisions of the trigeminal nerve. Her pterygoids and masseters were weak. She had bilateral lower motor neurone facial weakness and sensorineural deafness. Her oropharynx was numb and the tongue deviated to the left. Her limbs and trunk had large areas where sensation to pinprick and temperature was impaired. Her reflexes were normal and plantars flexor.

Corrected visual acuity was 6/6 N5 bilaterally. On examination by slit lamp the irides appeared normal and neither uveitis nor retinopathy were present. The pupils dilated poorly in the dark and measured 3.8 mm on the right and 4.4 mm on the left. Two drops of 0.01% arecoline (a parasympathomimetic agent) did not produce a supersensitive response. Light-near dissociation of her pupils was confirmed by television pupillometry.² Serum

Venereal Disease Research Laboratory and treponemal haemagglutination tests proved negative and the cerebrospinal fluid was normal. No abnormality was seen on a 9800 computed tomography brain scan but a magnetic resonance imaging scan showed an increase of T₂ lesions around the cerebral aqueduct at the level of the superior colliculi and at the anteriolateral angles of the lateral ventricles.

Magnetic resonance imaging scan at the level of the superior colliculi showing an increased T₂ lesion around the cerebral aqueduct which is just posterior to a central artefact.

Comment

This patient presented with acute sarcoidosis and neuro-ophthalmic complications. Ten years later she developed a cranial polyneuropathy with Argyll Robertson pupils.

One theory suggests that the Argyll Robertson syndrome is caused by interruption of the light reflex pathway and inhibitory supranuclear pathways to the Edinger-Westphal nucleus at a point dorso-rostral to the oculomotor complex,³ while fibres controlling accommodation, being placed more ventrally, are left unaffected. A lesion in the periaqueductal grey matter at the level of the superior colliculi is consistent with this theory.

Neurological damage is seen in about seven per cent of patients with sarcoidosis,⁴ and internal ophthalmoplegias are rare.⁵ The seventh cranial nerve is affected most commonly and often bilaterally. The second, fifth, and eighth cranial nerves are sometimes affected, but rarely the others. Normally, the cerebrospinal fluid shows pleocytosis and raised concentration of total protein: it may, however, be normal. Granulomas presenting as intracranial space-occupying lesions have been reported in sarcoidosis as has stenosis of the cerebral aqueduct. There has never been a report, however, of a localised granuloma in the periaqueductal grey matter causing Argyll Robertson pupils.

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² Lowenstein O, Lowenfeld IE. Electronic pupillography. A new instrument and some clinical applications. *Arch Ophthalmol* 1958;59:352-62.

³ Lowenfeld IE. The Argyll Robertson pupil, 1869-1969. A critical survey of the literature. *Surv Ophthalmol* 1969;14:199-299.

⁴ James DG, Sharma OP. Neurological complications of sarcoidosis. *Proc R Soc Med* 1967;60:1169-70.

⁵ Henkind P, Gottlieb MB. Bilateral internal ophthalmoplegia in a patient with sarcoidosis. *Br J Ophthalmol* 1973;57:792-6.

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