infection will be differentiated by the absence or presence of virus specific IgG antibody in serum collected early in, or preferably before, pregnancy.

The detection of infection in the first trimester, before the initial visit to the antenatal clinic, will remain difficult. The presence of virus specific IgM antibody in serum collected at the first clinic visit will suggest a maternal primary cytomegalovirus infection in the first trimester, but only if the woman lacks virus specific IgG antibody before pregnancy. Such IgM antibody will remain an insensitive marker of recurrent infection in that period of pregnancy. Hence the prospective study recommended by Dr Best will be both complex and expensive, requiring the examination of large numbers of samples of maternal urine for virus excretion. Nevertheless, further accurate knowledge of the pathogenesis of congenital cytomegalovirus infection, required to assess the potential role of vaccination in its prevention, will be obtained only in this way.

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## Is screening for bacteriuria in pregnancy worth while?

SIR,—The recent report on screening for asymptomatic bacteriuria in pregnancy by Dr M Campbell-Brown and colleagues (20 June, p 1579) is of particular interest to us, as, after 20 years, screening was stopped in this centre in 1986.

We had assumed that a quarter of patients with asymptomatic bacteriuria, defined as more than $10^5$ organisms per ml in a single midstream specimen of urine, would progress to a symptomatic urinary tract infection, defined as clinical evidence of pyelonephritis requiring admission to hospital and confirmed bacteriologically.[1,2] Since 1965 every patient had been screened at her first visit, and the interest in the subject was revived from 1980 onwards. All patients were treated on the basis of sensitivity, and 98% of the patients were sensitive to either sulphamethizole or nitrofurantoin (our first choice drugs in view of sensitivity and cost). Almost 80% of patients had a repeat midstream urine sample tested after treatment, and almost 80% were sterile; further treatment was given for "failed treatment."

Over six years the dose and duration of treatment were reduced progressively from a maximum of sulphamethizole 600 mg or nitrofurantoin 300 mg daily in three divided doses for 14 days in 1981 to sulphamethizole 300 mg and nitrofurantoin 150 mg for three days in divided doses. The incidence of failed prophylaxis—that is, patients with positive results on screening who subsequently develop a symptomatic infection—did not change.

The next logical step was a trial of treatment versus non-treatment. In 1985, 6883 patients were screened, yielding 220 positive specimens (3·2%). On the toss of a coin 100 patients were treated and 120 patients were not; the follow up rate was 81%. At follow up 73% of the treated patients and 48% of the non-treated patients were found to have sterile urine. In the treatment group further treatment, including if necessary maintenance treatment, was given to render the urine sterile. Three patients in each group were admitted with pyelonephritis. To cover the possibility that patients were being treated at home by their general practitioners, each patient was interviewed after delivery. Four in the treatment group and five in the non-treatment group had been treated at home for an unspecified urinary tract infection. Other findings were similar to those of Dr Campbell-Brown and coworkers, asymptomatic bacteriuria being commoner in patients with a history of urinary tract infection and in the lower social groups, particularly among unmarried mothers. Symptomatic urinary infection was more likely (2·3%) in patients with asymptomatic urinary tract infection than in patients with sterile urine (0·5%), figures identical with those of Dr Campbell-Brown and colleagues, but the belief that up to a quarter of untreated asymptomatic patients would develop symptomatic urinary tract infections is completely unfounded. Regrettably, this cheap and simple test, which met many of the criteria of the ideal screening test, was based on an initial false premise, or the natural history of the disease has altered dramatically.

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## Lower oesophageal contractility as an indicator of brain death

SIR,—The comments of Dr D J Hill (6 June, p 1488) and Drs A R Aitkenhead and D I Thomas (16 May, p 1287, and 27 June, p 1691) concerning lower oesophageal contractility need further clarification.

The motor function of the oesophagus is controlled by the oesophageal motility centre, which comprises the dorsal motor nuclei of the vagus nerves and adjacent reticular activating centre.[1] The fact that non-propulsive contractions of the oesophagus may be produced by acoustic stimulation or psychological stress suggests that this centre is in turn connected to, at least, auditory pathways and higher centres, afferent and efferent vagal fibres connecting the brain stem to the oesophagus. Though rather specific motor responses may be induced in the isolated opossum oesophagus, all the evidence suggests that in both animals and humans normal oesophageal motor function necessitates an intact brain stem and vagal connections.[2-6]

After bilateral vagotomy animals experience an initial period of complete oesophageal paralysis; intrinsic reflexes may evolve, perhaps within 12 hours, to allow some local reflex activity.[4,7] Thus after acute and complete brain stem death no spontaneous lower oesophageal contractions would be expected but a variable amount of provoked secondary peristaltic activity would appear. The observations of Drs M E Sinclair and P M Suter (11 April, p 935) and those of Drs Aitkenhead and Thomas with regard to one of their cases are entirely consistent with this interpretation of the known physiology. Drs Aitkenhead and Thomas, however, seem to be averse to this interpretation.

The brain stem is not a small, compact neurological entity but an extensive section of the brain. After trauma death of brain stem tissues typically ensues from oedema and infarction, which quite reasonably may affect some parts of the brain stem while sparing others. When most of the damage is supratentorial brain stem damage often results from tentorial herniation and oedema. In such circumstances the upper brain stem is much more likely to be damaged than the lower brain stem. The United Kingdom tests for brain stem death are simple clinical tests and are by no means exhaustive tests of all the measurable and known functions of the brain stem. The present criteria do not, for example, include tests of cardiac vagal tone, which could be shown by administering atropine.[8] It is not therefore surprising that the particular motor function of the vagus nerve concerned with oesophageal motility has some residual function in some patients who are considered by the United Kingdom to be brain dead.

In two of the four patients studied by Drs Aitkenhead and Thomas brain stem auditory evoked responses were measured and found to be absent. In these patients the extensive auditory pathway in the brain stem, which is superior to the vagal motor nuclei, may have been damaged while lower parts of the brain stem were preserved. Alternatively the auditory nerve may have been damaged by oedema or formation of a haematoma, as both of these patients had suffered severe head injuries.

I believe that it is reasonable to assume that the presence of continued spontaneous lower oesophageal contractility in paralysed and mechanically ventilated patients with head injuries must be regarded as evidence that a considerable part of the oesophageal motility centre is viable. This view is entirely compatible with the United Kingdom criteria for brain stem testing, which confine tests of motor responses within the cranial nerve distribution to somatic areas.[9]

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## Herpes zoster of second and third segments causing ipsilateral Horner's syndrome

SIR,—Dr H S K Wimalaratna and colleagues (6 June, p 1463) have brought to our attention the interesting association between thoracic herpes zoster and Horner's syndrome. They seem, however, to be confused over their pharmacological tests of pupil function. Firstly, cocaine does not distinguish between preganglionic and postganglionic Horner's syndrome but can be used