AIDS to colleagues who will perform venepunctures or open surgical procedures on that patient. Disclosure to a colleague concerned in a patient's treatment is not, in itself, improper. The dilemma arises when the patient withholds permission for disclosure. If in so doing he might jeopardise the health of another person I suggest that he has no right to refuse disclosure.

Is there, then, a manifest risk to other health care workers? Dr Gillon says that the probability is very low. McEvoy *et al* have documented seven cases of occupationally acquired human immunodeficiency virus. How would these seven colleagues feel if they knew that they had wilfully been denied the information that the patient from whom they contracted the infection had AIDS?

Someone once parodied John Stuart Mill: "Your right to swing your fist ends at my nose." I suggest that an AIDS patient's right to confidentiality stops when, in the words of the United States Supreme Court Justice Louis Brandeis, "there is a clear and present danger" to the health of another person. In the case of AIDS I suggest that Justice Brandeis's criterion is met and that the patient's right to confidentiality is, in those specific circumstances, forfeited.

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SIR,—During the past century medicine has continued to face fatal diseases for which there has been no cure: what is so special about the acquired immune deficiency syndrome (AIDS)?

Neither of us has any hesitation in treating patients with the disease, but, as with any similar illness, it is most helpful to have all possible information relating to the diagnosis before starting treatment. The failure of any patient who knows he has the illness to inform medical staff is a gross breach of the traditional relationship of trust that exists between them.

Purely practical considerations would also seem to dictate this—principles which have traditionally held in the case of diseases such as hepatitis B, tuberculosis, and syphilis. In none of these cases has awareness of the diagnosis affected the doctor's willingness to treat the patient, and there has been no debate on whether these should continue to be notifiable. Tuberculosis is eminently treatable and of low infectivity, yet a patient with open tuberculosis can still forcibly be retained for treatment. What are the features that distinguish AIDS from these diseases, each of which, in its time, has been untreatable and often fatal?

We sympathise with anyone with a terminal illness and will continue to care for them all, regardless of the diagnosis. Our only request is for equal consideration.

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SIR,—Dr Raanan Gillon has carefully explained why he advocates, almost absolutely, the maintenance of medical confidentiality for patients infected with the human immunodeficiency virus (HIV) or suffering from the acquired immune deficiency syndrome (AIDS) (27 June, p 1675). He has, however, avoided discussion of recent developments that may soon concentrate our minds wonderfully.

The General Medical Council's standards committee has set up a working party to look into the problems of confidentiality and consent when treating patients with AIDS. One member of the working party has, however, already made his own thoughts ominously clear. Professor Ian Kennedy of the Centre of Medical Law and Ethics has given advice to the social services select committee. His predictions have been reported in the *BMJ* but bear repeating[1]: suggesting that "the time was not too distant when a court action would be brought against a doctor for failing to advise the spouse of a patient who was infected with HIV and that spouse became infected. The court would be unlikely to accept a defence of confidentiality" (4 April, p 913). Referring to the specific example of the doctor who failed to warn his group partners (or presumably a consultant who failed to tell a general practitioner) of a patient with AIDS, the professor "had little doubt that if the other doctor or a third party suffered harm as a result of the omission, the first doctor could be held liable."

Despite these powerful warnings, the BMA has bravely decided to fight to the last member and has decreed that confidentiality should be upheld if patients refuse to allow disclosure. So there you have it. Professor Kennedy is surely right that a court case will soon be brought, but will it be for maintaining or breaking confidentiality? Why wrestle with knotty ethical problems? Lawyers solve Gordian knots much more simply by cutting them. Perhaps the GMC will now delay its working party's report until the result of the first case is known. Perhaps there might even be a gentleman's agreement. Doctors could be punished by the GMC for breaking confidentiality and by the courts for maintaining it.

AIDS has nothing to do with blame or punishment—unless, of course, you happen to be the messenger. The court case will be like an ancient ritual for public atonement. There will be one death (or two), one medical scapegoat destined for the wilderness, and various high priests who will come away smelling of roses.

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1 Warden J. Cautionary tales of pitfalls ahead. *Br Med J* 1987;**294**: 913.

Testicular torsion in older men

SIR,—Mr M J S Dennis and colleagues (27 June, p 1680) acknowledge the difficulties of establishing the diagnosis of testicular torsion, highlighting the consequences of delaying exploration and, in particular, of dismissing the diagnosis in older patients. Experience at the Leicester General Hospital supports the view that age does not preclude the diagnosis of testicular torsion.

This hospital's experience of patients with testicular torsion has recently been examined. Over the 10 years since January 1977, 243 patients have undergone emergency scrotal exploration and testicular fixation for acute testicular torsion. Of these, 196 patients were aged under 20, 31% being prepubertal; of the remainder, nine men (4%) were aged 30 to 35. A diagnosis of torsion was substantiated and successfully treated with bilateral testicular fixation in two men aged 52 and 57 years. The 57 year old patient was initially diagnosed as probably having an epididymo-orchitis, but, fortunately, prompt scrotal exploration was undertaken and showed a high investment of the tunica vaginalis with a double twist of the cord. Both patients gave a history of intermittent attacks of scrotal pain. A 53 year old man was referred with a diagnosis of acute torsion after three days of testicular pain. This was again thought to be epididymo-orchitis, but because of the lack of urinary symptoms and negative findings from a midstream urine specimen prompt exploration of the scrotum was undertaken, unfortunately showing haemorrhagic infarction of the testis with four twists of the cord. An orchidectomy was performed.

Over the same period epididymitis or orchitis was diagnosed in 389 patients, 60% being aged between 30 and 80; 90 patients (the largest single group) were in their second decade.

Torsion of the testis is a real surgical emergency for the reasons outlined by Mr Dennis and colleagues, and we agree that the diagnosis ought to be considered in any male with testicular pain, irrespective of age.

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School dinners

SIR,—Dr B A Wharton (27 June, p 1635) questions whether school meals should exist and, if so, in what form and raises fundamental issues about their effectiveness. He incorrectly, however, suggests that little or no work has been done on the nutritional value of school meals.

In fact, objective reports about the association between school meals and rate of growth in children and the nutritional state of children receiving free school meals have been published fairly recently.[1,2] Information is also available about the factors influencing uptake of school meals in Britain, which supports Dr Wharton's suggestion of converting popular food into healthy food.[3] In view of rapid changes in food policy in schools and in family social circumstances, it is essential to review the value of school meals periodically.

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Congenital cytomegalovirus infection

SIR,—Dr Jennifer M Best (6 June, p 1440) correctly emphasises that further prospective studies of cytomegalovirus infection in pregnant women will be required to establish the relative importance of primary and recurrent maternal infection as antecedents of congenital infection. Such studies will, however, be difficult to perform.

Though used in recent studies,[1-4] the detection of cytomegalovirus specific IgM antibody in maternal serum is not a sufficiently discriminatory marker of primary cytomegalovirus infection during the preceding three or four months. On the contrary, cytomegalovirus specific IgM antibody may persist for more than four months, may appear (albeit rarely) during recurrent infections, and is missing in at least 10% of primary infections.[5] Recurrent cytomegalovirus infection cannot be diagnosed reliably during pregnancy by tests for virus specific antibody as IgM antibody and even rising titres of neutralising antibody may be absent.[5,6] Consequently, a future prospective study of cytomegalovirus infection in pregnancy will have to investigate maternal virus excretion (in urine, for example[7]) as well as antibody response to document infection. Primary and recurrent

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