3 Froelicher VF, Morgan D. Exercise testing and ancillary techniques to screen for coronary heart disease. *Prog Cardiovasc Dis* 1981-2;**24**:261-74.
4 Bruce RA, DeRouen TA, Hossack KF. Value of maximal exercise tests in risk assessment of primary coronary heart disease events in healthy men. *Am J Cardiol* 1980;**46**:371-8.
5 Gordon DJ, Lars-Goran E, Karon JM, *et al*. Predictive value of the exercise tolerance test for mortality in North American men: the Lipid Research Clinic's mortality follow up study. *Circulation* 1986;**74**:252-61.
6 McHenry PL, O'Donnell J, Morris SN, Jordan JJ. The abnormal exercise electrocardiogram in apparently healthy men: a predictor of angina pectoris as an initial coronary event during long term follow up. *Circulation* 1984;**70**:547-51.
7 Hollenberg M, Zoltick JM, Go M, *et al*. Comparison of a quantitative treadmill exercise score with standard electrocardiographic criteria in screening asymptomatic young men for coronary artery disease. *N Engl J Med* 1985;**313**:600-6.
8 Chamberlain DA. Routine and exercise electrocardiography in aircrew; technique, interpretation and recommendations. *Eur Heart J* 1984;**5**(suppl A):55-60.
9 Froelicher VF, Thompson AJ, Wolthuis R, *et al*. Angiographic findings in asymptomatic aircrewmen with electrocardiographic abnormalities. *Am J Cardiol* 1977;**39**:31-8.
10 Barnard RJ, Gardner GW, Diaco NV. "Ischaemic" heart disease in firefighters with normal coronary arteries. *J Occup Med* 1976;**18**:818-20.
11 Borer JS, Brensike JF, Redwood GR, *et al*. Limitations of the electrocardiographic response to exercise in predicting coronary artery disease. *N Engl J Med* 1975;**293**:367-71.
12 Royal College of Physicians of London. The cardiovascular fitness of airline pilots. *Br Heart J* 1978;**4**:335-50.

When things go wrong—again

Wherever two or more American doctors are gathered together the word malpractice is likely to enter the conversation. Malpractice, and particularly the premiums needed to insure against it, looms large in the American medical consciousness—and no wonder. Malpractice litigation may account for a quarter of the amount spent on doctors' services in America (about $20 billion), and it stops doctors delivering babies, "takes the fun out of medicine," and still fails to compensate most of those injured by medical care, even those injured through negligence.[1] Legislatures in most if not all states are struggling, without much success, to contain the crisis.

We are still a long way from such misery in Britain, but this year's 87% increase in defence society subscriptions has produced squeals of pain from doctors (p 666). It follows last year's 70% increase, when we published a leading article arguing not for reform of a legal system manifestly incapable of dealing with this medicosocial problem but rather for a newly minted no fault system.[2] We have been arguing the case for such a system for years,[3] and in the past year the BMA has come out in favour of a no fault system, one that would cover medical misadventure rather than all disability.

Now the BMA wants a parliamentary select committee to investigate this problem, a recommendation to be wholeheartedly supported—and not just because doctors' pockets are suffering. Action now may avoid an American style crisis, and a select committee would consider the interests of all groups—patients, doctors, lawyers, and the defence societies. More than anything we need facts, and the select committee would have the power to require people to give evidence. The defence societies might have to release information that would give us more insight into the extent of malpractice. But that still would not tell us how many people are injured by medical care and what happens to them—because many such people make no complaint. Even after more than a decade of worrying about malpractice the Americans lack the basic data. Dr John Havard quoted last month (15 August, p 399) a statement from an American government committee that the debate on malpractice had "been based more on rhetoric, speculation and misconception, than on factual quantitative data."

We need to avoid such a fate, and a select committee inquiry would help. It would not in itself, however, be enough. We also need a large scale prospective investigation, although urgency may necessitate a retrospective study. The Nuffield Foundation was considering an investigation but seems to have dropped the idea. Perhaps it should think again, and perhaps the defence societies would like to contribute funds—while they still have some to contribute.

RICHARD SMITH
Assistant editor, *BMJ*

1 Mills DH. Medical insurance feasibility study. *West J Med* 1978;**128**:360-5.
2 Smith R. When things go wrong. *Br Med J* 1986;**293**:46-2.
3 Anonymous. Compensation: who cares? *Br Med J* 1982;**284**:1428-9.

Acute salpingitis

Acute salpingitis has become much commoner over the past decade, affecting particularly women aged 15 to 20.[1] The factors underlying this rise are sexually transmitted diseases, the use of intrauterine contraceptive devices, and infections associated with the termination of pregnancy. The infecting organisms have also changed, with chlamydia now being the commonest: it is implicated in almost two thirds of cases.[2-5] Gonorrhoea is still an important cause, with the gonococcus isolated in over half the cases.[2] Other organisms implicated include mycoplasma,[2] enterobacteria, and anaerobic bacteria. A further factor in the spread of acute salpingitis is carriage of bacteria by spermatozoa to the upper genital tract.[6]

Classically acute salpingitis presents with bilateral lower abdominal pain, fever, and purulent vaginal discharge. The lower abdomen may be tender and even rigid if peritonitis is present, with absent bowel sounds. There may be a purulent, offensive vaginal discharge, which is sometimes blood stained; cervical excitation; and pain and bilateral tenderness of the fornices with enlargement of the adnexae. These findings may be unreliable: Jacobson and Westrom showed that laparoscopy failed to confirm the clinical diagnosis of acute salpingitis in almost a quarter of cases.[7] In cases of doubt or when the condition fails to improve with antibiotic treatment laparoscopic confirmation of the diagnosis may be needed. This may show hyperaemia of the Fallopian tube, oedema, a purulent exudate, and possible evidence of previous tubal disease.

Other investigations include bacteriological culture of high vaginal swabs and cervical swabs, inserted directly into the media. The technique for taking chlamydial swabs must ensure that cells themselves are removed for culture. Estimations of antichlamydial IgG antibody may confirm the presence of chlamydial disease when the results of cultures have been negative, although a raised value does not necessarily indicate active disease.[2] Swabs may also be taken at laparoscopy, and an endometrial biopsy specimen for cytological studies may confirm the presence of acute salpingitis, although the appearances are not specific for a causative organism.[8]

Severely ill patients need admission to hospital with bed rest, antimicrobial therapy, and analgesia. High vaginal and cervical swabs should be taken and then antibiotic treatment given. As more than one organism is usually responsible for acute salpingitis several antimicrobials are often required, though some (such as doxycycline) should be avoided during pregnancy. A suitable regimen would be spectinomycin 4-6 g six hourly for 24 hours with doxycycline 100 mg twice daily for seven days and daily for a further 14 days, and rectal metronidazole 500 mg twice daily for 10 days. An alternative