Guidelines and clinical standards

EDITOR,—Professor Lilleyman's article on clinical standards touches on the trend towards guidelines.1 There is, understandably, no mention of variations in their quality. Although the risk of exposing to litigation those who do not follow guidelines may result in greater compliance, we also face the risk of being 'locked into' some inappropriate or unnecessary practices. One way of reducing this risk might be clearly to distinguish between areas in which there seems to be a compelling case for the suggested course of action and others where a personal or arbitrary stance is adopted.

One such example is advice from the British Committee on Standards in Haematology (BCSH) that the platelet count be raised to at least 50 x 10^9/l before lumbar puncture.2 This recommendation is based solely on the routine practice of the authors' hospitals (M E Murphy, personal communication). It may well be unnecessary in one large group of thrombocytopenic children — those with acute leukaemia at presentation. In our centre we have a policy to administer platelets to these children before lumbar puncture, yet have never seen a haemorrhagic complication. Is there a risk of clinically important haemorrhage in these children? Perhaps there is, and we have simply been fortunate over the last 20 years. What is missing is information to support the recommendation. No doubt others can think of examples of similarly questionable but didactic recommendations.

Of course, guidelines can be modified with experience (and the BCSH will look at this particular issue again) but one wonders if the process of modification may be simpler and more timely if areas of ignorance or doubt are not denied by the desire to provide an all encompassing impress of security and direction.

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Professor Lilleyman comments:
Dr Reid makes a good point with which I totally agree. There is, though, another aspect to the type of ex cathedra based guidelines he worries about, and that is whether anyone takes any notice of them — or even reads them. I would guess that few clinicians have changed their practice of giving or not giving platelet cover for lumbar punctures based on the reference he cites, and that few plan to.

But the fact that the recommendation exists could come back to haunt a clinician who faces litigation and who did not follow it. So I endorse the suggestion that guidelines should indicate areas of uncertainty. But I also believe that to be effective they should be backed by a system of external peer audit so that any concerns with them can be assessed. Guidelines drifting quietly into the literature mostly get filed or forgotten.

Pseudomonal rectovaginal abscesses in HIV infection

EDITOR,—Borgstein and Broadhead have interestingly described a series of nine cases of acquired rectovaginal fistula in children with presumed vertically acquired HIV infection.1 They suggest this may be caused by localised perianal sepsis. We would like to report a child with AIDS and severe perianal abscesses due to Pseudomonas aeruginosa infection.

A 6 month old patient presented with a rash, hepatosplenomegaly, and a severe bronchi-olitic illness associated with disseminated cytomegalovirus infection. HIV antibody, p24 antigen, and the polymerase chain reaction were all strongly positive. The cytomegalo-virus infection was successfully treated with intravenous ganciclovir, followed by continued maintenance via a Hickman line. Further illnesses included respiratory infections with respiratory syncytial virus and influenza C. At the age of 13 months the child was noted in the vulva. A further 2-3 cm perirectal abscess was noted on the same side. A swab of the discharge grew a heavy growth of P aeruginosa. This was treated with oral ciprofloxacin for 10 days, with complete resolution of both abscesses. There have been no further recurrences, and the child is alive now at 18 months of age.

Rectal and vulvovaginal abscesses are a major problem in immunosuppressed children without HIV infection, and are most commonly caused by anaerobic organisms.2 Frequent bacterial infections occur in children with HIV infection, including skin and perirectal abscess formation,3 with pseudomonal infections seen more commonly in children with AIDS. All nine cases reported by Borgstein and Broadhead fulfilled the clinical criteria for AIDS related complex/AIDS, and we suggest that the rectovaginal fistulas seen in these infants may have been secondary to pseudomonal infection.

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Acquired rectovaginal fistula

EDITOR,—We have seen examples in Zimbabwe of rectovaginal fistulae in HIV positive infants simi-lar to those described by Borgstein and Broadhead (see table).2 They were seen over a two year period; they and their mothers were HIV positive.

The fistulas occur just behind the fourchette in the vagina communicating with the rectum just above the levator muscles, the lowest point at which the vagina and rectum are most closely applied. In case 3 the histological findings described non-specific acute and chronic inflammatory tissue from the edge of the fistulae. No ‘owl’s eye’ intranuclear inclusions were seen, excluding cytomegalovirus as a cause.

An abnormally opening fistula in ano may be responsible, or ulceration breaking through the anterior wall of the anorectum into the vagina. Anal ulceration in adults may be caused by cytomegalovirus, cryptococcus, and herpes simplex. These ulcer are often indolent and run a protracted course. Chronic intersphincteric abscesses and fistulae also occur.4

All cases were managed by constructing a defunctioning sigmoid colostomy which has given symptomatic relief in all patients. Case 3 showed no evidence of the fistula healing. Death probably occurs fairly soon after the onset of the fistula as only one of the patients has come for regular follow up.

It may be possible to close the fistula surgically, although one would expect a high incidence of wound breakdown and of faecal fistula when the colostomy is closed.

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