CORRESPONDENCE

MIDAZOLAM AND CARDIOVERSION

Sir,—We read with interest the article by Fennelly and others [1], and wish to report our own experience, which is different in some respects. The main difference between their study and ours [2] was that the dose of midazolam we used was much greater in order to induce anaesthesia (and not sedation). Although benzodiazepines are known to cause antegrade amnesia, Krichbaum and Hamid [3] reported a few patients who were aware during cardioversion under midazolam sedation. Our own experience was that the anaesthesia produced by midazolam, in a much greater dose, was poor because many patients had flexion movements of the arms and legs and some needed to be restrained. Flumazenil clearly reversed the effect of midazolam, but many patients were resedated at the time of interview 4 h later. Also, the statement that this was “the first specific report of the use of midazolam, reversed by flumazenil, for anaesthesia for cardioversion” is incorrect, as both the above studies [2, 3] have examined midazolam sedation for cardioversion. Although the purpose of the present study was not to evaluate the usefulness of midazolam as an anaesthetic for cardioversion, we think that it is important for the reader to know that midazolam is not the best choice for this procedure, even though it can be reversed effectively by flumazenil.

A. Gupta
M. Vegors
C. Lenmarken
Linköping


DELAYED SEPTICAEMIA AFTER EXTRADURAL STEROID TREATMENT

Sir,—Further to the recent case report of a delayed complication after extradural steroid therapy [1], we wish to report a life-threatening event associated with extradural steroid administration.

A female of 54 yr was referred to the Pain Clinic for management of lower back pain which had been present for 9 months without improvement on conservative treatment. The pain radiated posteriorly to the left foot, was exacerbated by exercise and was restricting activity. The patient was generally well; she smoked 15 cigarettes per day, but denied respiratory symptoms. On examination, the single abnormal sign was limited straight leg raising on the left. Lumbar spine x-rays showed degenerative changes, particularly at the L4–5 region. A lumbar extradural injection of methyl prednisolone 120 mg (Depo-Medrone) was performed using a strict aseptic technique.

Nine days later, the patient began to notice a non-productive cough and became dyspnoic; rigors developed and the following day she was admitted to hospital, where she was found to have pyrexia (temperature 39.9 °C), tachycardia and mild neck stiffness. Clinical signs of consolidation in the right chest were confirmed by chest x-ray. She became hypotensive (systolic arterial pressure 70 mm Hg) and required resuscitation with i.v. fluids. A diagnosis of septicaemia secondary to a chest infection was made. Investigations revealed haemoglobin 134 g litre⁻¹, white blood cells 21.8 x 10⁹ litre⁻¹, with normal biochemistry; lumbar puncture was normal; blood and urine cultures subsequently were negative; no spurtum could be obtained. I.v. cefuroxime and erythromycin therapy was initiated, the patient’s condition improved rapidly and she was discharged home 6 days later.

We consider that this patient’s admission was precipitated by two factors: immunosuppression secondary to the steroid therapy and cigarette smoking. Steroids have been administered by the extradural route for 40 yr [2] and the technique is considered to be safe in experienced hands. Local adverse effects, particularly haemorrhage [3], abscesses [1, 4] and bacterial meningitis [5], are well described, but systemic effects are often not considered and two major reviews of extradural steroid therapy discussed septicaemia without the presence of extradural abscesses [6, 7].

Steroids depress the immune system via several routes [8]. Lymphopenia affecting particularly the T helper cells occurs as the lymphocytes are sequestered into bone marrow; the resting macrophages are inhibited because of reduction in complement and IgG receptors; antigen handling by macrophages is impaired, leading to a poor primary antibody response; neutrophils fail to be exported into tissues. The effects of “depot” preparations can be shown to persist for up to 3 weeks [9].

We suggest therefore, that any patient receiving a depot preparation of steroid should be advised to seek medical advice at the earliest symptom of possible infection, and that medical staff should be aware of the possibility of late septicaemia.

R. H. Elliott
B. J. Collett
Leicester


Sir,—We are grateful for the opportunity to comment on the letter of Elliott and Collett, in response to our report [1]. They report a case of septicaemia following extradural administration of steroids for the relief of back pain which is not comparable to our own. Ours was a thoracic administration, analgesies were administered and our patient developed a local septic complication. We would, however, take issue with the authors.

We would agree that smoking was a factor in the development of a chest infection in their patient, but dispute the importance of the extradural steroids. The small dose of methyl prednisolone administered (120 mg) into the extradural space would seem unlikely to be sufficient to cause generalized immunosuppression. Certainly, it is much smaller than that administered i.v. for therapeutic immunosuppression. In contrast, our patient was immunosuppressed by rheumatoid arthritis and the administration of long-term high-dose immunosuppressive drugs. This is seen in the white cell response to infection — 6.8 x 10⁹ litre⁻¹ in our patient, but 21.8 x 10⁹ litre⁻¹ in theirs.
STERIOD THERAPY AND EXTRADURAL ANALGESIA

SIR,—I read with interest the case report of Dr Sowter and colleagues [1] describing the delayed presentation of an extradural abscess in a patient with rheumatoid arthritis who had been receiving steroids, and the letter by McQuay and Jadad [2] who have criticized as premature the suggestion that extradural abscesses should be contraindicated in such patients. McQuay and Jadad base their criticism on the observation that this is but a single case; furthermore, they comment that there have been no cases of delayed extradural abscesses in patients receiving steroids by the extradural route for the management of chronic back pain and that there have been no reports of extradural abscesses in cancer patients taking oral steroids who receive extradural therapy.

In fact there have been reports of extradural abscesses occurring after administration of extradural steroids for back pain and an extradural abscess has occurred in a patient who was taking oral steroids but did not have cancer. Chan and Leung [3] described a diabetic patient who developed an extradural abscess after a single extradural injection of triamcinolone acetonide for sciatica. Similarly, Goucke and Grazioti [4] described a patient who developed an extradural abscess 3 weeks after having received three extradural injections of methylprednisolone and local anaesthetic for chronic back pain. Strong [5] described delayed presentation of an extradural abscess after placement of extradural catheters in two patients who received steroids; the first patient suffered from reflux sympathetic dystrophy and had been receiving oral prednisone. McQuay and Jadad also refer to a case of an iatrogenic extradural abscess that occurred in a patient 9 days after removal of an extradural catheter, suggesting Dr Sowter's paper to be the third report of extradural abscesses with delayed presentation. In fact there have been several other reports of iatrogenic extradural abscesses with delayed presentation which have occurred up to 5 months after placement of extradural catheters [5–7].

It is debatable if concurrent steroid use could be a contraindication to extradural therapy. However, it seems prudent to urge caution whenever a patient is receiving steroids or when steroids are to be administered extradurally. The risk and benefit must be considered, informed consent obtained, scrupulous aseptic technique, and the patient must be carefully followed up to check for signs of complications.

W. D. NGAN KEE
Wellington, New Zealand