Injuries to the chest could be managed by means of intercostal blocks. Thoracic epidural injections are likely to be hazardous.

Contraindications
Contraindications to local anaesthesia include sepsis or haematoma at the injection site, the need for excessively large doses of local anaesthetics, severe hypertension and untreated shock, absence of resuscitation equipment, and lack of expertise on the part of the operator. However, these techniques are all relatively simple and can be learned fairly easily.

Conclusion
In the disaster situation the use of regional anaesthesia utilizing minimal equipment offers excellent and prolonged relief of pain from the time of injury continuing into the postoperative period. The equipment required is not cumbersome and the techniques are easily mastered. It is suggested that use of these methods should be considered by all those involved in the management of disaster victims.

REFERENCES

Review Article
Magnesium deficiency provoked by diuretics

A. J. REYES, W. P. LEARY

Summary
Many diuretics cause hypermagnesiuria which may lead to magnesium deficiency, presenting as hypomagnesaemia, cardiac arrhythmias and tetany. Loop diuretics cause hypermagnesiuria mainly through direct blockade of magnesium reabsorption at the loop of Henle. Distal tubular diuretics block magnesium reabsorption at the distal convoluted tubule and also reduce magnesium reabsorption at the loop of Henle by an indirect mechanism.

Commonly used diuretics such as the thiazides, chlorothalidone and furosemide increase urinary magnesium losses. Deficiency of the ion develops after prolonged diuretic treatment, notably when other factors contributing to magnesium depletion are also present. Significant magnesium losses in association with diuresis may pass unrecognized because the signs and symptoms of magnesium depletion are usually attributed to potassium deficiency, which may also be present.

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Hypermagnesiuria provoked by diuretics
Urinary magnesium excretion is significantly increased in healthy adults by single therapeutic doses of diuretics which act principally in the loop of Henle (furosemide, piretanide) or in the first portion of the distal convoluted tubule (hydrochlorothiazide, xipamide, chlorothalidone). Magnesium depletion may complicate prolonged treatment with either type of diuretic.

Pathophysiology of magnesium deficiency provoked by loop diuretics
Approximately 70% of plasma magnesium is diffusible and therefore subject to filtration at the glomerulus. About half the filtered magnesium is reabsorbed in the thick limb of the loop of Henle, where diuretics such as furosemide, ethacrynic acid, bumetanide and piretanide appear to have a common receptor and exert their principal biochemical effects within the kidney. Loop diuretics inhibit magnesium reabsorption by a mechanism unrelated to the interference with chloride transport in the loop of Henle which primarily determines their natriuretic and diuretic effects.

Direct blockade of magnesium reabsorption within the loop of Henle might account for the hypermagnesiuria induced by loop diuretics, were it not for evidence that other processes are also involved. Mathematically derived curves describing sodium and magnesium flows in urine of healthy individuals given placebo show that the time courses of both excretions are parallel (Fig. 1). When the same individuals are given single therapeutic doses of furosemide, urinary magnesium flow is delayed with respect to sodium flow (Fig. 2). This strongly suggests that slow mechanisms, possibly endocrine in nature, may act in conjunction with
luted tubule is positively controlled by PTH,\textsuperscript{8,10} which would tend to diminish the urinary excretion of magnesium. When magnesium excretion exceeds intake an increasing mobilization of bone magnesium takes place, the magnesium balance becomes negative and deficiency may develop. This can be reflected by an early fall in the plasma magnesium level, or no change may occur before there has been a major decrease in the intracellular concentration of the ion affecting bone and other tissues.

Depletion of tissue magnesium inhibits the release of PTH, and, in addition, resistance to the action of this hormone develops limiting mobilization of bone calcium and magnesium and reduces the nephronal reabsorption of these ions.\textsuperscript{11,12} Hypocalcaemia develops as a result of these changes and tends to decrease magnesiuria slightly in a direct manner independent from PTH.\textsuperscript{16,18} The renal effect of PTH is not only diminished because sensitivity to the hormone is decreased but also, in the case of furosemide, because the diuretic blocks it directly.\textsuperscript{10} Loop diuretics dilute pre-urinary magnesium, and its reabsorption is thus further reduced.

Fig. 3. shows the changes following hypermagnesiuria provoked by loop diuretics, the mechanism whereby hypomagnesaemia is induced, aggravated, auto perpetuated and stabilized, and the relationship between these changes and calcium metabolism. The term hypomagnesaemia is used in the graph to denote magnesium deficiency.

Pathophysiology of magnesium deficiency provoked by distal tubular diuretics

Only 1-5\% of filtered magnesium is normally reabsorbed from the distal tubule. The hypermagnesuria provoked by distal tubule diuretics therefore cannot be explained solely on the basis of a direct blockade of magnesium transport across the nephronal wall at that level. This view is supported by experiments similar to those described for furosemide, in which it was found that an important dephasing exists between the curves expressing time-courses of excretion of sodium and magnesium after the administration of distal tubular diuretics such as hydrochlorothiazide,\textsuperscript{7} chlorthalidone\textsuperscript{4} and chlorexolone.\textsuperscript{6} This dephasing is more pro-
nounced than that which appears after the administration of loop diuretics and suggests that the hypermagnesiuria caused by distal tubule diuretics involves indirect mechanisms.

Some distal tubule diuretics, such as hydrochlorothiazide and zimamide (A. J. Reyes and W. P. Leary — unpublished data), provoke hypercalciuria for 24 hours following their initial administration but hypocalciuria thereafter if treatment is prolonged, whereas distal tubule diuretics, such as chlorothalidone and indapamide, cause hypocalciuria from the onset of treatment of healthy volunteers or patients with uncomplicated essential hypertension. Diuretics which provoke hypercalciuria initially cause less dephasing between the time-courses than those which reduce urinary calcium excretion from the onset of treatment. Hypercalciuric distal tubule diuretics may behave as loop diuretics initially, at least with respect to hypermagnesiuria during the first 24 hours of treatment, thereafter sharing indirect mechanisms with the other distal tubule diuretics. Hypocalciuria results in relative hypercalcaemia and decreased PTH release with diminished magnesium reabsorption at the loop of Henle. This process per se may explain the magnitude of the hypermagnesiuria caused by distal tubular diuretics. Other mechanisms are similar to those associated with the hypermagnesiuria of loop diuretics.

The potassium-sparing diuretic amiloride, which acts at the last portion of the distal convoluted tubule, does not provoke hypermagnesiuria (W. P. Leary, A. J. Reyes and K. van der Byl — unpublished data) because magnesium in the pre-urine is not transferred to the extracellular fluid at this site and because this diuretic does not affect the renal excretion of calcium to any significant degree.

### Magnesium deficiency

Circumstances other than treatment with diuretics may cause magnesium deficiency (Table 1). Some of these may also provoke potassium deficiency and may adversely affect patients on diuretic regimens. Magnesium deficiency seldom occurs as an isolated entity, although its consequences — notably its cardiac effects — may be fatal. Ventricular arrhythmias including ventricular fibrillation have been attributed to magnesium depletion, and deficiency of the ion increases the myocardial hypo-excitability induced by digitalis.

Other clinical manifestations of magnesium deficiency include the classic signs of tetany, which are usually attributed to calcium deficiency, dysphagia and haemolytic anaemia. Magnesium deficiency rarely occurs as an isolated entity, although its consequences — notably its cardiac effects — may be fatal. Ventricular arrhythmias including ventricular fibrillation have been attributed to magnesium depletion, and deficiency of the ion increases the myocardial hypo-excitability induced by digitalis.

### Multifactorial conditions

**Hungry bone syndrome**

**Hypoparathyroidism**

**Hypothyroidism**

**Excessive lactation**

**Malnutrition**

**Conventionally treated diabetic keto-acidosis**

**Phosphate deficiency**

**Protein malnutrition**

### REFERENCES


### TABLE 1. CONDITIONS WHICH MAY PRIMARILY CAUSE MAGNESIUM DEFICIENCY OR ACT AS PRECIPITATING OR AGGRAVATING FACTORS IN ITS DEVELOPMENT DURING DIURETIC TREATMENT

**Factors decreasing supply of magnesium to the internal environment**

**Dietary insufficiency**

- Low magnesium content in available foods
- Poor selection of foods

**Global intake of food inadequate to supply magnesium needs**

**Malabsorption syndromes**

**Extensive gut resection**

**Enteral and biliary fistulas**

**Factors which principally increase magnesium loss**

**Gastro-intestinal**

**Vomiting**

**Diarrhoea**

**Renal**

- Renal insufficiency with hypermagnesiuria
- Osmotic diuresis (glucose, mannitol, urea)
- Alcohol
- Medications inducing hypermagnesiuria
- Lisoplatin
- Gentamicin
- Cardiac glycosides
- Long-term parenteral fluid infusions

**Multifactorial conditions**

- **Hungry bone syndrome**
- **Hypoparathyroidism**
- **Hypothyroidism**
- **Excessive lactation**
- **Malnutrition**
- **Conventionally treated diabetic keto-acidosis**
- **Phosphate deficiency**
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