populations should heed that fact and should not imply that all patients (children with asthma, say) cause the general practitioner difficulties in management. The consultant sees many of our mistakes but few of our successes. The good consultant knows it.

The consultant sees the patient afresh, the mind uncluttered by repeated contact. The good consultant knows it is easy to diagnose myxoedema in a stranger and betrays no glee in the simple discovery.

Doctor-patient relationship

Most important of all, though, the good consultant realises that the doctor and his patient have a relationship going, a kind of continuity of care thing, a "mutual investment society" if you will. So the good consultant takes care not to upset that relationship when seeing the patient for the general practitioner. Not a word, not even a raised eyebrow at the choice of digoxin over diuretic, and certainly never a "My God. *Lassar's paste*. I thought that went out with the ark. Must have been what they were using when he was at medical school." The patient is in an anxiety induced state of heightened awareness when he sees the consultant; the merest hint of criticism is picked up and may dim his view of the doctor.

Patient and doctor can become estranged when the former is swallowed into the hospital system by too frequent follow up, and by cross referral to others into the "consultant circus syndrome." Often the letter says something like, "I have taken the liberty of referring him for a psychiatric opinion," and I have to say, if it seems to you like a liberty, don't take it. Night calls become nightmares if I haven't seen a sick man for years. The good consultant is aware too that doctors can collude by repeated referral and thus avoid responsibility for true caring for the patient. At other times he has a sixth sense telling him that sometimes the real reason for referral is because of strain in the patient's relationship with the general practitioner. The good consultant does not allow an adroit patient to play one doctor off against another, refusing to play "Let's you and him fight."

The good consultant has read Michael Balint and Eric Berne.

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Lesson of the Week

Compartment syndromes in unconscious patients: a simple aid to diagnosis

A C MACEY

Bilateral compartment syndromes occurred in an unconscious 13 year old girl. As diagnosis by clinical methods alone is difficult a simple, readily available manometric method was used to measure tissue pressures.

Case report

A 13 year old girl who had been knocked off her bicycle was admitted with a closed head injury such that she responded only to pain (score on the Glasgow coma scale 5 (E=1, V=1, M=3)) and fractures of the right clavicle, neck of right scapula, and both tibias and fibulas.

On arrival she was shocked and required resuscitation; the fractures were reduced and backslabs applied to her legs temporarily. After 24 hours leg perfusion and temperature were still subnormal. By 48 hours she was responding to some commands (score on the Glasgow coma scale 8), her core temperature and perfusion were normal, but both feet remained cool. Both legs were swollen and tense with no apparent skin circulation or capillary refilling.

Compartment syndromes may develop insidiously in unconscious patients up to 72 hours after injury

Tissue pressures in the peroneal and posterior compartments measured by Whitesides's needle technique¹ were 160 and 190 mm Hg, respectively, on the right and 130 and 120 mm Hg, respectively, on the left. Bilateral partial fibulectomies (subperiosteal) resulted in immediate clinical decompression of the legs and increased peripheral circulation. At follow up after one year her condition was satisfactory.

Discussion

A compartment syndrome occurs when increased pressure within a closed osteofascial space (muscle compartment) reduces capillary blood perfusion below that necessary for tissue viability.² The syndrome may develop insidiously up to 72 hours after injury, and monitoring should be maintained for at least this period.³ The success of treatment depends on early diagnosis. In Bradley's review 98% of the patients had established foot drop at diagnosis and fasciotomy resulted in complete recovery in only 13% of these—that is, clinical diagnosis alone allowed undetected, largely irreversible tissue ischaemia to progress and persist.⁴

Regional Orthopaedic Unit, Sligo, Ireland

A C MACEY, FRCS, FRCSI, orthopaedic registrar

Correspondence to: Mr A C Macey, Our Lady's Hospital for Sick Children, Crumlin, Dublin 12.

Top: Drawing saline into apparatus. Bottom: Manometer showing tissue pressure.

The symptoms and signs to be monitored are generally agreed to be pain, pain on stretching, paraesthesia, paresis, pulses, and pressure (by palpation). Their reliability in suspected cases is both supported and decried, but virtually all workers agree that in certain groups—namely, uncooperative or unreliable patients, unrespon-

sive patients, and patients with pre-existing neuropraxia—more objective methods of diagnosis are required.

Various techniques for measuring compartment pressures have been described, but only that of Whitesides uses equipment available in any ward. Readings with this technique are accurate to ± 3 mm Hg.

Whitesides's technique (figure)—Aspirate sufficient sterile saline into one arm of the extension tube to half fill it. Insert the 18 gauge needle into the compartment to be measured and turn the stopcock so that all arms are open. Increase the pressure in the system gradually by slowly depressing the plunger of the syringe while watching the column of saline. The manometer will rise, reflecting the pressure in the system. When this pressure has just surpassed the tissue pressure surrounding the needle a small amount of saline will be injected into the tissue and the saline column will move. The manometer reading at this point reflects the tissue pressure. Use of a wick or slit catheter instead of a green needle improves accuracy without reducing convenience.

The incidence of Volkmann's contracture after injuries to the limbs remains unacceptably high, suggesting missed or inaccurate diagnoses. These failures result in "a ruined limb for which surgery can offer only limited improvement."⁵ Thus the simple technique of Whitesides is a useful diagnostic aid that can be used in clinically complex cases, as that reported here. There is no single safe pressure, but 30-50 mm Hg is regarded as the range above which fasciotomy should be performed. The operative technique used should be appropriate to the pressure.

I thank Mr F T Shannon for permission to report on this patient and for his help in preparing my report.

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Is there any convincing evidence that aluminium hydroxide, taken in normal dosage as an antacid over months or years, can cause neurological damage?

There is good evidence that high aluminium concentrations in the brain are harmful and produce a dementing syndrome. This has been most clearly documented in patients with chronic renal failure who develop the syndrome of dialysis encephalopathy.¹ In such patients aluminium excretion is impaired because of renal failure and the aluminium accumulates from several sources. The first is tap water used during dialysis, and when this is deionised before preparing the dialysate the incidence of dialysis encephalopathy is greatly reduced. The second is aluminium hydroxide, commonly given to patients with chronic renal failure to control hyperphosphataemia. Indeed, there are reports of the syndrome occurring in patients with renal failure not dialysed but receiving aluminium hydroxide.² Hence it would appear that aluminium may accumulate in the body under conditions of high loading when renal function is impaired and this may be associated with neurological damage. The possibility that slow accumulation of minute quantities of aluminium might occur in individuals with normal renal function and become associated with neurological damage has been suggested. Evidence to support this theory has recently appeared after the finding of aluminium in the senile plaques that are one of the characteristic neuropathological findings in Alzheimer's disease (one of the commonest causes of dementia).³ There is no good evidence that the aluminium found in the brains of patients with Alzheimer's disease has produced the neuronal damage, though clearly this requires further study. Similarly, we need further information about the metabolism of aluminium in normal subjects. Present evidence suggests that only small quantities are absorbed from the gastrointestinal tract and that this is probably excreted readily in the presence of normal renal function. In short,

for the individual with normal renal function there is no good evidence that aluminium loading in the form of an antacid will lead to accumulation and neurological damage.—N E F CARTLIDGE, consultant neurologist and senior lecturer in neurology, Newcastle upon Tyne.

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Is postmenopausal osteoporosis inevitable? If not what proportion of women get it and is there any association with diet?

All vertebrate mammals lose bone with age, and in women the loss of bone starts at about the menopause. Symptoms due to osteoporosis depend on whether or not the residual bone mass reaches the "fracture threshold." Undoubtedly a small percentage of women (10%) are fast bone losers. By their 70s roughly half of women will develop spinal compression fractures. Nutrition—in particular, calcium intake—is an important determinant of bone mass in young adults. The effectiveness of calcium in reducing bone loss is not entirely settled and its action may be related to supplementation with oestrogens.—LESLIE KLENERMAN, consultant orthopaedic surgeon, London.

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