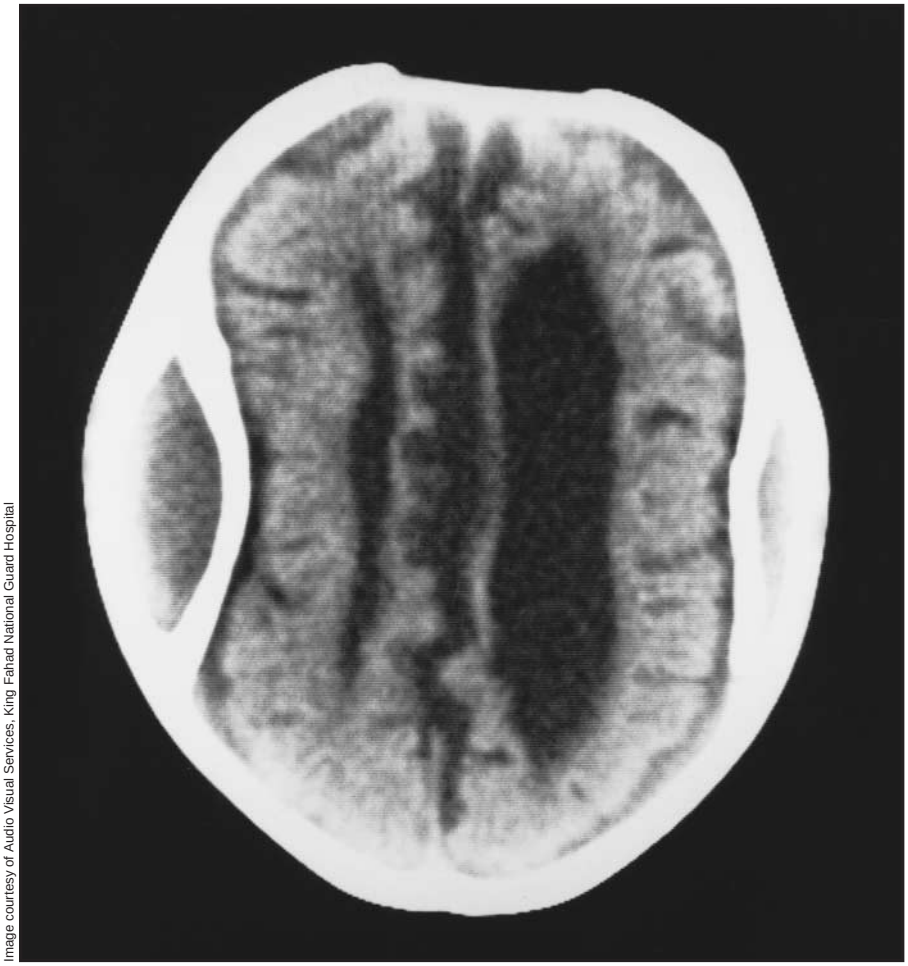
A baby with an armoured brain



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A male infant was born at term to a woman in her 20s after an uneventful pregnancy and uncomplicated vaginal delivery. He weighed 3500 g and had normal Apgar scores. A large, intact thoracolumbar myelomeningocele was present. There was no family history of neural tube defects, and no specific antenatal testing for these defects had been carried out. The myelomeningocele was associated with a complete sensorimotor paraplegia, absent anal tone, a neurogenic bladder and equinus deformities of the feet. A CT scan of the head performed within days after birth showed hydrocephalus associated with Chiari's malformation type II. At 5 days the myelomeningocele was repaired, and several days later a ventriculoperitoneal shunt was inserted.

At age 4 months the infant presented with a shunt infection, which was treated by removal of the shunt apparatus, external ventricular drainage and appropriate antibiotic therapy. At this time a CT scan showed no evidence of subdural hematomas. When the cerebrospinal fluid became sterile, a new ventriculoperitoneal shunt was inserted. At 10 months, the boy was asymptomatic with a functioning shunt. A CT scan, carried out to assess the ventricular size and the position of the shunt catheter, revealed bilateral subdural hematomas with dense calcification, particularly prominent within the inner membrane (Fig. 1). Because the boy was asymptomatic, surgery was not contemplated. He was followed regularly as an outpatient and continued to be well with normal development, but he was lost to follow-up at age 2 years.

Dense calcification and even ossification of a chronic subdural hematoma is rare but has been well documented.[1] When extensive, it has been likened to a shell or carapace, encasing the brain, thus the term "armoured brain."[2] The frequency with which radiologically demonstrable calcification occurs within the membranes of a chronic subdural hematoma has been reported to range from 0.3% to 2.7%.[3] However, Harwood-Nash[4] recorded a 10% incidence among children with chronic subdural hematomas studied by plain radiographs. Although usually seen in subdural hematomas resulting from trauma, it has also been reported in post-meningitic subdural effusions as well as subdural hematomas associated with ventricular shunting.[5] The latter condition may occur because of a partial collapse of the brain when the intraventricular pressure is reduced by the shunt; this collapse results in widening of the subdural space and stretching of the bridging veins, which thereby increases the risk of hemorrhage.[4] Although calcification of subdural hematomas seems more common in children, it has been reported in all age groups.[1] The interval between the occurrence of the hematoma and the development of the calcification varies from 6 months to many years.[1,5] Our patient had a normal CT scan at age 4 months; however, bilateral calcification was evident 6 months later.

The mechanism underlying the calcification remains unclear. Microscopic calcium deposits can often be found within the membranes of a chronic subdural hematoma, in its final stages of development.[1] In certain cases, this mineralization may progress to more extensive calcification and even ossification.[1,3] Etiological theories include poor circulation and delayed absorption of the hematoma fluid within the subdural space, which leads to stagna-

tion and eventually calcification.[3] In addition, an underlying metabolic abnormality such as an inherent tendency to calcification has been postulated.[1,5] The clinical presentation varies, from patients who are asymptomatic to those with symptoms and signs of raised intracranial pressure as well as seizures and mental retardation.[1,3,5]

Surgical removal of the calcification is difficult.[3] It should not be performed routinely; instead, it should be reserved for patients who have progressive neurological abnormalities or evidence of increased intracranial pressure.

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Too much of a good thing? Toxic effects of vitamin and mineral supplements

Reason for posting: Vitamin deficiency syndromes are uncommon in Western countries, but many patients consume over-the-counter vitamin and mineral supplements with the hope of improving their health and preventing disease.[1] Although the benefits of vitamin and mineral supplementation are commonly highlighted in both the professional and lay literature, their harmful effects often receive little attention. A recent review of 36 vitamins and minerals by the UK Food Standards Agency discussed the potential harms that can come from supplementation with some of these agents.[2]

The agents: For otherwise healthy individuals, daily consumption of a multivitamin is often recommended to round out a well-balanced diet.[3–5] Although some specific supplements are routinely recommended for disease prevention (e.g., folic acid for women of child-bearing age to prevent neural tube defects),[6] it is beyond the scope of this column to review the potential benefits of vitamin and mineral supplementation.

Scientific bodies around the world regularly review and recommend the daily vitamin and mineral intake levels (now expressed as Dietary Reference Intakes), taking into account age, sex, physiologic status (e.g., pregnancy) and concurrent disease states. Recommended intake levels are summarized in the purple pages of the *Compendium of Pharmaceuticals and Specialties*.[7] Consumption of doses at or near the recommended levels (as is often, but not necessarily, the case for multivitamins[8]) is unlikely to cause harm, and some vita-