this instance, development of myeloma was thought to be related to repeated exposure to intraoperative X-rays. Biological studies suggest that factors in the environment probably interact with genetic factors to increase myeloma risk. Case-control and cohort studies have identified exposure to ionizing radiation as an important environmental risk factor in the development of this disease (Morgan et al., 2002). Myeloma metastases often occur in the cervical spine, leading to upper limb pain. Spinal instability may also lead to root compression which may mimic compression syndromes. Fortunately, upper limb pain was not a problem in this case.

Treatment involved chemotherapy, radiotherapy and auto-bone marrow transplantation. The chemotherapy included vincristine. This induced so-called "vicristine neuropathy", which manifested as numbness in the hands. In a rat model, high doses of vincristine cause direct axonal toxicity (Silva et al., 2006), while low doses of the drug produce 'painful' peripheral neuropathy (Siau et al., 2006). The latter is of interest as it is associated with loss of epidermal innervation and an increase in PGP 9.5 in epidermal Langerhans cells. Neuropathic pain following complete and partial nerve transections is also associated with an increase in PGP 9.5 in epidermal Langerhans cells (Siau et al., 2006). My colleague experienced high dose toxicity which was only present while he was taking vincristine and the numbness continued for a few days after stopping it.

The disease recurred after the initial auto-bone marrow transplantation and he was offered thalidomide. He refused to take the drug after reading about its side effects, which include a dose-and duration-dependent effect on nerve function, with an irreversible neuropathy in about 40% of users of the drug. Thalidomide was banned worldwide in the 1960s when it was found to cause congenital anomalies, including phocomelia, when given during pregnancy. It has returned to the market for its unique effectiveness in multiple myeloma and other conditions such as leprosy and systemic lupus. Like the neuropathy of diabetes, thalidomide neuropathy is systemic in nature. This would suggest that it is not amenable to surgical intervention. However, Dellon and his colleagues have clearly demonstrated that thalidomide- and diabetes-induced neuropathies may benefit from nerve decompression (Rose et al., 2006). In both pathologies, electrodiagnostic testing may not detect mild compression neuropathies because of the underlying systemic neuropathy. Therefore, the indication for surgical decompression is usually based on a positive Tinel-Hoffman sign. Following nerve decompression, significant improvement of sensibility is noted (Rose et al., 2006).

After further treatment by allo-bone marrow transplantation, my colleague developed a chronic graft-versus-host reaction, that is the graft of new blood rejected the host organs, including any residual myeloma cells. This reaction is desirable to control any residual disease but can have undesirable effects if it is aggravated. He did not know that this reaction can be "photosensitive". During the first summer after the transplant, he developed acute carpal tunnel syndrome which arose as a result of acute autoimmune tenosynovitis secondary to sun exposure. Fortunately, the symptoms resolved quickly with high dose oral steroid treatment for a few days. He now has to avoid prolonged sun exposure for the rest of his life and is maintained on a low dose of prednisone to control the graft-versus-host reaction.

Although he has experienced no further complications and, after 5 years, is now back to full practice with no problems, one further hand complication is not uncommon with this disease and should be mentioned. Amyloidosis may occur secondary to multiple myeloma and precipitate carpal tunnel syndrome by laying down deposits in the carpal canal (Kelly and Moran, 2005).

I hope this experience is worth documenting in this journal. The extraordinary number of hand sequelae of this condition and its management might appear to be divine retribution when the patient was, himself, a hand surgeon. Hopefully, this case may act as an aid to memory of the interactions of this seemingly remote condition to our speciality.

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Aplasia of the Flexor Digitorum Profundus Tendon of the Small Finger

Dear Sir,

A 9 year-old boy presented with absence of active flexion of the distal interphalangel joint of the right small finger. His mother did not know if he had ever been able to bend this finger fully and no one in the

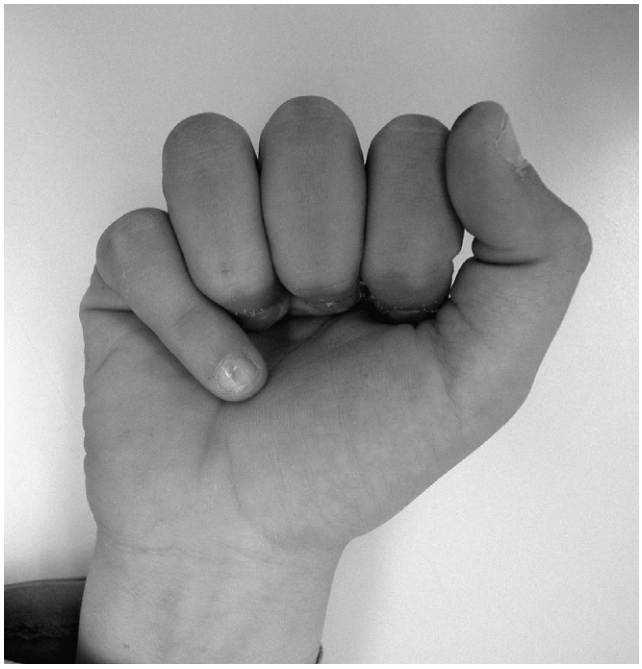

Fig 1 Absence of active flexion of the distal interphalangeal joint of the small finger.

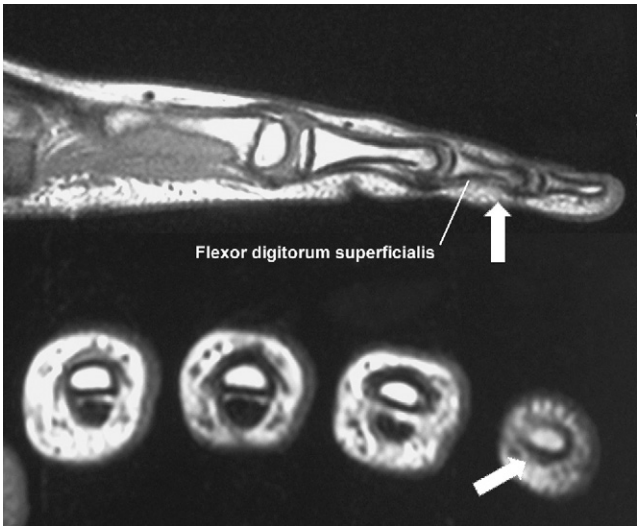

Fig 2 MRI of the small finger; the arrows indicate the missing deep flexor tendon. Authors:

family remembered an injury. The child, himself, did not report any pain or loss of function. Neither his younger sister nor his parents showed this functional impairment. The whole finger appeared to be slightly hypoplastic compared to the contralateral little finger. When asked to make a fist, the boy managed, skilfully to hide the functional loss by flexing his distal phalanx using the neighbouring finger. However, without this support, the loss of flexion of the terminal phalanx was clearly apparent (Fig 1). The function and strength of flexor digitorum superficialis, the lumbricals and the extensor apparatus were normal. Sensibility and circulation were normal. Plain X-ray showed a hypoplasia of the metacarpal and phalangeal bones with a 5 mm shortening of this finger. Ultrasound demonstrated that the flexor digitorum superficialis tendon was present but the flexor digitorum profundus tendon was completely absent. MRI (Fig 2) confirmed the ultrasound findings and did not detect a proximal tendon stump in the hand or distal forearm.

Anomalies of the profundus flexor tendons are rare. A congenital abnormality of the flexor digitorum profundus with a flexion contracture of the distal interphalangeal joint was published by O'Brien and Hodgson (1974) and anomalous accessory flexor digiti minimi profundus muscles were reported by Wahba et al. (1998). To our knowledge, Frohse was the first to mention absence of the superficial and profundus flexor tendons in "Handbuch der Anatomie des Menschen", in 1908 (Frohse and Fränkel, 1908). Another case of an absent flexor digitorum profundus tendon of the index finger was described more recently (Kay and Lees, 2000) in a child who was treated by two-stage palmaris longus grafting to achieve a full range of motion of the digit, so improving pinch and key grip. Because our patient had almost no functional limitations, we did not treat the defect. Should intervention be necessary, two-stage tendon grafting, using the first operation to create the space and pulleys of a tendon sheath would seem to be logical.

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