the variability between years, a 3 year aggregate rate was also calculated for each group (Table 2). The standard deviation and 95% confidence intervals were calculated for each group. Data for the years 1973-5[23] was age standardised and a rate of fracture per 100 000 of population was calculated for each group. The standard deviation and 95% confidence intervals were calculated for each group (Table 2). This earlier data was then compared with our data to see if there had been an increase in the hip fracture rate.

Results

The age standardised rates of hip fracture were lower for Maori than nonMaori (Table 2). The difference was particularly marked in the female population. NonMaori females had the highest fracture rate, and were higher than all other groups including Maori females. Maori females had a higher fracture rate than nonMaori males, but nonMaori males had a higher rate than Maori males. When compared with earlier data,[14] the age specific fracture rate was noted to have increased in all groups except for Maori males.

Overall there has been a 54% increase in hip fracture rates in Maori and a 59% increase in nonMaori between 1973-5 and 1989-91.

Discussion

The incidence of hip fracture has been found to be high in northern European women,[13,19] and in North American white women.[4,3,6,7] The hip fracture rates in nonMaori women in New Zealand are at similar levels. In contrast North American blacks, Hispanics, Asians and white men have lower rates.[4-7] The Bantu of South Africa have also been shown to have low hip fracture rates while white South Africans have similar rates to North Americans and Europeans.[9,20] A study of Japanese living in Japan and in Hawaii shows that these groups have hip fracture rates half that of caucasians living in North America.[10] However there was no significant difference between the rates of Japanese living in Japan and Hawaii suggesting that genetic factors are of more importance than environmental factors.

Our study has shown that nonMaori women who are predominately a caucasian population have high fracture rates which are similar to those for North American and European women. The hip fracture rates are significantly lower for Maori women. We have also found that Maori males had a significantly lower rate than nonMaori males.

The reason for these differences is unknown. There is obviously a genetic difference between the populations. There has been no published data recording the levels of osteoporosis in these two populations. Bone density has been found to be greater in blacks in the United States than in the white population, however Asians appear to have a lower bone mass than whites. Yet both Asians and blacks have lower fracture rates than whites indicating that factors other than osteoporosis may be important.[21] There does not appear to be any significant difference in diet, physical activity and exposure to sunlight in the two groups in New Zealand to explain the different fracture rates. This probably indicates that genetic factors are the most important.

It has been shown that age-specific hip fracture rates are rising in New Zealand and other countries.[1,11-13,22] In comparing our data with that of 1973-5[14] there has been a rise in all groups except Maori males. However it appears that the factors causing an increase in the rates over time are affecting the Maori group almost as much as the nonMaori group.

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Unrecognised arterial injury of the forearm: presenting as acute compartment syndrome.

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Although injury to the brachial artery is not uncommon in cases of elbow trauma, injury to the ulnar artery is rare, and is usually associated with ulnar nerve injury. This paper presents a patient with an isolated injury to the ulnar artery leading on to compartment syndrome which remained unrecognised for 2 days.

The patient

A 24 year old woman was pushed by her husband through a glass window in October 1994. She presented to the casualty department with a small laceration over middle of medial aspect of the right forearm. As the wound was small (0.5 cm) and an x-ray did not reveal any foreign body, the casualty officer cleaned the wound and discharged the patient home with analgesics.

Next day, the patient returned to the casualty department with a deterioration of pain. This time she was seen by senior house officer and assessment showed a swollen tense forearm, but pulses and colour of the skin were normal. There was hypaesthesia in the middle and index fingers. Pain was worse on passive movement of wrist. Capillary circulation was good. The doctor documented that a compartment syndrome was unlikely and he treated the patient with stronger analgesics.

24 hours later, the pain was worse, and she was seen by yet another doctor in the casualty department who confirmed the findings recorded earlier. However, he was not sure whether patient was a malingerer and decided to discuss with the registrar.

On examination the patient's forearm flexor compartment was swollen, tense and tender and the fingers and thumb were held in a flexed position. There was blunting of sensation in the distribution of the median nerve. Passive extension of her fingers

was not possible, as it produced severe pain in the forearm. The capillary circulation to the hand was normal and radial pulse was palpable. The wound, 0.5 cm in the middle of the forearm over medial aspect looked satisfactory.

Under general anaesthesia, emergency fasciotomy of both deep and superficial anterior compartments was performed through a longitudinal incision from the elbow to the wrist. The deep compartment contained a large haematoma extending in to the wrist. Parts of flexor digitorum profundus and flexor pollices longus were necrotic and were excised. The ulnar artery in the midforearm was badly lacerated with loss about 90% circumference for about 3 cm. This was ligated. The median nerve was decompressed throughout its length and the carpal tunnel was released. The wound was left open and was closed after 4 days. No growth detected on culture.

On review 6 weeks later she had normal muscle power. Sensation in the distribution of median nerve was normal.

Discussion

Compartment syndrome is a relatively common condition that can cause serious limb- or life-threatening consequences. Often a junior doctor in the emergency department is the first to examine and diagnose this orthopedic emergency.

It is important to draw the distinction between the classical signs of an acute vascular injury and the classical signs of an acute compartment syndrome. The former is described by the four Ps — Pain, Pallor, Pulselessness and Paralysis — and would indicate injury to the brachial artery or proximal arteries. A rich collateral supply would prevent acute vascular insufficiency resulting from distal injury, such as to the ulnar artery.

In the present report, junior doctors failed to make a diagnosis of compartment syndrome as three out of four signs of vascular injury were absent. It is my contention that a compartment syndrome is better described by the 4 Ss — severe pain (out of proportion to the clinical situation), stretch pain, sensory abnormalities, swollen and tense muscles. The presence of a distal pulse does not exclude the diagnosis. This is because the critical closing pressure of an artery is higher than the capillary perfusion pressure.[1]

In the present case, the initial wound was only 0.5 cm. However small the wound is it should be explored to its full extent,[2] particularly if there are physical signs that suggest involvement of neurovascular structures.

This presentation of compartment syndrome is rare. One such delayed presentation has been reported[2] wherein damage to radial artery remained unrecognised for 3 days, when a sudden secondary bleed caused an acute compartment syndrome of the forearm.

The diagnosis of this potentially serious complication depends upon a high index of clinical suspicion and once diagnosed, decompression of the compartment should not be further delayed.[3]

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LETTERS

Letters to the editor should be signed by all authors, typewritten in doublespacing, not exceed 500 words and 10 references. References should be in the Vancouver style. Over long letters may be shortened without reference to the author unless it is specifically stated otherwise. Priority of publication may be given to short letters.

Immunisation rates of infants

The article by Essex et al (NZ Med J 1995; 108: 244-6) claims that the immunisation rates in a cohort of infants studied at age 1 and 2 years are 92.5% and 83.3%. These figures are significantly better than those reported by the CDC survey of 56.6% and 61% respectively. A significant feature of this reported study is the high drop out rate from 4286 to 2827, a 66% retention rate. In calculating the immunisation rates the assumption has been made that the immunisation rate amongst those who dropped out of the study would have been the same as those who remained in the study.

The arguments to support this assumption are flimsy. It is our experience that it is precisely those children who are lost to follow up by Plunket who are most likely to miss out on immunisations. These are the children who move house frequently, who are not brought for routine checks, and who move around the country.

If the assumption is made that all children lost to follow up did not get immunised then the rates would be 78% and 55% at age 1 and 2 years respectively. The true rate is likely to fall somewhere between these levels and those calculated in the paper.

I believe that their assertion that the immunisation rates are 92.5% and 83.3% at age 1 and 2 years is not supported by the evidence that they present.

It is disturbing enough that the Plunket Society which is contracted to provide well child health care for most of our children fails to do this for 34% of children at age 2.

It is even more disturbing that Essex and Geddes believe that those children that Plunket fail to follow up some how or other achieve the same immunisation rate. Do they really believe that having Plunket follow up makes no difference to whether a child is immunised?

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Breast cancer screening

Recently a decision in principle has been taken to set up a national breast cancer screening programme in New Zealand, following much discussion and preliminary results from the pilot programmes.[1] Accordingly, many readers will be interested in a recent Lancet paper[2] which argues that public funding for breast cancer screening is not justifiable. This article has serious deficiencies, both in the context of mammography in general, and specifically in relationship to New Zealand programmes.

Wright and Mueller comment on four major aspects. First, they claim that the benefits seen in early trials of mammography are not maintained in subsequent trials. This results from an analysis in which they confuse the size of an effect with its statistical significance, and in which they use data for including women under age 50 in whom no clear benefit of mammography has been seen in most studies.[3] This is despite the fact that they themselves accept this lack of benefit and state that "the issue of screening women in that age group does not warrant further discussion". They also omit one of the major trials, but include the Canadian trial, which for women over 50 is not a trial of screening, but a comparison between two different modalities of screening.

Second, they consider the absolute benefits of mammography, using rather superficial analyses on outdated data. Their main analysis is based on the Swedish trial quoting a 1985 publication, and they conclude that there are four deaths averted per 10 000 women screened. One can compare this to an up to date publication from that study itself,[4] which gives a result of 19 deaths averted per 1000 women screened in the age group 50-64, and a somewhat higher result for older women.

Third, under the issue of the harm caused, they claim that many women come to open biopsy, and that there are high levels of public fear and anxiety. In the current New Zealand pilot programmes, for every 1000 women screened, about 70 will require further investigation, of which, indeed, only seven will be diagnosed as breast cancer. But of the remaining 63, the further investigations required consist in the great majority (80%) of one visit to an assessment centre; our data demonstrate that this takes less than an hour, is accompanied only by low levels of anxiety comparable to that of the screening itself, and does not require invasive procedures. Very few women proceed to open biopsy; the ratio of benign to malignant biopsies is close to 1:1. It is likely again that these authors' conclusions are based on including younger women, and using North American