5 European Working Party. Streptokinase in recent myocardial infarction: a controlled multicentre trial. *Br Med J* 1971;iii:325-31.
6 Bett JHN, Biggs JC, Castaldi PA, *et al.* Australian multicentre trial of streptokinase in acute myocardial infarction. *Lancet* 1973;i:57-60.
7 Aber CP, Bass NM, Berry CL, *et al.* Streptokinase in acute myocardial infarction: a controlled multicentre study in the United Kingdom. *Br Med J* 1976;ii:1100-4.
8 Rentrop KP, Blanke H, Karsch KR, *et al.* Acute myocardial infarction: intracoronary application of nitroglycerin and streptokinase. *Clin Cardiol* 1979;**2**:354-63.
9 Fioretti P, Simoons ML, Serruys PW, van den Brand M, Fels PW, Hugenholtz PG. Clinical course after attempted thrombolysis in myocardial infarction. *Eur Heart J* 1982;**3**:422-32.
10 Khaja F, Walton JA, Brymer JF, *et al.* Intracoronary fibrinolytic therapy in acute myocardial infarction. Report of a prospective randomised trial. *N Engl J Med* 1983;**308**:1305-11.
11 Kennedy JW, Ritchie JL, Davis KB, Fritz JK. Western Washington randomised trial of intracoronary streptokinase in acute myocardial infarction. *N Engl J Med* 1983;**309**:1477-82.
12 Yusef S, Collins R, Peto R, *et al.* Intravenous and intracoronary fibrinolytic therapy in acute myocardial infarction: overview of results on mortality, reinfarction and side-effects from 33 randomised controlled trials. *Eur Heart J* 1985;**6**:556-85.
13 TIMI Study Group. Special report. The thrombolysis in myocardial infarction (TIMI) trial. Phase 1 findings. *N Engl J Med* 1985;**312**:932-6.
14 Verstraete M, Bernard R, Bory M, *et al.* Randomised trial of intravenous recombinant tissue-type plasminogen activator versus intravenous streptokinase in acute myocardial infarction. *Lancet* 1985;i:842-7.
15 Gurewich V, Pannell R, Louie S, Kelley P, Suddith RL, Greenlee R. Effective and fibrin-specific clot lysis by a zymogen precursor form of urokinase (pro-urokinase). *J Clin Invest* 1984;**73**:1731-9.
16 Been M, de Bono DP, Muir AL, Boulton FE, Hillis WS, Hornung R. Coronary thrombolysis with intravenous anisolylated plasminogen streptokinase complex BRL 26921. *Br Heart J* 1985;**53**:253-9.
17 Norris RM, Brandt PWT, Caughey DE, Lee AJ, Scott PJ. A new coronary prognostic index. *Lancet* 1969;i:274-8.
18 Yusuf S, Ramsdale D, Peto R, *et al.* Early intravenous atenolol treatment in suspected acute myocardial infarction. *Lancet* 1980;i:273-6.
19 Hampton JR, Gorlin R. Platelet studies in patients with coronary artery disease and in their relatives. *Br Heart J* 1972;**34**:465-71.
20 Silver MD, Baroldi G, Mariani F. The relationship between acute occlusive coronary thrombi and myocardial infarction studied in 100 consecutive patients. *Circulation* 1980;**61**:219-27.
21 Tiefenbrunn AJ, Sobel BE. Tissue-type plasminogen activator (t-PA): an agent with promise for selective thrombolysis. *Int J Cardiol* 1985;7:82-6.
22 Schroder R, Biamino G, Leitner EV, *et al.* Intravenous short-term infusion of streptokinase in acute myocardial infarction. *Circulation* 1983;**67**:536-48.
23 Gruppo Italiano per lo studio della streptochinasi nell'infarto miocardico (GISSI). Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. *Lancet* 1986;i:397-402.
24 Simoons ML, Serruys PW, vd Brand M, *et al.* Improved survival after early thrombolysis in acute myocardial infarction. *Lancet* 1985;ii:578-82.
25 Smith RAG, Dupe RJ, English PD, Green J. Fibrinolysis with acyl-enzymes: a new approach to thrombolytic therapy. *Nature* 1981;**290**:505-8.
26 Been M, de Bono DP, Muir AL, *et al.* Clinical effects and kinetic properties of intravenous APSAC—anisoylated plasminogen-streptokinase activator complex (BRL 26921) in acute myocardial infarction. *Int J Cardiol* (in press).
27 Ganz W, Geft I, Shah PK, *et al.* Intravenous streptokinase in evolving acute myocardial infarction. *Am J Cardiol* 1984;**53**:1209-16.
28 Davies GJ, Chierchia S, Maseri A. Prevention of myocardial infarction by very early treatment with intracoronary streptokinase. Some clinical observations. *N Engl J Med* 1984;**311**:1488-92.
29 Reimer KA, Lowe JE, Rasmussen MM, Jennings RB. The wavefront phenomenon of ischemic cell death 1. Myocardial infarct size vs duration of coronary occlusion in dogs. *Circulation* 1977;**56**:786-94.
30 Erhardt LR, Unge G, Boman G. Formation of coronary arterial thrombi in relation to onset of necrosis in acute myocardial infarction in man. A clinical and autoradiographic study. *Am Heart J* 1976;**91**:592-8.
31 O'Doherty M, Tayler DI, Quinn E, Vincent R, Chamberlain DA. Five hundred patients with myocardial infarction monitored within one hour of symptoms. *Br Med J* 1983;**286**:1405-8.
32 Vincent R, Martin B, Williams G, Quinn E, Robertson, G, Chamberlain DA. A community training scheme in cardiopulmonary resuscitation. *Br Med J* 1984;**288**:617-20.
33 Schroder R, Vohringer H, Linderer T, Biamino G, Bruggemann T, Leitner EV. Follow-up after coronary arterial reperfusion with intravenous streptokinase in relation to residual myocardial infarct artery narrowings. *Am J Cardiol* 1985;55:313-7.
34 Kennedy JW, Ritchie JL, Davis KB, Stadius ML, Maynard C, Fritz JK. The Western Washington randomized trial of intracoronary streptokinase in acute myocardial infarction. A 12-month follow-up report. *N Engl J Med* 1985;**312**:1073-8.

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SHORT REPORTS

Boys with late descending testes: the source of patients with "retractile" testes undergoing orchidopexy?

The incidence of cryptorchidism, with its associated increased risk of subfertility and testicular malignancy,[1 2] seems to be increasing.[3] Scorer and Farrington carried out a study of the incidence of cryptorchidism in the late 1950s.[1] Any boy found to be cryptorchid at birth was examined at 3 months of age and, if still cryptorchid then, at 1 year of age. Those boys whose testes were descended either at birth or at 3 months were not re-examined. Only if the testes were still undescended at the age of 1 was the boy considered to be truly cryptorchid; this approach has been generally accepted by paediatric surgeons.

In their study of a group of boys born in the mid to late 1950s Scorer and Farrington found that 0·8% were truly cryptorchid.[1] Data from the Hospital Inpatient Enquiry, however, show that about 1·9% of the same cohort of boys underwent orchidopexy.[3] It has been argued that the additional boys undergoing orchidopexy represent those who were misdiagnosed cases of retractile testes.[4] We believe that we have identified the major source of these cases.

Patients and methods

As part of a long term prospective study all boys born in the John Radcliffe Hospital, Oxford, are examined at birth. Boys who are cryptorchid are re-examined at 3 months of age or, in the case of premature infants, 3 months after their expected date of delivery. A cryptorchid testis is defined as one where the centre of the testicle is less than 4 cm below the pubic tubercle (2·5 cm for babies weighing less than 2500 g) when measured at its lowest point after manipulation but without applying tension.[1] All cryptorchid testes are then classified as either non-scrotal or scrotal. A non-scrotal cryptorchid testis is one which is always found outside the scrotum and cannot be manipulated into the scrotum without tension. A scrotal cryptorchid testis is one which may not be found in the scrotum but can be manipulated into the scrotum without tension, although it remains less than 4 cm below the pubic tubercle.

Results

We examined at around 1 year of age the first 45 boys (who had not moved out of the area) whose testes were descended at the 3 month examination but not at birth—described as late descenders—and a group of 20 control babies whose testes were descended at birth.

The testes of all the 20 control babies were still descended at 1 year. In contrast, of the 45 late descenders, 18 (40%) had a cryptorchid testis at 1 year; the least descended testis was in a non-scrotal position in 14 (78%) of these 18 boys. A high proportion (29 (64%)) of the late descenders had had bilateral undescended testes at birth, and these boys had a slightly decreased risk of being cryptorchid at 1 year (10/29 (34%) compared with 8/16 (50%)). We also recorded the presence of other anatomical abnormalities in these boys and found a hydrocele either at birth or at 3 months in seven (39%) of the 18 late descenders who had a cryptorchid testis at 1 year, compared with five (19%) of the 27 who did not.

Comment

These late descenders whose testes then appear to develop true cryptorchidism probably explain the discrepancy between the 0·8% of boys identified with a true cryptorchidism in 1960 and the 1·9% who underwent orchidopexy.[1 3]

The term retractile testis describes a testis that is drawn up into the superficial inguinal pouch by the cremasteric muscle. We believe that this term is not appropriate for the late descended testes which are then found to be cryptorchid at 1 year. These testes are normally outside the scrotum and can be manipulated only with great difficulty into the scrotum under tension.

A possible explanation for this finding is that cryptorchidism may be acquired after birth by the resorption of an occult inguinal hernia.[5] Although we found no frank hernias in any of these 45 boys, a hydrocele was found in high proportion of the group with late descending testes that were cryptorchid at 1 year.

Screening between birth and school age in Britain is designed to detect all cases of undescended testis. Our results suggest than any baby whose testes are not descended at birth should be examined again at 3 months and again at 1 year. Whether a late descending testis that becomes cryptorchid again requires treatment is not known.

PA and AP re-examined the babies at 12 months. This report was prepared by PA, MBJ, MCP, and CC.

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1 Scorer CG, Farrington GH. *Congenital deformities of the testis and epididymis*. London: Butterworths, 1971:15-27.
2 Chilvers C, Dudley NE, Gough MH, Jackson MB, Pike MC. Undescended testis: and malignancy. *J Pediatr Surg* (in press).

3 Chilvers C, Pike MC, Forman D, Fogelman K, Wadsworth MEJ. Apparent doubling of frequency of undescended testis in England and Wales in 1962-81. *Lancet* 1984:330-2.
4 Cooper BJ, Little TM. Orchidopexy: theory and practice. *Br Med J* 1985;**291**::706-7.
5 Atwell JD. Ascent of the testis: fact or fiction. *Br J Urol* 1985;**57**:474-7.

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Effect of rapid referral on thickness of melanomas

The prognosis of patients with malignant melanoma is made worse by delay in seeking help.[1] Therefore, in 1981 a pigmented lesion clinic was started in Southampton with the object of minimising the delay between patients presenting to their general practitioner with a malignant melanoma and the melanoma being excised in hospital. The patients' prognosis was determined by the accepted method of measuring tumour thickness.

Patients, methods, and results

When the clinic was established general practitioners were sent a colour pamphlet of the common pigmented lesions and encouraged to use the clinic as a rapid referral service for patients suspected of having a melanoma. The clinic was held once a week, and all patients referred that week with pigmented lesions were examined by a dermatologist whether or not their doctor had requested an urgent referral. The dermatologist screened out the benign lesions, and the potential melanomas were excised the same day.

During the next three years nine months 1230 patients with pigmented lesions were examined. Most had benign lesions, but some 7% had basal cell carcinomas and 75 melanomas were excised. The ratio of women to men with melanoma was 1·8:1 and the most common age of presentation 50-60. About 70% of the patients had blue or green eyes or burnt before tanning when exposed to sunshine, and 23% had lived in or near the tropics for more than one year. Twenty eight per cent had presented to their general practitioner within three months of first noticing their tumour, 38% had waited three to six months, 16% had waited six to 12 months, and 18% had delayed for more than one year. Forty eight per cent of the melanomas were on the leg, 20% on the trunk, 15% on the arms, 8% on the face, and 9% on the palms, soles, and genitals.

At the end of four years the histological findings were reviewed and the slides coded to enable tumour thickness to be measured without bias. The results (table) were analysed by non-parametric statistical methods. The melanomas excised in the pigmented lesion clinic were not significantly thinner than those excised in Southampton during the four years before the clinic started or those excised during the study period by other specialists in Southampton. Nor were there significantly more thin, good prognosis melanomas excised in the pigmented lesion clinic.

Mean thickness of melanomas excised by various agencies in Southampton during 1976-85

	No	Thickness (mm) Mean	SD
Melanomas excised after referral to pigmented lesion clinic 1981-5	75	2·42	2·57
Melanomas excised by other specialists in Southampton 1981-5	45	3·19	2·75
Melanomas excised in Southampton 1976-80	129	3·06	3·13

Comment

The results suggest that minimising the delay between the patient presenting to the general practitioner and the excision of the tumour in hospital does not improve the prognosis of malignant melanoma.

The prognosis of patients with melanoma examined in the pigmented lesion clinic was similar to the 62% five year survival rate found in a larger study in Scotland.[2] The aetiological role of sunlight in melanomas occurring in Britain was emphasised by the finding that most of the patients with melanoma examined in the pigmented lesion clinic were fair skinned, many of the lesions were on parts of the body habitually exposed to sunlight, and that one in five patients had lived in tropical or near tropical countries for more than one year.

We conclude that the prognosis of malignant melanoma in Britain is not significantly improved by rapid referral to hospital. Educating the public that enlarging pigmented lesions may be malignant and that early medical help can save lives may be more successful. This has been successful in Australia,[3] and a recent study from Glasgow suggests that a similar campaign would be successful in Britain.[4] The need for such preventive medicine in Britain will increase as the incidence of melanoma rises inexorably.[5]

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1 Pack GT, Gerber DM, Scharnagel IM. End results in the treatment of malignant melanoma; report of 1190 cases. *Ann Surg* 1952;**136**:905-11.
2 Mackie RM, Smyth JF, Soutar DS, *et al.* Malignant melanoma in Scotland 1979-1983. *Lancet* 1985;ii:859-62.
3 Balch CM, Soong SJ, Milton GW, *et al.* Changing trends in cutaneous melanoma over a quarter century in Alabama, USA, and New South Wales, Australia. *Cancer* 1983;**52**:1748-53.
4 Doherty VR, Mackie RM. Reasons for poor prognosis in British patients with cutaneous malignant melanoma. *Br Med J* 1986;**292**:987-9.
5 Office of Population Censuses and Surveys. *Cancer statistics: registrations*. London: HMSO, 1985.

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The neuropathy of the critically ill

An acute and often severe axonal neuropathy occurring at the peak of serious illnesses, with various causes but all complicated by shock and sepsis, has recently been recognised.[1] The only series reported has been from an intensive therapy unit in Canada, suggesting that this problem is overlooked elsewhere.[2,3] We report a further case to bring this complication to the attention of general physicians and surgeons caring for patients affected by it.

Case report

A 54 year old woman was admitted to the intensive therapy unit at this hospital in December 1985, having collapsed with abdominal pain. Her blood pressure was unrecordable. Pancreatitis was diagnosed when her serum amylase activity was found to be over 4000 IU/l. Secondary septicaemia was diagnosed when blood cultures grew *Escherichia coli*. She was treated with fluid replacement, infusions of dopamine and noradrenaline, and penicillin and gentamicin. She developed the adult respiratory distress syndrome and required ventilation. Apart from hypoxaemia, metabolic derangements included a moderate degree of renal failure, hyponatraemia, and later hypernatraemia, and for a while she had a high blood glucose concentration of around 25 mmol/l (450 mg/100 ml).

By the tenth day in hospital she was thought to be less responsive than her condition warranted, with no movement having been observed in her limbs. An electroencephalogram showed some generalised disturbances consistent with a metabolic encephalopathy. A few days later a neurological opinion was sought. She was now blinking in response to visual threat and trying to talk. She had been off the ventilator for two days, her cranial nerves were functioning normally, and there was no voluntary or reflex movement in her limbs. No muscle wasting was observed, reflexes were absent, and the plantar response was downgoing. Sensation was impaired in all four limbs distally. Electrophysiological tests showed no response in abductor pollicis brevis when the median nerve was stimulated at the elbow, but a small action potential (0·7 μV) was present in response to stimulation at the wrist. The lateral popliteal nerve was inexcitable, and no sensory action potential could be recorded from the median or sural nerves. Cell count in the cerebrospinal fluid was normal, as was the protein concentration (0·2 g/l). Urinary porphyrins were negative. Her chart was reviewed for any drug that is known to cause a neuropathy and to confirm that vitamin B complex and intravenous feeding had been given early in her illness. She did not abuse alcohol. We failed to obtain a biopsy specimen of the sural nerve, but a biopsy specimen of muscle taken at the same time contained small nerve fascicles that showed a lack of myelinated axons and a severe loss of non-myelinated axons with evidence of primary axonal degeneration. The muscle itself showed some neuropathic changes, but more striking was an inflammatory myositis.

Over the next three months, despite having been totally paralysed in her limbs for at least six weeks, she made an excellent recovery.