the indigenous Pathans are. Diagnosis was made clinically and by examination of Giemsa stained smears of exudate.

Sores were cleaned, infiltrated with 2% lignocaine, and then curetted with removal of all raised inflamed edges. This was a rapid procedure (total procedure time four to 10 minutes, mean 6·8 minutes) and well tolerated, with only three patients complaining of pain. The 10 inpatients and 40 outpatients had their dressings changed daily after cleaning with calcium hypochlorite solution. The outpatients' wounds were assessed weekly, though this assessment was reliable in only 20 outpatients. This gave a total of 30 patients (78 lesions) in whom healing time was recorded accurately. The table shows healing times of the 78 lesions, taken as the time to complete re-epithelialisation of the granulating curetted wound.

Time taken for healing after curettage

Size of lesion	No of sores healing within:				Total
	2 weeks	3 weeks	4 weeks	>4 weeks	
Any size	12	34	27	5	78
Up to 1·5 cm diameter	12	27	6		45

Comment

This small study showed that after simple curettage alone 73 out of 78 adequately observed lesions healed within four weeks. Thirty nine out of 45 smaller sores (up to 1·5 cm diameter) healed within three weeks. These results are comparable with those achieved using any other method of treatment. This definitive treatment is rapid and requires only one attendance, which is a distinct advantage in an unreliable population. The cosmetic result, particularly for facial sores, is remarkably good, with none of the disfigurement that ensues from chronic, self limiting tissue destruction. Cutaneous leishmaniasis is becoming an increasingly important problem, particularly throughout the Middle East and wherever more widespread habitation within desert areas is promoted by irrigation schemes. With increasing ease of travel, and with many expatriates working in such areas, patients with this condition are likely to be seen in Britain. Curettage, an old and simple treatment that is effective, may be preferable to using the toxic parenteral pentavalent antimonials recommended in standard modern textbooks.[4]

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Implications of diagnostic delay in Duchenne muscular dystrophy

Duchenne muscular dystrophy is the commonest of the childhood muscular dystrophies. Inheritance is X linked. Progressive proximal muscle weakness in early childhood leads to loss of ambulation between 8 and 12 years and death in the late teens or early 20s. Newborn screening programmes have been undertaken elsewhere[1] to ensure early diagnosis of sporadic cases and counselling of families at risk. British paediatricians, however, have largely taken the view that preclinical detection of an incurable disease is not justified.[2 3]

The greater availability of genetic counselling together with more precise methods of detection of carriers[4] will in the future greatly reduce the number of second cases in known Duchenne families. Where second cases do occur they will increasingly be due to births before the diagnosis of the first affected boy.

We report a study of 34 boys with the disease from 16 families, each with two or more affected boys and none with a diagnosed case in an earlier generation.

Methods and results

The families were drawn from 35 kindreds seen throughout Britain as part of a genetic linkage analysis using DNA restriction fragment length polymorphisms to study the disease locus[5] and develop more accurate methods of carrier detection.[4] For each affected boy details of age at onset, presentation and diagnosis were collected.

Of 18 families with two or more affected boys in a sibship and no known case in a previous generation, clinical details were available in 16. The table gives the results for 34 affected boys from the 16 sibships. Among the 16 families, 10 had been incorrectly diagnosed, one mother had not been counselled, and five had not sought medical attention at the time of conception of the affected brothers.

Clinical details of 34 affected boys from 16 sibships

Family	Sib	Date of birth	Mode and age of presentation (years)	Age at diagnosis (years)
1	1	15/5/66	1-2, toe walking, Achilles tendon lengthening age 5	8
	2	29/8/67	Waddle*	7
	3	11/5/73	Raised CK activity*†	1-2
2	1	1965	Abnormal gait, unable to run age 4	9
	2	1973	*†	Raised CK activity at age 1
3	1	27/3/71	Gait problems, unable to run age 4½	7
	2	5/5/76	*	Raised CK activity age 2
4	1	11/2/68	Walking on toe age 2, clumsy, orthopaedic referral age 4	>3 (two weeks before birth of 2nd child)
	2	3/1/72	*†	1 month
5	Twins‡	13/7/65	Excessive falling, abnormal gait, age 4	6
	2	30/7/70	*†	1, raised CK activity
6	1	7/9/72	Delay in walking age 2	5
	2	2/2/75	*	1-2, raised CK activity
7	1	2/2/60	Tightening Achilles tendon age 4-5	10
	2	25/10/62	Abnormal gait, unable to climb age 4	7-8
8	1	28/4/65	Gait problems age 2	3
	2	7/11/68	*†	6 months, raised CK activity
9	1	4/9/63	Falling excessively, flat footed age 4	9
	2	10/1/69	Uncertain†	4, raised CK activity
	3	20/10/71	Uncertain†	6, raised CK activity
10	1	1974	Walking poorly, falling age 4-5	4½
	2	1976	*	2, raised CK activity
11	1	27/12/61	Psychomotor retardation age 2½	9
	2	5/6/63	Psychomotor retardation, gait problems age 3-4	7
12	1	8/11/65	Psychomotor retardation age 2	7
	2	2/12/68	Gait problems age 3†	4
13	1	29/8/75	Delayed motor development age 1-1½	3
	2	16/12/79	*†	Raised CK activity 1st year of life
14	1	14/1/67	Not sitting up at 9 months, delayed motor development age 1-2	6
	2	2/5/70	*†	3
15	1	14/8/75	Delayed walking age 1½	>4
	2	4/2/80	*†	Raised CK activity at 6 months
16	1	11/6/56	Abnormal gait and walking poorly age 1½	3½-4
	2	7/2/61	*†	3

CK = Creatine kinase.
*Screened because of family history.
†Conceived after misdiagnosis in older affected sibling.
‡Twin boys analysed as one.

Comment

The 16 families studied were representative of those in whom neonatal screening could have prevented the birth of a second affected boy. Nevertheless, 10 of the 16 index patients had been medically examined but not correctly diagnosed and one other mother had not been informed of the genetic implications of the disease, resulting in the conception of 12 further affected boys; hence increased awareness of the disease by practitioners would have a considerable effect. Similarly, Gardner-Medwin[3] reported that in 15 of 24 first cases symptoms developed before the second affected boy was conceived, though it is uncertain how many in that series had been brought to medical attention.

In our study five of six index children (38% of families) who presented with motor or psychomotor delay could have been diagnosed if all children who had not walked by the age of 18 months had had their creatine kinase activity estimated, as suggested by Gardner-Medwin.[3] A further seven (44% of families) presented with clumsiness of gait or excessive falling. Diagnosis in such cases requires familiarity with the typical Duchenne waddle and abnormal consistency of the calf muscles. Finally, no doctor should do or recommend Achilles tendon lengthening operations until the aetiology is firmly established. Against a background of a relatively rare but important disease—whose prevalence of 3/100 000 means that a considerable number of family practitioners will have no case on their panels—a series of well designed circulars sent to all relevant practitioners, together with financial support for attendance at clinical demonstrations, could provide a real and inexpensive alternative to a newborn screening programme. Such a programme would also circumvent the problem of causing unnecessary distress to parents by diagnosing an incurable disease in the neonatal period.[3]

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Plasma prolactin concentrations in a large population of healthy old people

The physiological importance of the change in dopaminergic mechanisms during aging has been clearly established. In both animals and man the function of presynaptic and postsynaptic dopaminergic mechanisms is altered during later life with a considerable decrease in synthesis and receptor function.[1-4] In vivo measurement of dopaminergic function in man is difficult owing to the lack of variables that accurately reflect such function. Although plasma prolactin concentrations reflect the result of various neuronal interactions, dopamine is thought to exert an important inhibitory control on release of prolactin. Thus an age dependent reduction of dopaminergic function in the tuberoinfundibular system might induce an increase of plasma prolactin concentrations. We carried out a study to investigate this.

Subjects, methods, and results

We have carried out a large scale study of a homogeneous population of elderly people in the small town of Ome (Lombardy, northern Italy), studying the general health of the inhabitants aged over 59 and checking certain clinical and biochemical variables indicative of specific diseases.

We measured plasma prolactin concentrations in 260 old people. Blood was collected from the brachial vein between 0700 and 0900 always in the same environment and by the same medical team. Prolactin concentrations were measured by radioimmunoassay using a commercial kit (Amersham International, England). No evidence of endocrinological or neuropsychiatric disease was detected.

Among women plasma prolactin concentrations were significantly higher in those aged 74-78 and those aged 79 and over than in those aged 59-63 (table), and a significant correlation was found between age and plasma prolactin concentrations ($p < 0.05$). By contrast, no age related differences were observed in the men. Surprisingly, the mean plasma prolactin concentration in the men as a whole was the same as that in the women, although in younger people (aged below 45) concentrations are lower in men.

Correlation between age and mean (SD) plasma prolactin concentrations in healthy old people

Age group (years)	Women		Men	
	Prolactin (ng/ml)	n	Prolactin (ng/ml)	n
59-63	7·91 (3·96)	53	10·41 (5·20)	34
64-68	9·30 (4·72)	30	10·21 (4·55)	29
69-73	9·00 (4·13)	33	9·03 (4·42)	32
74-78	10·15 (5·01)*	21	8·80 (2·05)	10
⩾79	10·36 (3·58)*	13	9·10 (2·95)	5

*$p < 0.05$ compared with women aged 59-63 (two tailed Student's t test).

Comment

It is possible that the inhibitory dopaminergic control on secretion of prolactin in the men had already reached a plateau at earlier ages. In contrast, it is possible that in women the dopaminergic control of secretion of prolactin becomes more important at the end of the fertile period. After this period the derangement of dopaminergic transmission leads to a small but important increase in secretion of prolactin.

The significant increase in plasma prolactin concentration detected confirms the hypothesis that dopaminergic function is decreased in older women. The increased incidence of breast tumours observed at this age may depend on the changes in plasma prolactin concentrations. The data also indicate that a mild dopaminomimetic drug may be useful in treating the typical disturbances induced by aging.

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Morbidity in diabetic and non-diabetic patients after major vascular surgery

Postoperative morbidity is generally thought to be higher in diabetics,[1 2] but there are no reports of studies that have included matched non-diabetic controls. We carried out a study to investigate postoperative morbidity in diabetics undergoing major vascular surgery and in non-diabetic controls matched for type of surgery, age, sex, weight, and complicating diseases.