valence of abnormal tolerance tests clearly rises after the age of 50.

Table VI gives some indication of the problem which a practitioner could create for himself by testing the urine of every patient in his practice. Assuming that a finding of glycosuria can be followed by a glucose-tolerance test, the largest group, that of non-diabetic glycosuria, could be diagnosed and discarded.

With enzyme testing after a meal as the screening method the "follow-up" group would consist of seven cases of florid diabetes, which certainly needed discovery, and 10 cases of G.T.T. diabetes, which would entail a positive effort to recall them for an annual review. If clintest rather than the enzyme test were used the number of positive results could be reduced by about half (Redhead, 1960) but would include nearly all the florid cases. The testing of *fasting* specimens by clintest would, for practical purposes, reveal only florid diabetes, but more than half of these cases awaiting diagnosis will not be found by this method.

Summary

A total of 345 oral 50-g. G.T.T.s were carried out on a sample of the general population. The subjects were chosen from those of the survey population (19,412) who had not found glycosuria using clintest after the largest meal of the day. They were approximately matched for age and sex with those who had found glycosuria.

Twenty-seven had a diabetic abnormality, 52 had lag-storage curves, and 63 had other miscellaneous abnormalities. Only 191 had completely normal glucose tolerance, though a further 10 were unclassifiable and two had renal glycosuria.

Under conditions of the G.T.T. one hour after glucose 21% of the men and 9.3% of the women now had glycosuria detectable by clintest; 9.3% of the men and 3.8% of the women had glycosuria detectable by clintest.

No diabetic abnormality was found under the age of 30 and only 2.9% at age 30-49. At 50 and over, however, about one in six of all men and at 70 and over about one in four of all women had a diabetic abnormality.

The mean fasting blood-glucose value was constant (87 mg.) until 70. At 70 and over the increase, although significant, was comparatively slight (99.1 mg.). One hour after the glucose the mean blood-glucose values increased steadily with age and showed a wide range of readings. At two hours the mean values still exceeded the fasting values and showed a greater range.

If the results from the investigation of those with diabetes or glycosuria (584) in the 19,412 who constituted the survey population are combined with that of the control group subjects (345) we estimate that the prevalence of diabetic abnormality in the general population is 6.2%, or 14.5% of those over 50.

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"IDIOPATHIC" PES CAVUS AN INVESTIGATION INTO ITS AETIOLOGY

BY

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Pes cavus is common and often one of the most severe of foot deformities; yet our knowledge of its causes is scanty and we can only guess at the mechanisms by which it is produced. Prompted by this lack of knowledge, it was decided five years ago that a "pes cavus clinic" should be set up so that a systematic prospective investigation into the causes of "idiopathic" pes cavus could be undertaken. This clinic started at the Royal National Orthopaedic Hospital in 1958 with the intention that all new patients should be thoroughly examined and investigated and that the clinic should be kept going indefinitely so that we could learn more of the development of the deformity and its prognosis.

This interim report follows a review of the first 77 new patients coming to the clinic.

A great deal has been written about pes cavus, but most authors refer to the mechanism of production of the deformity or its treatment and very few to the possible cause of the deforming mechanism. Turner (1952) was probably the first to emphasize that there was an underlying abnormality of the central nervous system in so-called

idiopathic cases. This association had been noted before by Plowright (1928) in one large family, some of whom had peroneal muscular atrophy while others had pes cavus without detectable neurological disorder. Spillane (1940) studied another family, some of whom suffered from peroneal muscular atrophy and Friedreich's ataxia, and he showed that patients with these disorders may have children who have pes cavus but are otherwise clinically normal. O'Connor (1959) expressed the opinion that the underlying cause was to be found in some abnormality of the spinal cord.

The many theories propounded for the local mechanism producing the deformity are listed in Table I. By far the most popular and generally accepted opinion is that of Duchenne (1867). He believed that the cavus foot and claw toes are produced simply by weakness of the interossei. This is an attractively simple theory and one which is generally assumed to be correct. Duchenne based this belief upon the study of 12 patients in whom he showed that electrical stimulation of the interossei gave a reduced or absent response. However, these investigations were

confined to patients known to have congenital or infantile paralysis, and his illustrations suggest that he was dealing mainly with severe degrees of cavus deformity.

TABLE I.—*Theories of the Local Mechanism Producing Pes Cavus*

Theory of Mechanism	Authors
Weakness of short muscles of hallux and interossei	Duchenne (1867)
Weakness of extensor digitorum longus with weakness of tibialis anterior in more severe cases	Fisher (1889)
Weakness of calf	Royle (1927)
Temporary paralysis of small muscles of foot supplied by lateral plantar nerve	Mills (1924)
Functional insufficiency of interossei with weakness of extensor digitorum longus	Little (1938)
Overaction of intrinsic caused in some cases by a short tendo Achillis	Dickson and Diveley (1939)
Paralysis of extensor digitorum longus, tibialis anterior, tendo Achillis, or peronei	Steindler (1917)
Simple disturbance of muscle balance between flexors and extensors so that there is an overaction of extensor digitorum longus	Ollerenshaw (1927)
Childhood illness—cavus produced by the weight of bedclothes	Pugh (1927)
Isolated palsy of flexor hallucis brevis with overaction of extensor hallucis longus	Ducroquet (1910)
Exaggerated action of peroneus longus	Tubby (1896)
Loss of synergic muscle control without specific weakness and due to an effect of poliomyelitis on spinal cord and higher centres	Saunders (1935)
Weak peronei causing overaction of tibialis anterior and tibialis posterior	Golding-Bird (1883)
Weakness of tibialis anterior with normal extensor digitorum longus	Cole (1940)
Rheumatoid arthritis of tarsal joints	Tubby (1912)
Unsuitable footwear	Bentzon (1933)
Primary contracture of plantar fascia	Rugh (1927)
Primary abnormality of bone	Brewster and Larson (1940)
Primitive laxity of dorsal ligaments of foot	Kirmisson (1906)
General disruption of co-ordination between all elements of foot musculature	Turner (1952)
Overaction of calf muscle	Altakoff (1931)
Congenital abnormality	Gilroy (1929)

Retrospective Survey

Before starting the main investigation we carried out a retrospective survey of the hospital records of patients attending the Royal National Orthopaedic Hospital with a diagnosis of pes cavus during 1949–58. The most interesting finding of this survey was the small proportion of patients in whom a neurological cause could be established (Table II). No less than 81% were placed in the idiopathic group even though they had been examined and documented by consultants and registrars who were well aware that pes cavus might be related to neurological

TABLE II.—*Retrospective Survey of Patients Seen During 1949–58*

Diagnosis Recorded in Hospital Records	No. of Patients
Idiopathic	465 (81%)
Poliomyelitis	54
Spina bifida	21
Peroneal muscular atrophy	15
Friedreich's ataxia	7
Traumatic	6
Cerebral palsy	5
Post-diphtheritic	2
Congenital	1
Angioma of medulla	1
	112 (19%)

disease that was difficult to detect. The number of patients who had had poliomyelitis was high, largely because of the hospital's special interest in this disease.

We had hoped to get information from analysing the 465 cases of idiopathic pes cavus, but this was impossible to do retrospectively. Although it can probably be assumed that the reflexes were always tested, they had been recorded in only 28% of the patients and the family history in only 11%.

The Pes Cavus Clinic

Since the pes cavus clinic was set up in 1958 nearly all new patients with pes cavus referred to the Royal National Orthopaedic Hospital have been passed on to this special clinic. Each patient is first examined by an orthopaedic surgeon (D. R. S.), who pays particular attention to the history and characteristics of the local deformity, and the

distribution of any muscle weakness in the lower limbs. These findings, as well as the results of later examinations, are recorded on a punch card designed for the purpose. At the same attendance a permanent record of the deformity is made by photographs of the feet and footprints, and x-ray films of the feet and lumbar spine are taken. The patient is then seen by a neurologist (P. H. S.), and at this examination available relatives are also interviewed and examined. Finally, electrodiagnostic tests are performed (D. A. B.) following a standard routine for all patients. Normally the more severely affected limb is selected for electrodiagnosis, and in this limb flexor hallucis brevis, extensor digitorum brevis, gastrocnemius, and peroneus longus are tested. In each muscle the intensity-duration curve is plotted, electromyography is performed, and motor-nerve conduction times are measured.

When the referring surgeon decides that surgical correction of the deformity is indicated he is asked to do muscle biopsies.

Results

At the time of this review 77 new patients had been investigated, and we feel that the results are of sufficient interest to justify an early report of these findings. However, the clinic continues so that more new patients can be investigated, but more particularly so that all the patients can be followed up for some years to come.

The main finding was that neurological examination revealed evidence of disorder in the central nervous system in 51 (66%) of the 77 patients (Table III). This is in striking contrast to the figures in the retrospective survey.

TABLE III.—*Patients Seen in the Pes Cavus Clinic*

Clinical Diagnosis	No. of Patients
Idiopathic	26 (33·8%)
Peroneal muscular atrophy:	
Definite	14
Probable	8
Possible	4
Friedreich's ataxia:	
Uncomplicated	1 (1·3%)
With peroneal muscular atrophy	2 (2·6%)
Myelodysplasia	7 (9·1%)
Poliomyelitis	4 (5·2%)
Spastic monoplegia	1
Spastic paraplegia	1
Polyneuritis	1 (1·3%)
Poliomyelitis and probable peroneal muscular atrophy	1 (1·3%)
Muscle dysplasia	1 (1·3%)
Trauma	1 (1·3%)
Neurological signs but no diagnosis established	5 (6·5%)

66·2%

Peroneal Muscular Atrophy.—The high incidence of peroneal muscular atrophy is particularly striking, one-third of the whole series being placed in this category. Twenty-two patients were classified as "definite" or "probable" peroneal muscular atrophy. Of these, 10 had a family history of peroneal muscular atrophy, and 19 a family history of cavus deformity; 20 had muscle weakness; 19 had abnormal reflexes; and 6 had diminished sensibility. Four children were diagnosed under "possible peroneal muscular atrophy." Three of them had a family history of cavus deformity, and had mild muscle weakness themselves; and one child had more pronounced muscle weakness but no family history.

Family History of Cavus Feet.—Of the 26 patients with peroneal muscular atrophy, 22 gave a family history of cavus deformity. In sharp contrast, the 25 patients with other neurological causes for their cavus deformity included only one who admitted to a family history of cavus. In the idiopathic group, 11 of the 26 patients gave a family history of cavus.

Radiological Evidence of Spina Bifida.—Of the 77 patients, 34 (44%) had radiological evidence of a neural arch defect visible on antero-posterior views of the lumbo-

sacral region. The incidence was, however, no greater in patients with abnormal neurological findings than in the idiopathic cases, and it was present in only four out of the seven patients considered clinically to have myelodysplasia.

Electrodiagnostic Findings.—In the patients in whom a neurological diagnosis could be established clinically, electrodiagnosis was not often of value in establishing which neurological disease was responsible for the denervation. All too often the only possible comment was "long-standing denervation." However, electrodiagnosis was useful in detecting abnormality and establishing the presence of denervation, and in some cases this assisted the neurologist in making a firm diagnosis. Of particular interest was the finding of electrical abnormality in seven cases in which no neurological signs were detected. The abnormal findings included denervation on intensity-duration curves, abnormalities on electromyography, and delays in motor-nerve conduction. In the patients with peroneal muscular atrophy the electrical findings were abnormal in all definite cases and in seven of the eight probable cases. However, the findings were normal in the four possible cases. In the other patients with abnormal neurological signs the findings were abnormal except in three with myelodysplasia with no muscle weakness, two in whom no definite diagnosis could be established, one with uncomplicated Friedreich's ataxia, one with poliomyelitis, one with muscle dysplasia, and one with traumatic pes cavus. Intensity-duration curves showed denervation in the intrinsic muscles of the feet in 35 patients (45%).

Distribution of Muscle Weakness.—Muscle weakness below the knee was detected in 42 patients (55%), but it must be emphasized that clinical testing was often difficult in the presence of fixed deformity. It is well known that muscles, apparently weak while acting over deformed joints, may regain nearly normal power as soon as the fixed deformity is corrected, and this must be remembered when charting muscle power in the cavus foot. Although every attempt was made to allow for this possible error, we are by no means satisfied that our estimates are any more than a reasonable guess in most cases. The distribution of estimated muscle weakness is shown in Table IV. This shows a clear preponderance of peroneal and extensor weakness, but no really significant pattern emerges.

TABLE IV.—*Distribution of Estimated Muscle Weakness*

Muscle Groups	No. of Patients
All muscles below the knee	4
Peronei only	9
Peronei and all extensors	4
All extensors only	3
Peronei, extensor hallucis longus, and extensor digitorum longus	4
Peronei, tibialis anterior, and extensor digitorum longus	4
Peronei and extensor hallucis longus	3
Peronei and extensor digitorum longus	2
Peronei and tibialis anterior	4
Peronei, extensor hallucis longus, and tibialis anterior	1
Extensor hallucis longus and extensor digitorum longus	2
Tibialis anterior and extensor hallucis longus	1
Calf	1
	42

Discussion

We have not unravelled the mystery of pes cavus; nevertheless we have shown that careful examination can often uncover an underlying cause. In the retrospective survey of patients' notes at the Royal National Orthopaedic Hospital no less than 81% had been placed in the idiopathic group. In the pes cavus clinic this percentage has dropped to 34% on clinical examination alone.

There still remains about one-third in whom no neurological abnormality can be detected clinically, and our attention in the future must lie largely with this group.

It has been interesting to note that seven (27%) of the remaining 26 so-called idiopathic cavus feet had electrodiagnostic evidence of muscle denervation; and 11 (42%) had a history of similar foot deformity in their family. We now intend to investigate further the families of patients with cavus feet and to test the significance of these electrodiagnostic findings by follow-up over several years.

It is unfortunate that clinical testing of muscle power below the knee has provided little help in establishing the mechanism of production of the deformity. We believe that pes cavus is the result of muscle weakness or imbalance in most cases, and it was disappointing that no recurrent pattern of weakness emerged.

Summary

A clinic to study pes cavus has been set up, and the first 77 patients are analysed.

A retrospective survey of the notes of 577 patients seen during 1949–58 showed that in only 19% had a neurological cause been established. In the pes cavus clinic this figure has risen to 66% on clinical examination alone; and of the remaining 34% with no clinical abnormality seven patients had electrodiagnostic abnormalities and 11 had a family history of cavus deformity.

No significant pattern of muscle weakness emerged to establish the mechanism of production of the deformity.

We wish to thank the surgeons of the Royal National Orthopaedic Hospital for allowing us to investigate their Patients. The Pes Cavus Clinic was started at the suggestion of Professor J. I. P. James, and it is a pleasure to acknowledge and thank him for his help and encouragement.

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