mentioned by most writers. Constipation, nausea, and vomiting are less common but have been mentioned in most investigations. Opinions vary about the hypotensive effects of the drug. Horatz (1954), reporting on 439 cases, thought that the danger of orthostatic collapse necessitated great caution in management, but most investigators were not seriously concerned with this complication, and the general consensus of opinion was that in this respect pacatal is safer than chlorpromazine.

Of the more serious complications following use of chlorpromazine, only Werenberg (1955) reports the incidence of epileptic fits, which occurred in 7% of 100 patients. Of these cases, four had been leucotomized, one had had fits previously, and two had had frequent electroplexy. Skin reactions and oedema are rare. Kroner (1955) reports a few cases of light sensitivity, but no mention is made of this complication anywhere else in the literature. No case of jaundice has yet been reported.

As with chlorpromazine, the blood picture varies, but a mild leucopenia is the most commonly reported finding. Three cases of agranulocytosis, however, have been recorded, two of them fatal (Wenderoth and Lennartz, 1955; Werenberg, 1955; Gore and Biezanek, 1956).

Bowes (1956) mentions one case of atonic bladder and one of paralytic ileus, while Kline and Jacob (1955) considered the drug so toxic that they abandoned it altogether. Their experience was, however, limited to seven cases.

In the present series, among the 50 patients receiving pacatal, dryness of the mouth was an almost universal complaint and in some cases was very troublesome but did not necessitate taking anyone off treatment. Cycloplegia was also common and necessitated two patients having to discontinue treatment. Physostigmine drops alleviated the complaint to some extent. Constipation was occasionally troublesome, but less so than with chlorpromazine, and neither nausea nor vomiting was noted. Twelve patients underwent extensive laboratory investigations with negative results, at periods varying from 5 to 13 weeks after treatment started. Haemoglobin levels and sedimentation rates were unchanged and the lowest recorded total white count was 4,800. Urinalyses were invariably normal, and alkaline phosphatase levels, non-protein nitrogen, and serum bilirubin were within normal limits.

In 2 of the 50 cases receiving chlorpromazine it had to be discontinued. One developed jaundice and another had an intractable dermatitis while on the drug.

Summary and Conclusions

Fifty cases falling within the schizophrenic group of disorders were treated with pacatal, while a similar number received chlorpromazine.

The method of selecting patients and the scheme of dosage are outlined. It is considered that the two groups are comparable so far as prognosis is concerned.

Assessment of results was made on a five-point scale by an independent observer who knew nothing of the type of treatment. Assessment was made either after 13 weeks or when treatment had to be discontinued for one of various reasons.

The results are significantly better in those patients receiving chlorpromazine, and there is nothing in this trial to suggest that pacatal has any value in the treatment of a mixed group of schizophrenic patients.

Both from a review of the literature and from experience in this trial pacatal would appear to be less toxic than chlorpromazine. Jaundice does not seem to be a risk in using pacatal, but agranulocytosis is a dangerous complication with both drugs.

I thank Dr. H. C. Beccle, physician-superintendent of Springfield Hospital, for his kindness in making the independent assessment of results; and Mr. Mayall, the chief pharmacist, for his

invaluable assistance in arranging the order of treatment. I am indebted to Messrs. William R. Warner and Co. Ltd. for their generous supplies of pacatal.

ADDENDUM.—Since this article was written, two reports on the use of pacatal in psychiatry have appeared in this country. J. G. Thorpe and A. A. Baker (*J. ment. Sci.*, 1956, 102, 790) agreed that pacatal was less effective but less toxic than chlorpromazine, while P. H. Mitchell, P. Sykes, and A. King (*British Medical Journal*, 1957, 1, 204) found it of some value in controlling incontinence, noisiness, and aggression in deteriorated female schizophrenics, but considered it to be much more toxic. They reported one death due to agranulocytosis and two cases of jaundice.

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ARACHNODACTYLY WITH CONGENITAL LESIONS OF THE URINARY TRACT

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A considerable number of cases of arachnodactyly have been reported since Marfan's original description of a girl aged 5 years with spider fingers (*pattes d'araignée*) and poorly developed musculature (Marfan, 1896). Although the deformities described in this case were predominantly skeletal, subsequent reports of patients with this syndrome have indicated that it may be associated with other congenital abnormalities. Salle (1912) was the first to recognize the association with patent foramen ovale, and Börger (1914) reported details of a patient who had dislocation of the lens and iridodonesis with arachnodactyly. The frequency with which congenital anomalies of the heart may occur in patients with Marfan's syndrome has been stressed by Piper and Irvine-Jones (1926). Baer, Taussig, and Oppenheimer (1943) associated arachnodactyly with developmental abnormalities of the media of the aorta, and a further case with dissecting aortic aneurysm was described by Etter

and Glover (1943). High arched palate and anomalies of the lungs have also been reported (Börger, 1914).

Rados (1942), reviewing 204 previously reported patients with Marfan's syndrome, stated that no involvement of the genito-urinary system had been described. Genital and urinary tract abnormalities have, however, been recorded in association with arachnodactyly, although they appear to be rare. Börger (1914) and Delord and Viallefont (1936) described genital hypoplasia in the male, and other writers (Haridas, 1941; Stern, 1944) reported undescended testes in their patients. Olcott (1940) reported the case of a patient with arachnodactyly in whom "the left kidney was in the pelvis of the body with its hilum against one of the flat surfaces," presumably a description of an ectopic left kidney; and Fischl and Ruthberg (1951) recorded three cases of Marfan's syndrome, in one of which the left kidney was ectopic and hydronephrotic.

No previous report has drawn attention to the association between arachnodactyly and abnormalities of the urinary tract. This paper describes the clinical features and post-mortem findings in two patients who had Marfan's syndrome, and who died with renal failure due to congenital lesions of the urinary tract.

Case 1

A brewery clerk aged 33 was referred to Hammersmith Hospital on October 14, 1954, because of extreme weakness of his arms and hands, and protuberance of his abdomen. He had always been thin, being known as the "Belsen horror" when serving in the R.A.F. in 1945, but his arms and hands had become progressively thinner and weaker during the previous five years, and his speech somewhat slurred. He was myopic and completely colour-blind. There had been no frequency of micturition, no dysuria, and no haematuria. He shaved daily, and sexual function was normal.

On examination he was 5 ft. 10 in. (177.8 cm.) tall and weighed 8 st. 7 lb. (54 kg.). His fingers were very long and thin and his palate was high and arched. There was marked wasting of the muscles of his face, neck, and shoulder girdle, with the exception of the deltoids, which gave him an emaciated appearance contrasting strikingly with his dysarthric protestations of good health. He had bilateral ptosis and frontal recession of the hair, which, with the wasting, produced a typical myopathic facies. The abdominal and thoracic muscles were unaffected, but there was slight weakness and wasting of the extensors of the feet. The biceps, brachioradialis, and ankle jerks were diminished. Sensation was normal. The grip was myotonic, and percussion myotonia, exaggerated by neostigmine, was demonstrated in both thenar eminences. Blood pressure was 180/120 mm. Hg, but the heart was not enlarged and there were no murmurs. The abdomen was distended by two large polycystic kidneys which filled both loins. The testes were soft, but of normal size, and the prostate was small.

Investigations.—The blood count showed a mild normochromic anaemia (Hb, 10.9 g. per 100 ml.; W.B.C., 6,000 per c.mm.), and the E.S.R. (Westergren) was 60 mm. in one hour. Twenty-four-hour urine output averaged two litres, with a protein content of 900 mg. a day. The blood urea was 168 mg. per 100 ml. Plasma proteins were normal (albumin 4.2 g., globulin 2.1 g.). Serum electrolytes were: Na⁺ 136, K⁺ 4.5, Cl⁻ 103.5, HCO₃⁻ 21.3 mEq per litre. Alkaline phosphatase was 17.5 King-Armstrong units per 100 ml. Radiography of the abdomen after perirenal air insufflation confirmed the presence of two large polycystic kidneys.

The blood urea rose to 300 mg. per 100 ml. during the month after his admission to hospital. In an attempt to improve his renal function, a Rovsing operation was per-

formed on the right side, many of the cysts being punctured. Following the operation, however, his condition deteriorated rapidly and he died.

Necropsy Report.—The necropsy confirmed the clinical findings. The most striking features were the two enormous polycystic kidneys. The right kidney weighed 2,024 g. and the left 2,610 g. There was no abnormality in the cardiovascular system. The testes were normal.

Family History.—The patient had no siblings. His father was aged 56 and alive and well. His mother had died at the age of 63 from cancer of the lung. She was said to have had excessively long fingers. The mother had four brothers and four sisters, of whom only one sister was living, the remainder having died suddenly from unknown causes before the age of 40. The patient's maternal grandfather had been colour-blind. There was no history of polycystic kidneys in his family.

Case 2

G.C., a young storeman aged 16, attended hospital on August 17, 1955, complaining of shortness of breath on exertion, lethargy, and weight loss for two months. Pallor had been noted by his family for the preceding six months. A deformity of the left knee had been present since birth. There was no history of haematuria, polyuria, or frequency of micturition.

On examination he was a tall slender youth—height 5 ft. 6½ in. (169.5 cm.), weight 7 st. 6 lb. (47.2 kg.). The skin was pale but there was no oedema. The length of the forearms and legs was striking, his span measurement being 3 in. (7.5 cm.) greater than his height, and the symphysis pubis to ground measurement 5 in. (12.5 cm.) greater than from symphysis to vertex (Fig. 1). The fingers and toes were long and thin, and there was slight webbing between the fingers. There was old subluxation of the left knee, and the right thumb was hyperextensible at the metacarpo-phalangeal joint. The patella on the right side was very mobile and higher than normal, the distance from its lower border to the tibial tubercle measuring 2 in. (5 cm.). There was a moderate kyphosis and slight "pigeon-chest" deformity. The palate was normal and there was no dislocation of the lens. Blood pressure was 150/100 mm. Hg; the heart was not enlarged and there were no murmurs. Neither kidney was palpable; testes and prostate were of normal size, and pubic hair was of masculine distribution. Muscular development was poor, tendon reflexes were uniformly diminished, and abdominal reflexes were absent.

Investigations.—The blood count showed a normochromic anaemia (Hb, 8.1 g. per 100 ml.; M.C.H.C., 32.5%; W.B.C., 6,000 per c.mm.). E.S.R. (Westergren) was 72 mm. in one hour. Twenty-four-hour urine output varied from 1,200 to 3,200 ml., and there was a heavy albuminuria, ranging from 4.7 to 9.7 g. in 24 hours. Urinary deposits contained no cells or casts, and cultures were repeatedly sterile. The range of urinary specific gravity was 1004–1008. The blood urea was 104 mg. per 100 ml. Plasma proteins 6.6 g. (albumin 3.9 g., globulin 2.7 g.). Serum electrolytes were: Ca⁺⁺ 4.55, Na⁺ 145, K⁺ 3.6, Cl⁻ 104.5, HCO₃⁻ 17.2 mEq per litre. Alkaline phosphatase was 55 King-Armstrong units per 100 ml.

FIG. 1.—Case 2. Illustrating the long extremities and subluxation of the left knee.

Bone x-ray films revealed a coarse trabecular pattern with subperiosteal bone erosions in the fingers and right femur suggestive of secondary hyperparathyroidism. Retrograde pyelography (Mr. R. Shackman) showed a small kidney on the right side with some dilatation of the right ureter; the left renal pelvis was slightly larger than normal and the calices were truncated.

The patient was treated with repeated blood transfusions to correct the anaemia, a low-protein diet, and a high fluid intake. Despite a progressively increasing blood urea level, he was able to remain at work for 11 months. He was admitted to hospital for the last time on July 22, 1956, and died in uraemic coma one week later.

Necropsy Report (Dr. M. Ralston).—Post-mortem examination confirmed the clinical findings and revealed the nature of the renal lesions. The dissection of the urinary tract is shown in Fig. 2. Both kidneys were much reduced in size,

FIG. 2.—Case 2. Dissection of the urinary tract.

the right weighing 16 g., the left 72 g. Both showed scarring of the capsular surfaces, the right having rather coarse scars, the left being more finely and regularly scarred. The cut surfaces of both showed cortical narrowing and loss of pattern. The right renal pelvis and ureter were greatly dilated down to a point 3 cm. above the right ureteric orifice, where the lumen narrowed abruptly. The left ureter was a little dilated throughout its length. Microscopically both kidneys showed the features of chronic pyelonephritis. Sections of the right ureter, however, revealed no explanation for the narrowing. Other post-mortem findings of note were secondary parathyroid hyperplasia (combined weight 260 mg.), with resultant effects on the bones, pulmonary oedema, and superficial haemorrhagic ulceration of the terminal ileum. The aorta showed no evidence of medial degenerative changes.

Family History.—The relevant clinical findings are summarized in the accompanying Table. No genito-urinary abnormalities were detected in the parents or siblings of this patient. The brothers and sisters were, with one exception, tall and remarkably thin. The father had aortic incompetence, but the Wassermann reaction had been negative, and there was no other cardiac lesion or any previous history of rheumatic fever. The mother was normal in all respects. Of the siblings, F.C. had unexplained exophthalmos, O.C. had a pulmonary cyst and a congenital deformity of the right middle lobe, and J.C. suffered from epilepsy. All these abnormalities may be found in association with arachnodactyly. One sister (E.C.) had arachnodactyly without any other abnormality.

Discussion

Both of these patients had arachnodactyly and congenital abnormalities of the urinary tract. The polycystic kidneys in Case 1 were clearly of congenital origin. The lesion in Case 2 is less easily defined, but the narrowing of the right ureter was presumably congenital, for microscopical examination of the wall of the ureter at this point revealed no evidence of either inflammation or fibrosis. The small right kidney is fully explicable as the end-result of pyelonephritic scarring without invoking congenital hypoplasia. Theoretically it is possible that pyelonephritis has been superimposed

Age, Height, Finger Length, Height of Patella, and Clinical Findings in Parents and Siblings of Case 2

Subject	Sex and Age	Height		Length of Fingers	Length of Patellar Ligament*		Other Findings
		in.	cm.		R.	L.	
					in. cm.	in. cm.	
Father	M 51	70	177.8	Normal	1.8 4.6	1.8 4.6	Aortic incompetence
Mother F.C...	F 50	66	167.6	,,	1.5 3.8	1.5 3.8	—
	M 31	71½	181.6		2 5	2 5	Tall and thin. Unexplained exophthalmos
R.C.	M 29	75½	191.7	Long	2 5	1.8 4.6	Very tall and thin
O.C.	F 27	69½	176	,,	2 5	2 5	Tall and thin. Right middle lobe divided by horizontal fissure. Cyst of right lower lobe
J.C. ...	F 23	66	167.6	Normal	1.5 3.8	1.5 3.8	Epilepsy. Old infantile right hemiplegia with shortening of right leg
R.C. E.C.	M 21	75	190.5	,, Arachnodactyly	1.8 4.6	1.8 4.6	—
	F 18	71	180.3		2 5	2 5	
G.C. Case 2	M 16	66½	169.5	,,	2 5	—	Subluxation of left knee. Kyphosis. "Pigeon-chest." Congenital stricture of right ureter

* Length of patellar ligament is the distance from the lower border of the patella to the tibial tubercle.

on a congenitally hypoplastic kidney, but unequal involvement of the kidneys in pyelonephritis is well recognized, and the obstruction on the right side would explain the greater severity of the process in the right kidney. The radiological appearance of the bones and the raised alkaline phosphatase indicated that this patient had renal osteodystrophy, and the parathyroid glands found at necropsy were characteristically hypertrophied. These bone changes are often found in young patients with chronic pyelonephritis and uraemia.

Patients with Marfan's syndrome may have a number of associated abnormalities. Congenital defects and spider fingers may also be found in other members of their families (Rados, 1942). The coincidence of colour-blindness, illustrated by Case 1, has been recorded previously (Neresheimer, 1916). This patient also had dystrophia myotonica. Poor muscular development has been a feature of the syndrome since it was first described by Marfan (1896), and amyotonia congenita has been reported in patients with arachnodactyly (Young, 1929), but we have found no previously recorded cases with dystrophia myotonica. Our first patient had neither testicular atrophy nor cataract, and no other case of dystrophia myotonica had been recognized in his family. The myotonia and the distribution of the muscle wasting were, however, characteristic of this disease. His mother was said to have had excessively long fingers, and it is of interest that most of her family had died suddenly before the age of 40.

The associated abnormalities in the second patient involved the skeletal system. In addition to long fingers, he had long arms and legs. The subluxation of the left knee and the hyperextensible right thumb were typical findings in a patient with arachnodactyly, and the patella on the right was high. "High patella" has frequently been described in patients with Marfan's syndrome since Méry and Babonneix (1902) drew attention to this abnormality in a further report of Marfan's original case. Fischl and Ruthberg (1951) stated that the patella was 3 cm. above the normal position in one of their cases, but no report has defined the normal position of the patella. The distance of 2 in. (5 cm.) from the lower border of the patella to the tibial tubercle, which was found in the second patient and in four of his siblings, was greater than in any of 25 control subjects,

in whom the mean distance, measured by the same observer, was 1.4 in. (3.5 cm.), with a range of 1.1 to 1.8 in. (2.7 to 4.6 cm.).

Examination of the family of Case 2 revealed a number of interesting abnormalities. Only one (E.C.) had arachnodactyly, but five out of the six siblings were strikingly tall and thin, and the patella was high in four. Whittaker and Sheehan (1954) have recorded instances of aortic lesions without arachnodactyly in the families of patients with Marfan's syndrome, and although the father had no skeletal abnormality it is tempting to consider his aortic incompetence as possibly of congenital origin. Unexplained exophthalmos, illustrated by F.C., has been described in patients with arachnodactyly (Baer *et al.*, 1943), and abnormalities of the lobes of the lungs, which were present in the sister, O.C., are not uncommon (Rados, 1942). Another sister suffered from epilepsy, a finding also reported by Marfan in one of the siblings of his original case (Marfan, 1896).

The aetiology of Marfan's syndrome, like so many other congenital anomalies, remains obscure. Weve (1931) explained the skeletal and visceral abnormalities on the basis of a congenital mesodermal dystrophy, but it has been repeatedly stressed that, if this theory were true, urogenital abnormalities should also occur (Rados, 1942). In fact, genital abnormalities have been reported from time to time. The two cases reported by Olcott (1940) and by Fischl and Ruthberg (1951) illustrate that congenital lesions of the urinary tract may also be found with arachnodactyly. These findings lend some support to Weve's hypothesis, but it is interesting to speculate how a dystrophy affecting one of the germinal layers may at the same time result in excessive growth of the extremities.

Summary

A brief survey of the abnormalities which may be found in patients with Marfan's syndrome is given. The apparent rarity of the association of arachnodactyly with genito-urinary abnormalities is stressed. Two patients who had arachnodactyly and congenital lesions of the urinary tract are described. The first had bilateral polycystic kidneys, and the second had chronic pyelonephritis resulting from a congenital stricture of the lower end of the right ureter. These findings lend some support to the hypothesis that the visceral abnormalities associated with Marfan's syndrome may be due to a congenital mesodermal dystrophy.

The first patient also had dystrophica myotonica. The coincidence of arachnodactyly with this disorder has not previously been described.

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A SIMPLE METHOD OF BLOOD-SUGAR ESTIMATION

BY

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Many methods of blood-sugar estimation are available at present—for example, Hagedorn and Jensen, Maclean, Folin, Harman, and Somogyi. In recent years, moreover, the "true glucose" methods have become more and more popular, Nelson's modification of the Somogyi technique, giving close approximation to the true glucose level, being widely used in hospital practice. While there can be no doubt that these methods, particularly the latter, give accurate and consistent results, all of them require laboratory facilities to a greater or less extent and are best carried out by a person experienced in the particular technique involved. Very often, however, when faced with a problem involving the need for a blood-sugar estimation, it is adequate for the clinician to know that the blood sugar lies within a certain range. The method described below requires a minimum of technical equipment and training, and can be readily performed in the consulting-room or the patient's home, the results being available within ten minutes.

Method

The outfit for this simple method is manufactured by Ames Company (London) Ltd., who supplied it for the purpose of this investigation. The test is in essence a quantitative copper reduction method, the test solution being compared with a standard colour range chart, and the blood-sugar range being read directly from this chart. The total equipment is two standard test-tubes (7.2 by 1.4 cm.—"clintest" pattern) graduated in millilitres, a metal test-tube rack, two pipettes, filter papers, and two small bottles of reagents conveniently presented as tablets (A and B). No external heating is required. The whole apparatus can be packed in a box some 3 by 3 by 1 in. (7.5 by 7.5 by 2.5 cm.), and is therefore very readily portable.

The test-tubes are set up in the rack, 2 ml. of water is put into one tube, then one tablet A is added and allowed to dissolve completely. Then 1 ml. of venous blood is placed in this solution and thoroughly mixed with a glass rod, this procedure deproteinizing the blood. A filter cone is then placed in the second test-tube and the solution from the first test-tube poured into the cone. A clear filtrate is obtained and 1 ml. is collected. (These measurements are very easily made, as the tubes are graduated in millilitres.) To this solution one tablet B is added; vigorous boiling occurs, during which time the tube should not be touched. After the boiling ceases the test-tube is shaken briskly three or four times, there is a wait for 30 seconds, and the colour is then compared with the standard scale provided. This colour scale shows three definite stages—blue, olive green, and tan—corresponding to blood-sugar levels of 100, 150, and 200 mg. per 100 ml., but with a little experience intermediate levels of 125 and 175 mg. can be judged reasonably accurately. This range—that is, 100 to 200 mg. per 100 ml.—is the standard range claimed by the manufacturers. An attempt has been made to extend this range to increase the usefulness of the method. The effective range of the scale can be readily extended to a level of 400 mg. per 100 ml. by simple dilution of the filtrate, 0.5 ml. of filtrate being collected, making up to 1 ml. with water, the actual testing being carried out as above, and the result obtained being multiplied by two. Further dilutions to an upper level of 800 mg. per 100 ml. can be readily made if desired.