describes how older children can help younger children's health and development.

Booklet on the University of London's master of science course in mother and child health. This describes the course. The curriculum it gives may be of help to others setting up teaching programmes.

Visual Communication Handbook: Denys Saunders. For those desiring to become more effective in communication.

Breast Feeding in Practice: E Helsing and F Savage King. An aid to health workers to guide and support mothers in successful breast feeding.

Breast Feeding, the Biological Option; *Child Care in the Tropics*; *Care of the Newborn in Developing Countries*; *Practical Mother and Child Health in Developing Countries*; *A Handbook of Tropical Paediatrics*: all by G J Ebrahim. Designed for use in small hospitals and health centres. (English Language Book Society editions.)

Paediatric Practice in Developing Countries: G J Ebrahim. Continues the themes of the preceding five titles and is meant for the district officer who has to provide a comprehensive child care service for the district population.

Better Child Care: Voluntary Health Association of India. Illustrated memory and teaching aid for talking with parents.

The Care of Babies and Young Children in the Tropics: David Morley. Written for European mothers taking their children to hot climates.

Child Health in a Changing Environment: G J Ebrahim. Intended for national planners, it seeks to establish the scientific base for primary health care.

Nutrition in Mother and Child Health: G J Ebrahim. Designed for nutrition workers.

Lesson of the Week

Danger of inadequate urological supervision of patients with congenital neuropathic bladder

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It is customary in the United Kingdom for children with congenital neuropathic bladder to be under close urological supervision. The care these same patients receive as adults is sometimes haphazard as may be illustrated by six cases referred to us in the past four years.

Patients with congenital neuropathic bladder need lifelong urological supervision to avoid deterioration of the upper renal tracts

Case reports

Six patients who had had a low level myelomeningocele closed early in life presented with urological problems. With the exception of one (case 1) all walked well. Two patients (cases 2 and 3) had urgency of micturition but were able to stay dry, the others were incontinent of urine but managed this satisfactorily with a penile appliance. All patients had otherwise enjoyed good health.

Case 1—A 21 year old man had had a myelomeningocele at level L4, complete: he was confined to a wheelchair. An intravenous urogram at the age of 3 years had shown normal upper renal tracts. He then had no further urological supervision until he presented with a short history of urinary infections and pain in the right loin. An intravenous urogram showed pronounced bilateral hydronephrosis and bladder stones, and a cystogram showed right vesicoureteric reflux and obstruction of the bladder outlet at the level of the external urethral sphincter. After removal of the stones his hydronephrosis resolved, but he still had vesicoureteric reflux with residual obstruction of the bladder outlet and might require urethral sphincterotomy in the future.

Case 2—A 17 year old man had had myelomeningocele at level S1, incomplete. Details of his previous care are lacking but he had certainly had no urological investigations for at least seven years before presenting with a two year history of increasing urgency and

incontinence. An intravenous urogram showed moderate bilateral hydronephrosis, and a videourodynamic study indicated obstruction of the bladder outlet due to detrusor sphincter dyssynergia. After endoscopic urethral sphincterotomy his hydronephrosis resolved (though with residual left renal parenchymal damage) and his incontinence improved.

Case 3—A 21 year old man whose myelomeningocele was at level L5, incomplete, had been under urological supervision until the age of 15 but was then discharged because his bladder was "normal"; he stated that he had always suffered from urgency of micturition. He was reinvestigated because of haematuria when an intravenous urogram showed pronounced bilateral hydronephrosis. A resection of the bladder neck was performed elsewhere and he was referred to us when this procedure did not improve his upper renal tracts. A videourodynamic study showed detrusor sphincter dyssynergia. Endoscopic sphincterotomy improved his hydronephrosis but he became completely incontinent after this operation and now needs to use a penile appliance.

Case 4—A 30 year old man had had a myelomeningocele at level L4, complete. Details of his previous care are incomplete though as a child he had had a bladder neck Y-V plasty. No urological supervision was arranged after he left residential school, and he sought our advice purely for a recent problem of impotence. A routine test showed blood urea concentration to be 13 mmol/l (78 mg/100 ml), and a consequent intravenous urogram showed gross bilateral hydronephrosis. A videourodynamic study showed a flaccid but trabeculated bladder with minimal obstruction of the outlet. He was treated for nine months with an indwelling urethral catheter and subsequently with phenoxymethylamine. His glomerular filtration rate (as estimated by ^{99m}Tc-diethylenetriamine penta-acetic acid renography) improved from 33 to 74 ml/min and a recent intravenous urogram showed resolution of his hydronephrosis but with considerable residual renal parenchymal damage.

Case 5—A 36 year old man (myelomeningocele at level S2, incomplete) had never had any urological investigation until he presented with a short history of pain in the loin and haematuria. His serum

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creatinine concentration was 192 $\mu\text{mol/l}$ (2.2 mg/100 ml), and an intravenous urogram showed gross bilateral hydronephrosis with several large prostatic calculi. He has been managed for four years with an indwelling urethral catheter, with some improvement in his hydronephrosis but no diminution in his serum creatinine concentration. Because of repeated symptomatic urinary infections the prostatic stones have been removed.

Case 6—A 25 year old man had had a myelomeningocele at level S1, incomplete. At the age of 7 years an intravenous urogram was normal though a cystogram showed appreciable residual urine. He then defaulted on follow up and was not investigated further until he was referred with a short history of lower abdominal pain. An intravenous urogram showed pronounced bilateral hydronephrosis with bilateral renal calculi and one large bladder stone (fig). The bladder stone was removed and after a short period of drainage by suprapubic catheter his hydronephrosis improved. He awaits sphincterotomy.

Comment

Patients with neuropathic bladder may present two problems. The first, urinary incontinence, is immediately obvious. The second, deteriorating renal function, is more easily overlooked as it is nearly always asymptomatic in its early stages. It is worth noting that serum concentrations of urea and creatinine are apt to remain misleadingly normal until renal damage is well advanced. In cases 1 and 5 the possibility of renal complications was considered only when they developed appropriate symptoms, and in cases 2, 3, 4, and 6 it was seemingly ignored because they had no such symptoms. In every case deterioration of the upper renal tracts had almost certainly begun months or even years before they were referred and it is likely that had they been treated earlier the results would have been better.

Upper urinary tract problems in patients with neuropathic bladder are nearly always the direct consequence of obstruction of the bladder outlet sometimes compounded by the effects of vesicoureteric reflux and infection of the urinary tract. In children with congenital lesions of the spinal cord, though the incidence of these complications increases with the extent of the neurological deficit,¹ even those with minimal paralysis are at some risk which continues into adult life. In our experience this risk is substantially greater in men than in women. Hydronephrosis can usually be reversed by adequate treatment of obstruction of the bladder outlet and the earlier this is done the less the likelihood of permanent renal damage.²

Adults with congenital neuropathic bladder deserve the same close urological care that is given to children, no matter how slight their disabilities appear and no matter how well they may seem. This can be achieved only by good liaison between

Intravenous urogram showing pronounced bilateral hydronephrosis with bilateral renal calculi and one large bladder stone.

paediatric and urological colleagues, particularly during adolescence, when the patients can be so easily lost to follow up. Patients with congenital neuropathic bladder need lifelong urological supervision.

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What might be the reason for urine darkening when exposed to light for several hours?

Urine that darkens on standing for a few hours is a classic sign of an *acute porphyria*—either acute intermittent porphyria (Swedish type porphyria), variegate porphyria (South African type porphyria), or hereditary coproporphyria. In these conditions there are metabolic defects in the haem synthetic pathway. Stimulation of haem synthesis, usually by exposure to a drug which induces the liver microsomal oxidising system, gives massive overproduction of the haem precursors δ -amino laevulinic acid and porphobilinogen, which are excreted in urine. On standing exposed to light and atmospheric oxygen, porphobilinogen in the urine spontaneously polymerises to give the deep red porphobilin that imparts the classic port wine colour. Some of the other causes of darkening of the urine on standing include:

Indicanuria—In protein malabsorption or with increased intestinal transit time tryptophan may be converted to hydroxyindole by bacterial activity in the gut, absorbed, conjugated in the liver, and excreted as indican. Urine containing a high concentration of indican may be dark, or may darken on exposure to air. Rarely, indican may be deconjugated and oxidised by bacterial activity in voided urine to give a deep blue or purple colour due to indigo blue.¹

Melanuria—In some cases of disseminated malignant melanoma the tumour may produce a melanogen that is excreted in urine. There is a generalised slate grey discoloration of skin, and the urine turns dark brown on standing as the melanogen is oxidised to melanin pigments.

Haemoglobinuria and methaemoglobinuria—The haem pigments may be oxidised on exposure to air to their methaem derivatives, which are brown. This is unlikely to have given rise to the question, since the initial pink or red discoloration would have been obvious.

Alkaptonuria—Garrod's classic inborn error of metabolism is characterised by the excretion of homogentisic acid in urine, which darkens on standing.

Food and drug dyestuffs—Ingested pigments may impart their colour to the urine. It is unusual for the colour to appear after the urine is voided, but some—for example, phenolphthalein—are indicators whose colour is apparent only under alkaline conditions, which may be produced by urea splitting micro-organisms as the urine stands.—ROSS BOSWELL, senior lecturer in chemical pathology, Southampton.

¹ Payne B, Grant A. Purple urine bags. *Lancet* 1978;*ii*:502.