ABSTRACTS FROM OTHER JOURNALS

AN EXPERIMENTAL INVESTIGATION INTO THE EFFECTS OF PROLONGED VESICO-URETERIC REFLUX: JOHN E. S. SCOTT (1964). *Brit. J. Urol.* **36**, 391.

The author describes some dynamic and structural effects of vesico-ureteric reflux produced artificially in seven female dogs by resection of the mucosal flap valve covering their ureteric orifices. Three dogs died (one from acute pyelonephritis) too soon after this procedure to allow proper study. The remaining four survived in good health and with sterile urine, and although manometric studies revealed that they had lost the increase in ureteric peristalsis that normally occurs with increasing bladder volume and pressure, neither macroscopic nor microscopic signs of infection could be found in the kidneys of the dogs when they were killed, 19 to 24 months later.

EXPERIMENTAL STUDY OF URINARY INFECTION AND VESICO-URETERIC REFLUX: JOHN E. S. SCOTT (1964). *Brit. J. Urol.* **36**, 501.

The author has studied the effects on four dogs of attempts to cause urinary infection by deliberately introducing organisms into either the bladder or a vein, some six or twelve months after artificially producing unilateral vesico-ureteric reflux by resection of the mucosal flap valve overlying the right ureteric orifice of each dog. He found it very difficult to cause any infection and it was never persistent; nevertheless the changes of chronic inflammation were found in the right-sided kidneys, but not in the left.

The conclusion from these two papers is that in dogs at any rate, vesico-ureteric reflux aggravates but does not seem to cause urinary infections of the kidney.

VESICO-URETERAL REFLUX DEMONSTRATED WITHOUT RADIATION EXPOSURE: A. D. AMAR (1964). *J. Urol.* **92**, 286.

The best method of detecting vesico-ureteric reflux is radiography, preferably ciné; this does, however, expose the patient, often a child, to the risks of X-ray. In this paper the author describes a method which needs neither X-rays nor radio-isotopes. Indigo-carmine is put into the bladder through a urethral catheter; the catheter is removed; the patient empties his bladder; a cystoscope is then passed; any residual dye still in the bladder is removed; the ureteric orifices are observed through the cystoscope, and vesico-ureteric reflux diagnosed if a blue efflux is seen. The bladder mucosa absorbs little, if any, of the dye, which cannot, therefore, be excreted by the kidney.

T. NEWSAM, F.R.C.S.E.

MANAGEMENT OF NEUROGENIC URINARY BLADDER IN PARAPLEGIC DOGS BY DIRECT ELECTRIC STIMULATION OF THE DETRUSOR: MARTIN SCHAMAUN and ADRIAN KANTROWITZ. *Surgery*, Oct. 1963, Vol. 54, No. 4, p. 640.

Renal infection, calculosis, amyloidosis and hydronephrosis still cause an appreciable morbidity and mortality among paraplegic patients. They are all largely attributable to our present unsatisfactory management of the paraplegic bladder. For this reason interest in the United States and Canada has been devoted to the development of devices to empty the bladder of paraplegic dogs by electrical stimulation. In this account, a description is given of an investigation on 18 mongrel dogs, in whom electrodes were implanted in the bladder. In nine of the dogs the cord was divided; bladder pressures were recorded and the bladder was electrically stimulated and the effects of prostigmine methylsulphate were observed.

Each stimulation raised intravesical pressure to between 40 and 90 cm. pressure of water and resulted in 50-80 ml. urine flow. However, in the paraplegic dogs in the later

stages, the emptying was incomplete despite pressures which were higher than that achieved immediately after cord transection. This failure was attributed to spasticity of the striated external sphincter muscle resulting in increased intra-urethral resistance.

J. R. SILVER, M.R.C.P.E.

AN EXPERIMENTAL STUDY OF THE TREATMENT OF THE NEUROGENIC BLADDER: BRADLEY, W. E., WITTMERS, L. E. and CHOU, S. N. (1963). *J. Urol.* **90**, 575-582.

A method of treating the neurogenic bladder of paraplegic dogs by direct electrical stimulation is described. The signal is transmitted through the skin, by a flat circular-loop antenna, placed 1 to 2 inches from the skin over the implanted receiver. The receiver, approximately 2½ cm. in diameter and 1 cm. thick and coated with silastic, is implanted subcutaneously, lateral to the sheath of the rectus abdominis. No battery for the receiver is required as power output is derived from the transmitted signal to which the receiver is tuned. Using transperitoneal exposure, tape electrodes were sutured anteriorly and posteriorly to the serous fundus of the bladder. A biphasic pulse, 1-5 milliseconds in duration, 5-15 V, as measured across the bladder, with the bladder at body temperature, was sufficient to produce evacuation.

The experimentally produced lesions were either transection of the spinal cord at the cervico-thoracic junction or destruction of the cauda equina. Over-distension of the bladder made difficult emptying response to stimulation; restoration to normal excitability after release of distension required 4-8 hours. Bladder response was observed to be diminished after the use of indwelling catheter.

The difficulties encountered were spread of the stimulus to the contiguous structures and pain on stimulation in cauda-equina dogs.

The longest surviving dog is well and alive 13 months after implant. He is apyrexial, has sterile urine and normal renal function.

EFFECT OF LOWER URINARY TRACT INFECTION UPON URETERAL FUNCTION: SCHOENBERG, H. W., BEISSWANGER, P., HOWARD, W. J., KLINGEMAIRER, H., WALTER, C. F. and MURPHY, J. J. (1964). *J. Urol.* **92**, 107-108.

The bladder was exposed suprapubically in 22 dogs, a pouch was formed between the muscular coat and the mucosa into which was inserted a 1½ cm. circle of agar cut from a plate containing pathogenic B. *Proteus vulgaris* and a 2 sq. cm. area of the bladder adjacent to the agar implant crushed. All of the animals developed persistent infection of the lower tract. The animals were examined by ciné-fluorographic cysto-urethrography at intervals of 2, 4 and 6 weeks following the operation. Four of them exhibited bilateral and one unilateral vesico-ureteric reflux on all post-operative studies; three more animals began to show reflux after 4 weeks and another one after 6 weeks. Altogether in 9 of 22 experimental animals reflux developed during the period of observation. Cystometry revealed that intravesical voiding pressure did not exceed 40 cm. of water which had been found normal on non-infected animals. It appears, therefore, that infection alone, without evidence of bladder-outlet obstruction or alteration of the hydrodynamics of the lower tract, may result in vesico-ureteric reflux.

H. FRANKEL, M.R.C.P.

NEUROGENIC PATHWAYS CONCERNED IN REFLEX VASODILATATION IN THE HAND WITH ESPECIAL REFERENCE TO STIMULI AFFECTING THE AFFERENT PATHWAY: O. APPENZELLER and H. SCHNIEDEN. *Clin. Sci.* 1963, Vol. 25, No. 3, pp. 413-421.

Kerslake and Cooper showed in 1950 that vasodilatation of the hand occurred in response to heating of the trunk or legs by means of a radiant heat cradle. They subsequently

showed that the afferent pathway was via the sympathetic nervous system. However, the site of the receptors and their afferent pathway is unknown. In the present study, the authors studied 12 patients with lesions in the brain stem, spinal cord and peripheral nervous system, and 11 normal volunteers.

They measured the hand blood flow by venous occlusion, plethysmography, and heated the body by a heat cradle. It was shown that the increase in hand blood flow produced by 40 seconds cradle heating, was dependent on intact nerve afferents and efferents in structures higher than the brain stem.

Reflex vasodilatation in the hand was also produced by infra-red and ultra-violet irradiation of the chest. With the latter low energy source of radiation accommodation of the reflex response occurred; a rise in skin temperature of the chest produced by a rubefacient cream was not associated with a significant change in hand blood flow.

PERIPHERAL NERVE IMPLANTATION IN EXPERIMENTAL PARAPLEGIA: K. A. J. CSEUZ and T. J. SPEAKMAN. *J. of Neurosurgery*, July 1963, Vol. 20, No. 7, p. 557.

Thirty-seven adult mongrel dogs were rendered paraplegic by removing a 1 cm. segment of the spinal cord in the thoracic region. In 23 dogs the sympathetic trunk was re-implanted in the distal stump of the severed spinal cord, and in 10 dogs a similar procedure was carried out with an intercostal nerve. In four dogs no re-implantation procedure was carried out. These served as controls.

The four control dogs moved about either by dragging their paralysed hind limbs or by 'straining forward and by flexion of the neck. The paralysed portion of the body was next visibly raised off the ground mainly by the pull of the longissimus dorsi muscles. Reflex stepping of the hind limbs propelled the body forward whenever the hind paws were in contact with the ground.'

In contrast to these control findings, three of the dogs with sympathetic implants and one of the dogs with an intercostal nerve implant demonstrated almost normal walking activity. Stimulation of the implanted sympathetic chain evoked a potential in the distal severed segment of the cord. Re-operation in which the nerve implant was destroyed caused a deterioration in their walking ability. Post mortem showed active growth of the implant had taken place.

The implications of this work are discussed.

J. R. SILVER, M.R.C.P.E.