transfer. Infected staff may not return to the unit until their S.H. test has become negative. Such staff are then of great value to the unit since they are regarded as immune.

Patients are immediately divided into three groups on the basis of S.H. testing: (*a*) infected—patients with hepatitis or S.H.-positive carriers; (*b*) uninfected—S.H.-negative patients who during the preceding six months have neither had contact with S.H.-positive patients or their blood nor received unscreened blood or plasma; and (*c*) potentially infected—the remainder who, though S.H.-negative, could be incubating the infection. Patients in the first group are isolated from all other patients, while those uninfected—mainly on home dialysis—may mix among themselves but must be isolated from the other groups. Potentially infected patients are isolated from each other so far as possible and also from those in the other two groups. Ultimately, the former are allocated to these two groups, but clearly this cannot be done until six months have elapsed since finding the last positive S.H. test on the unit.

REPEAT TESTING

In our experience repeated S.H. testing of infected patients is unnecessary, as most infected dialysis patients become permanent carriers, and even if their S.H. test becomes negative they should still be regarded as infectious. A separate dialysis unit for each group is required if segregation is to be effective.

These measures should continue until the outbreak is under control—that is, until no new cases have been associated with the unit during the previous six months. The development of hepatitis in relatives of S.H.-positive patients on home dialysis need not, however, affect the hospital programme.

Transplantation of S.H.-positive patients seems to us to be too dangerous unless an immune hospital team is available.

Future Implications

Hepatitis has introduced doubts about the future of dialysis and transplantation. Centres should be designed to permit easy division into three self-contained subunits in the event of an outbreak of hepatitis.

To encourage recruitment of nursing staff to dialysis units incentive payments ("leads") should be made, similar to those paid to nurses on psychiatric and geriatric units. We suggest that hepatitis contracted in dialysis and transplantation units should be regarded as an industrial hazard for the purpose of the National Insurance Acts, and that those exposed to these high risks should in addition be covered by adequate life assurance.

We are indebted for their help to Drs. G. Turner, G. Bruce White, D. Watson, and J. Pennington. We would also like to thank Misses S. Hall and O. Dinsdale for biochemical laboratory work. In addition, we acknowledge the help and loyal co-operation of numerous colleagues and staff on the unit.

Requests for reprints should be addressed to Dr. B. J. Haiwe.

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Investigation and Treatment of Resistant Urinary Incontinence

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British Medical Journal, 1971, **1**, 543–545

Recently much interest has been expressed about the management of urinary incontinence.[1 2] This paper describes the investigation and management of 211 consecutive cases of incontinence of urine seen in the unit for electronic control of incontinence at the Shaftesbury Hospital (St. Peter's Hospitals and Institute of Urology, London) since October 1967.

Patients and Methods

The patients are investigated and subsequently treated in a self-contained unit (see Plan). Patients are usually admitted on Monday afternoon and discharged on Friday afternoon of the same week. During this period the following investigations are undertaken: (1) physical examination, including a careful neurological examination; (2) electromyographic (E.M.G.) studies of the legs and perineum; (3) cystometrogram; (4) urethral pressure profile;[3-5] (5) pressure flow study; (6) examination under anaesthesia—which includes the urethral pressure profile and a cystometrogram, both these investigations being undertaken in conjunction with perineal stimulation of the pelvic floor;[6] (7) intravenous pyelogram; and (8) micturating cystogram. The last investigation is undertaken after the preliminary investigations have been completed, at a time when treatment has been decided on, and repeated with and without the prescribed treatment as applicable.

Essentially the patients seen in the unit have "resistant" incontinence—that is, incontinence which has persisted despite orthodox treatment. Thus the patients referred have almost always undergone surgical or gynaecological procedures, or have incontinence for which there is no apparent remedy. In this study we have found that a patient's incontinence must be clearly defined as being primarily due to either sphincter disturbance—for example, female stress incontinence or male postprostatectomy incontinence—or detrusor dysfunction (neurogenic incontinence of all varieties, such as multiple

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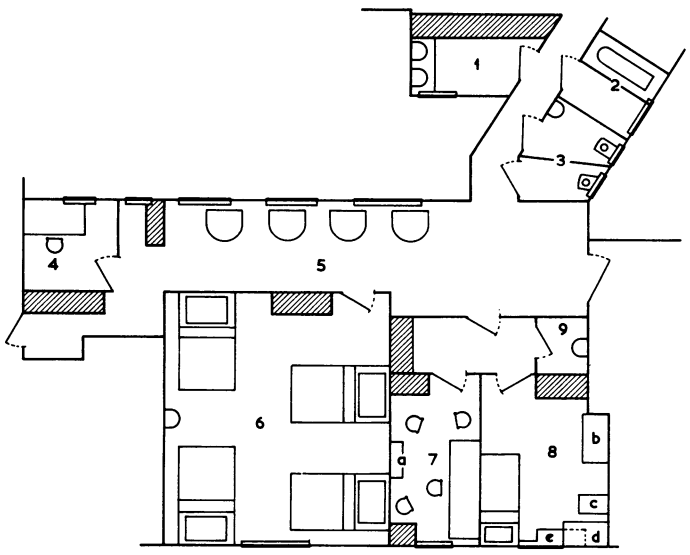

Plan of unit. (1) Sluice, (2) bath, (3) lavatories, (4) sister, (5) waiting area, (6) 4-bedded ward, (7) surgeon, (8) examination room, (9) changing room. Shaded areas are for storage. (a) *X*-ray box, (b) pressure recorders, (c) oscilloscope, (d) E.M.G. monitor, (e) flow meter.

sclerosis or spina bifida and enuresis). Only when the aetiological diagnosis has been made can treatment be rationally directed.

Results

The age and sex distribution of the 211 patients is given in Table I; 79 were male and 132 female, a sex ratio of 2 : 3.

TABLE I—*Age and Sex Distribution*

	Age in Years								
	⩽10	11—	21—	31—	41—	51—	61—	⩾71	Total
No. of males	12	10	8	6	4	12	20	7	79
No. of females	8	10	12	10	22	38	17	15	132

AETIOLOGICAL FACTORS

The aetiological factors responsible are shown in Table II and the patients' ages are correlated with these factors in Table III. The post-traumatic, stress, congenital, and postureterocolic anastomosis groups are examples of sphincter deficiency, and the psychogenic, enuretic, and neurogenic groups are examples of detrusor dysfunction. Some of the congenital cases—for example, spina bifida—may also be cases of detrusor dysfunction.

The peak in the women aged 51-60 (see Table I) is due to the number of patients attending in this age group with stress incontinence, while that in the men aged 61-70 is due to the number of patients presenting with prostatectomy incontinence.

TABLE II—*Aetiological Factors*

	Males	Females	Total
Stress	0	70	70
Neurogenic	26	38	64
Post-traumatic	37	1	38
Enuresis	9	11	20
Psychogenic	4	5	9
Congenital	3	5	8
Ureterocolic	0	2	2
Total	79	132	211

TABLE III—*Age Groups of Patients Correlated with Aetiological Factors*

		Stress	Neurogenic	Post-traumatic	Enuresis	Psychogenic	Congenital	Ureterocolic
Male	Youngest		8	9	4	33	17	
	Oldest		71	80	29	38	23	
	Mean		38·4	52·8	16·5	35·5	17·3	
Female	Youngest	7	6		7	21	6	56
	Oldest	87	82		30	53	38	77
	Mean	51·3	51·2	55	16·2	40·4	24·0	66·5
Both sexes	Youngest		6	7	4	21	6	
	Oldest		82	80	30	53	38	
	Mean		46·7	52·8	18·4	39·3	21·5	

TREATMENT

The type of treatment recommended and usually undertaken is shown in Table IV. When orthodox surgical procedures are indicated the patients have usually been sent back to the department of referral with the appropriate recommendation. Otherwise all treatment has been undertaken in the department, including all the implants and most of the urinary diversions. Many methods of treatment are available, but

TABLE IV—*Type of Treatment Given, if any, in the 211 Cases*

		Total	Post-traumatic	Stress	Psychogenic	Enuresis	Congenital	Neurogenic	Ureterocolic	Failure		
										Primary	Secondary	Outright
Surgery	Urethral dilatation	1						1				
	Urethroplasty	3	3									
	Sphincterotomy	1						1				
	Transurethral resection	6	1					5				
	Surgical repair	18		18								
	Gracilis sling	1						1				
Implant		33	18	3		1	3	8				8
Anal plug		21	12	1	1		2	3	2	2		4
Vitalograph		4		2	1			1				
Electronic Pessary		40		32	1	1	1	5		21	1	2
Clip		32		24		3		5		2	2	2
Drugs	Antibiotics	1				1						
	Emepronium	29	2			7		20		1	2	
	Imipramine	29	1	7	5	12		4				
	Diazepam	1				1						
	Pyridostigmine	3						3				
	Dexampheta-mine	1				1						
2-hourly emptying		6						6				
Catheter		3		1				2				1
Diet		1		1								
Diversion		12		1				11				
No treatment		9	1	1	1		2	4				
Refused treatment		3		2				1				
Overall failure (34)		12									5	17

most patients have been treated by implant, electronic pessary, pubovaginal spring device, anal plug, emepronium, or imipramine.

Evidently the first line of approach often proves unsatisfactory ("primary failure") (Table IV); and 26 of our patients needed alternative treatment. Of these, only five did not obtain benefit ("secondary failures"). Seventeen patients were classed as outright failures from the start. No recommendation was possible in nine cases, and three patients refused the suggested treatment. The overall number of successes was 177, and overall failures 34.

Discussion

An electronic implant for controlling incontinence of urine was first described by Caldwell *et al.*,[7 8] and this method of

treatment is now well established.[9,10] S. Alexander and his colleagues described an external stimulator in 1968 and reported their further experience in 1970.[11,12] Their most recent report is on 18 patients—good results were obtained in 12 out of 16 non-neurogenic, and in none of two with neurogenic incontinence.

The use of an anal plug for the control of faecal and urinary incontinence was first described by B. R. Hopkinson and R. Lightwood.[13,14]

From this unit P. R. Riddle and his colleagues described early results in 1968,[6] and N. W. Harrison and P. J. Paterson completed a survey of 21 patients treated by electronic pessary in 1970.[15] In the latter series of 21 patients 11 obtained benefit from the treatment and 10 did not.

The results of a preliminary trial with the pubovaginal spring device have already been reported.[16] In a larger series the device was used in 43 patients, of whom 33 received some benefit.[17]

Up until now reports have been confined to the use of a single device, usually the one which has been developed in the department submitting the report. We feel that it is imperative in the patients' interests to provide the whole range of treatment. The function of this unit is thus twofold—firstly, to investigate each patient comprehensively so that the diagnosis and classification of the incontinence may be accurate; and, secondly, to adopt whatever treatment these investigations have shown to be the most appropriate.

We believe that the results of our present study, in which 177 out of 211 patients received benefit, justify the care necessary in investigating these patients and the expense of equipping and maintaining such a comprehensive unit. We wish to emphasize that any unit attempting to help incontinent patients must not confine itself to one particular device but be able to select and use from the currently available range of treatment that device or drug regimen which detailed investigation has shown to be most likely to succeed.

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Therapeutic Conferences

Angina Pectoris—II

FROM THE DEPARTMENT OF THERAPEUTICS AND PHARMACOLOGY, ABERDEEN UNIVERSITY

British Medical Journal, 1971, 1, 545–547

Case 2—Crescendo Angina

DR. WOOD: This man is aged 40, and has had increasing angina for five years which has occurred increasingly at rest in the past few weeks. He is a non-smoker, but is 10 kg overweight. There is a bad family history of myocardial infarction. He has an arcus senilis, his blood pressure is 130/80 mm Hg and the heart is neither enlarged nor in failure. The E.C.G. has ischaemic features. He was admitted to hospital for stabilization of dosage of an oral anticoagulant.

PROFESSOR MACGREGOR: This man has a poor prognosis. Why did you anticoagulate him?

DR. WOOD: The role of anticoagulants is much debated, but there is less argument about their use in men under 45 who develop crescendo angina. We prefer warfarin to phenindione, which causes rashes and some severe hepatic and renal disturbances. The patient at present has a thrombotest of 10%, within the therapeutic range of 7–12% of the control value.

PROFESSOR MACGREGOR: What about his serum cholesterol?

DR. WOOD: This is 390 mg/100 ml, but the plasma

Appointments of Speakers
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J. C. PETRIE, M.B., M.R.C.P., Lecturer in Therapeutics
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