in treated and "control" groups.^{5,6} In some of these studies, however, the antimicrobial treatment was inadequate or intermittent or monitoring of compliance was inadequate: so not surprisingly little difference was found between the control and treated groups. These results have combined with the mistaken belief that further damage and scarring did not occur in schoolchildren to allow lackadaisical treatment. The authors of the excellent Newcastle study concluded that while continuous prophylactic antimicrobial treatment was highly effective in preventing and reducing urinary tract infection in treated girls as opposed to controls, screening of schoolchildren should be postponed until a non-radiological method could be devised to detect those with renal scarring.⁵ We now have this method—ultrasound. Should we screen now?

The paper from Smellie *et al* brings hard facts to bear on a previously speculative subject (p 1957). They studied the development of renal scars in 74 children (including some with latent urinary tract infection) who had 87 kidneys affected including 74 previously normal. Thirty four children were aged 5 years or more when scarring occurred. Diagnosis and treatment had been delayed in 45, and in 58 symptomatic urinary tract infection occurred after a normal intravenous urogram and before scarring occurred. Vesicoureteric reflux was detected in 91% of these children. Interestingly, few scars, in this series, appeared in children aged over 7. Many paediatricians will see these results as confirming their view that both urinary tract infection and latent urinary tract infection should be treated.

The conclusions of the authors of the collaborative study are that early diagnosis, prompt effective treatment, investigation, and long term supervision of urinary infection are essential if renal scarring is to be reduced both in those with urinary tract infection aged 0-5 years and also older children (p 1957). These are admirable sentiments, but the deed may prove harder to achieve than the wish. While the present study proves the occurrence of scarring it does not prove how to prevent it. The facts do not support the use of ureteric reimplantation as a method of preventing scars. Although no satisfactory controlled long term trial has yet been published of long term continuous prophylactic antimicrobial treatment (properly supervised, compliance ensured) to prove that scars are less likely and kidney growth more satisfactory than when infections are not prevented (or left untreated), the supposition is strongly in favour of treatment. Certainly leaving a child with urinary tract infection untreated, uninvestigated, and unsupervised in the future can no longer be justified—if it ever could. In recent years the fashion has been for shorter and shorter courses of antimicrobial treatment and for a rather slipshod attitude to following up infected babies and toddlers until middle school age. Smellie and her coworkers have effectively shattered complacency, and we must all look anew at this problem.

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Recurrent ventricular tachycardia

Depending on the exact definition of sudden death there are between 50 000 and 100 000 victims in Britain each year¹—and increasing numbers of potential victims are being resuscitated from sudden collapse outside hospital. Most of these episodes are not associated with myocardial infarction,² though most patients have coronary artery disease.³

The exact mechanism of sudden death is not understood. Recently published information from fortuitously taped electrocardiographic recordings during sudden death has shown, however, that ventricular fibrillation is invariably preceded by a period of ventricular tachycardia.^{4,5} In two recent series of patients who had survived cardiac arrest outside hospital ("failed" sudden death) ventricular tachycardia could be induced in 61% of 153 patients, and ventricular fibrillation was rarely induced directly but was caused by degeneration of ventricular tachycardia.^{6,7} In patients who present with recurrent ventricular tachycardia the annual mortality from cardiac death is 30%, and in most of these death is sudden.⁸

Ventricular tachycardia is now known also to be responsible for symptoms in clinical settings where until recently it had been almost unsuspected. For example, several studies have shown that ventricular tachycardia may be induced in half the patients with recurrent syncope of unknown origin; treatment based on the results of provocation testing has reduced the risk of recurrence.^{9,10} The cause of syncope in patients with electrocardiographic evidence of intraventricular conduction disorders is generally assumed to be intermittent complete atrioventricular block. These patients, too, have a propensity to ventricular tachycardia, which could account for syncope.¹¹

The correct diagnosis of ventricular tachycardia is achieved by painstaking inspection of the current electrocardiogram and detailed comparison with all previous recordings. A clear understanding of current diagnostic criteria is essential.¹² The initial management strategy will depend on the clinical presentation. Intravenous infusion of an antiarrhythmic drug is appropriate if the tachycardia is haemodynamically stable, otherwise direct current shock is the method of choice. After successful termination of the tachycardia treatment must be given to prevent recurrences. In patients with coronary artery disease interventions designed to increase coronary blood flow or improve myocardial efficiency rarely affect the incidence of ventricular tachycardia, and long term antiarrhythmic treatment is usually required. When appropriate, the drug which has been effective intravenously may be given by mouth. Drugs of class I (such as disopyramide, tocainide, and flecainide) and class III (such as amiodarone and sotalol) are most effective for prevention of recurrences. At present, however, the efficacy of any chosen drug cannot be predicted and a therapeutic trial has to be made in every case. Empirical treatment with follow up on an outpatient basis is not only unreliable but potentially dangerous, especially if the patient has unstable tachycardias. Continuous electrocardiographic monitoring (tape or telemetry) with supervision in hospital is appropriate for patients with frequent episodes but is unsuitable if attacks are rare. Antiarrhythmic drugs may cause a reduction in the frequency of ventricular extrasystoles without suppressing sustained tachycardia or fibrillation¹³; when this effect has been shown by electrocardiographic

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monitoring it must not be assumed to reflect adequate protection. Furthermore, spontaneous variation in the type and frequency of ventricular extrasystolic activity must be taken into account when assessing the effect of drugs.¹⁴ In a few patients ventricular arrhythmias may be provoked consistently by stress testing, and this can be used to assess the efficacy of drugs.

Intracardiac electrophysiological studies have contributed greatly to the management of ventricular tachycardias once cardiac stimulation had been shown to induce many clinical tachycardias.¹⁵ These techniques have recently been reviewed by Buxton and Josephson.¹⁶ An important part of the method is the identification of a drug which will suppress the induction of tachycardia. Swerdlow *et al* have shown that this effect predicts improved survival.⁸ By contrast, failure to identify effective suppressant treatment by intracardiac studies is a strong predictor of sudden death. Another important aspect of these studies is the identification of proarrhythmic effects, which may be seen with all class I and III drugs.^{17,18} Given the limitations mentioned above, tape monitoring can also be helpful in detecting these effects—but they may be identified more safely and reliably by intracardiac stimulation.¹⁹

If drug treatment fails, alternatives include electrophysiologically guided surgery¹⁶ and implantable electronic devices.²⁰ Both approaches are firmly based on the results of intracardiac studies collected over the past decade.¹⁶

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Artificial urinary sphincters

Urinary incontinence, defined as an involuntary loss of urine, is at best an embarrassment and at worst a social disaster. Continence depends on the urinary sphincters, and these are complex and clever mechanisms: they open and close on demand, hold tight while we cough and sneeze, and rarely let down the normal adult. So we should not be surprised that biomechanical research has had such a hard time finding a suitable substitute. Appropriate inert materials with trouble free mechanics are needed together with a suitable surgical technique for implantation.

Firstly, the surgeon must determine why the patient is incontinent, for only a few will be suitable for an artificial sphincter. Patients with incontinence due to overflow from obstruction or in association with infection often need simpler management. The activity of the bladder muscle (detrusor) must be known: if the bladder is atonic and the sphincters adequate then clean intermittent catheterisation is all that is needed. Instability or overactivity of the bladder remains one of the major challenges in urology, for such contractions of the bladder may overcome the normal sphincter mechanisms and give a type of incontinence which is very difficult to treat.

The ideal patient for an artificial sphincter should therefore have a bladder of adequate capacity and compliance with no instability and no outflow obstruction. The bladder also needs to empty either with a detrusor contraction or manual compression. Though methods have been devised to modify some or all of these factors this assessment remains true.

Early attempts to increase the resistance of the outflow tract artificially relied on unvaried pressure on the tissues of the bladder neck or urethra. The most subtle of these was the Kaufman device—a small silicone filled bag fixed against the male urethra to increase the passive resistance.¹ Long term results show a high incidence of urethral erosion or stricture, infection, failure to control the incontinence, and the need later to remove the device.² Simple in concept and easy to insert, these devices have now been superseded by newer developments.^{3,4}

Brantley Scott and his associates have developed an ingenious artificial sphincter, the latest modification being the AMS 800.^{3,5-7} An inflatable cuff is implanted around the bladder neck or bulbous urethra. The pressure within the cuff is maintained by a pressure regulating balloon in the pelvis away from the cuff, and the two are connected by a control pump placed subcutaneously in the scrotum or labia. Squeezing the pump empties the cuff to allow voiding, and after several minutes the cuff automatically reinflates. The latest modification in the form of a poppet valve allows deactivation of the cuff, used, for instance, in the initial healing stages.

This type of inflatable artificial sphincter is better than its predecessors, but erosion, mechanical failure, infection, and expense are all continuing problems. With careful selection of patients and scrupulous surgical technique, however, up to 90% of both adults and children can be rendered acceptably continent.⁷

Patients being considered for surgery need full urodynamic, radiological, and neurological examination. Simpler treatments such as pharmacological manipulation or standard surgical intervention should be tried first.⁸ Once a suitable patient is found outflow obstruction must be relieved, and this may mean a complete sphincterotomy or bladder flap urethroplasty.⁷ The best success has been