mean postoperative stay increased as the size of the hospital decreased. Heasman and Carstairs[2] found a shorter mean postoperative stay in Scottish teaching hospitals than non-teaching hospitals. They also suggested that the duration of postoperative stay at smaller hospitals might be related to periodic visits by a consultant from another hospital. In hospitals 5 and 6, and particularly in 7 and 8, the duration of postoperative stay was related to operating days and the days of consultant visits.

West and Roberts[4] suggested that increased pressure on hospital facilities had shortened the mean postoperative stay for a hospital management committee area. One measure of pressure on facilities is the length of waiting lists, and patients at the larger hospitals (with short postoperative stays) did wait longer for their herniorrhaphies than patients at the smaller hospitals. Hospital 2 had a longer waiting list than hospital 1 (14·3% compared with 3·4% of patients waited more than two years). Hospital 2 sent more patients to the convalescent home than did hospital 1 (29·1% compared with 12·6%). Despite these two findings, the postoperative stay in hospital 2 was longer than that in hospital 1.

As expected, the development of postoperative complications significantly increased the mean postoperative stay. Ashley *et al*[5] found comparisons between hospitals hard to make because of the unstandardised nature of clinical records. In the present study more postoperative complications were recorded at the larger than the smaller hospitals. This may simply reflect a difference in recording in the case notes.

The results of this study emphasise the extent of the variation in stay after elective inguinal herniorrhaphy. Assuming that a shorter mean postoperative stay is economically desirable and has no detrimental effects on the patients,[6 7] our results strongly suggest that changes in hospital organisation[8] rather than consultant attitudes will be required.

We thank the consultants who gave permission for their cases to be studied; the medical records staff for their help; and the Department of Health and Social Security, who funded the study.

Further details on factors having a significant effect on postoperative stay may be obtained from WEW.

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SHORT REPORTS

Sphygmomanometers: errors due to blocked vents

Inaccuracies in the performance of mercury sphygmomanometers are widely reported. Sources of error include design,[1 2] operation,[3 4] and maintenance.[5] Despite reported inaccuracies few attempts have been made to measure the actual errors produced at different rates of fall of the mercury column. These errors occur when the chamois leather vents at the top of the glass tube and mercury reservoir become partially blocked.[5] We have checked the performance characteristics of 32 ward sphygmomanometers in a teaching hospital and report here the results.

Methods and results

The instrument under test was disconnected at the tubing between mercury reservoir and cuff. The open ends of the tubing were then connected to two limbs of a metal T-piece. The third limb of the T-piece was connected to a properly functioning mercury column. The two mercury columns were aligned in the viewfinder of a TV camera connected to a video recording system. The cuff from the instrument under test was wrapped around a rigid tube and the two mercury columns pressurised to 180 mm Hg. The pressure was released at various rates, including 2, 5, 10, and 20 mm Hg/s. The descent of the mercury columns was recorded on videotape. Stop-action images on the video monitor were then used to check errors at pressures of 150, 120, 90, and 60 mm Hg. The 32 sphygmomanometers were also checked for scale zeroing and other deterioration.[5]

Numbers of sphygmomanometers in each error range taken at a true pressure of 120 mm Hg. All errors are positive

Rate of fall (mm Hg/s)	Error range (mm Hg)							
	0–	2–	4–	6–	8–	10–	15–	25–35
2	8	13	6	4	1			
5	7	7	10	6	1	1		
10	6	5	7	8	4	2		
20	5	3	6	6	4	3	4	1

The table shows the relation between the rate of fall and the error. A correction was applied for errors resulting from poor zeroing. A + sign indicates that the instrument under test gave raised pressures. Eleven sphygmomanometers had zeroing errors of +1 mm Hg or more. Thirteen had leaks, five defective control valves, and 12 other functional defects—for example, damage to hinges, which prevented the mercury column being positioned vertically. Fourteen of the instruments were wall-mounted.

Comment

The combination of lack of maintenance and excessive rate of fall produced large errors. The errors were caused by a deterioration in the frequency response owing to debris blocking the vents in the glass tube and reservoir. Only five instruments produced results the same as the reference instrument. All others gave pressures exceeding the true value. Twenty-four of the sphygmomanometers produced errors of between +2 and +10 mm Hg at the slowest rate of fall. The error increased at faster rates. The worst result was an error of +33 mm Hg. By following recommended practice of a rate of fall of 2 mm Hg per heart beat such a large error may be avoided. But such an error can occur when the operator inflates the cuff and then lets the mercury column fall quickly until it seems to be close to the expected systolic value.

The table gives results obtained only at a true pressure of 120 mm Hg. Similar errors are produced at lower pressures. The median error for a rate of fall of 5 mm Hg/s at a true pressure of 120 mm Hg was +5·0 mm Hg; at a true pressure of 60 mm Hg it was +4·5 mm Hg. Half of the instruments produced errors of between +4·5 and +14 mm Hg at a true reading of 60 mm Hg and deflation rate of 5 mm/s.

In the hospital concerned faulty sphygmomanometers are returned to the manufacturer for repairs, this action being initiated by the user. All sphygmomanometers tested were in regular use, and no complaints had been made about their performance.

These findings reinforce the need to make adequate arrangements

for maintaining sphygmomanometers and for instructing users in the correct method of operation.

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Two patients with schizophrenic-like psychosis after treatment with beta-adrenergic blockers

We have recently seen two patients suffering from psychotic illnesses which developed during treatment with β-adrenergic-blocking drugs that had been prescribed for cardiovascular illnesses.

Case 1

This 53-year-old woman's sister, son, and cousin suffered from schizophrenia. She had no psychiatric history, although she had always been suspicious of people. She had been taking β-adrenergic-blocking drugs for angina for several months, and three weeks before admission the dose of propranolol hydrochloride had been increased to 40 mg three times a day. Since then she had gradually become more psychotic with delusions that there were "mental breakdowns" and "abuse" from the occupants of a house near where she lived, and she spent much time looking for the house. She also believed that her telephone calls were being intercepted and repeatedly referred to "the north London conspiracy." She had auditory hallucinations, claiming that two or three voices were talking together about her and making a running commentary on her behaviour. Immediately before admission she had been wandering all over London and had not slept for a week or eaten for three days; the propranolol had then been withdrawn by her GP. On admission trifluoperazine and haloperidol were substituted. She returned home against medical advice after two days in hospital. Since discharge she has taken no neuroleptic medication and has remained well for three months, with no psychotic symptoms.

Case 2

This 66-year-old woman had no family or personal history of psychiatric illness. She had been treated for two years with oxprenolol hydrochloride 80 mg three times a day, but nine months before referral the dose had been increased to 160 mg thrice daily. She had gradually become withdrawn and began to suffer from delusions and hallucinations. She believed that the people next door were plotting against her, could see into her room, and had placed an electrical gadget in the wall to watch her and read her thoughts. She heard voices repeating her thoughts out loud and commenting on her actions. Before her illness she had been on good terms with her neighbours, and the condition came to light one evening when she started screaming at the window that she was being burgled and the police were called. Under outpatient supervision her dose of oxprenolol hydrochloride was reduced to 80 mg three times a day and treatment was started with 10 mg of trifluoperazine and 50 mg of orphenadrine thrice daily. Her mental state improved rapidly and after one week she was free from all her psychotic symptoms and remained well at one month's follow-up.

Comment

Depression and delirium are well-known side effects of beta-blockers,[1] and visual hallucinations have also occurred. The auditory hallucinations experienced by both these patients were first-rank Schneiderian schizophrenic symptoms. Both patients were undoubtedly suffering from a schizophrenic-like illness rather than a toxic confusion state. The relationship between their psychoses and their treatment with beta-blockers may have been a chance one, as both schizophrenia and treatment with beta-blockers are common. Nevertheless, in each case the onset of psychotic symptoms dated from the increase in dose of the drugs, and recovery was quick and complete when the dose was reduced; the first patient recovered with very little neuroleptic medication.

Whether beta-blockers have a central action is uncertain, and their effect in treating anxiety is probably mediated through the cardiovascular system.[2] Biochemical and neuropharmacological studies in animals have provided convincing evidence of the existence of central β-adrenoceptors, and antagonists have been shown to act on them in animals.[3] In man electrocardiographical changes have been shown in chronic schizophrenic patients taking increasing doses of propranolol,[4] and the double-blind trial of Yorkston *et al* clearly showed that propranolol was an effective treatment for patients with schizophrenia who had not improved on neuroleptic medication.[5] The effect of the beta-blockers on these two patients therefore seems to be paradoxical. The role of beta-blockers in the mechanism of schizophrenia is clearly complex and requires further investigation.

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Serum ferritin concentration and oral iron treatment in patients on regular haemodialysis

Iron deficiency contributes to the anaemia of some patients receiving maintenance haemodialysis. Oral iron can both prevent and correct iron deficiency anaemia and marrow iron depletion in such patients,[1 2] and continuous oral iron is, therefore, commonly prescribed. Iron overload is generally assumed to be unlikely with oral—as distinct from parenteral—iron, but this has been little studied and the usual dose required to maintain haemodialysis patients in iron balance has not been established. We have used serum ferritin concentration as an index of iron state and here report observations in three groups of patients undergoing haemodialysis and taking different amounts of oral iron.

Patients, methods, and results

Sixty-one patients (36 men) were studied. All but one patient underwent dialysis for two 10- or 11-hour periods or three 5- to 8-hour periods per week using standard Kiil or Multipoint dialysers (Meltec Ltd). The single exception underwent dialysis for three 6-hour periods weekly using an X23 coil dialyser (Extracorporeal). Membranes used, other dialysis techniques, diet, and supplements have been described.[1] Patients had received dialysis for two months to 12 years (mean 42 months; 17 for less than 12 months). Twenty-seven patients had received blood transfusions. The mean transfusion rate for all 61 patients was 0·16 units/month. Only three patients had received intravenous iron at any time and more had received it within a year of starting the study. Great economy was exercised in blood sampling for routine investigation. This accounted for blood loss of 5 to 15 ml a month. On starting haemodialysis patients were prescribed ferrous sulphate 200 mg thrice daily (equivalent to 180 mg elemental iron daily) for one month, and those who tolerated this were then prescribed 400 mg thrice daily (360 mg elemental iron daily). A few patients were transferred to other preparations if intolerant to ferrous sulphate. Many had received oral iron before haemodialysis. Actual—as opposed to prescribed—daily iron intake was assessed by detailed and sympathetic questioning by two of us (AMC and JNF). Blood