under my care. The figures include patients admitted for elective orthopaedic surgery as well as those admitted as a result of acute trauma. The average age of patients currently in an acute bed is 78 years. Twenty-seven per cent of the patients have been in hospital for more than four months awaiting geriatric care. They are not well enough to return to the community. A further 18% of patients are also waiting geriatric management, so that 45% of female patients do not require a bed in an acute orthopaedic ward. This explains why patients may have to wait several years before they can be admitted for elective orthopaedic procedures.

The main problem is the aftercare of the geriatric patient. This depends not only on the provision of geriatric services but, probably more importantly, also on the social conditions in the area. For example, in our area 20% of the housing is substandard. The geriatric norm of 10 beds per 1000 people aged over 65 years is totally meaningless, as it does not take into account these differing social conditions. Patients who are fit enough to be discharged home, provided they had adequate home facilities, may have to remain in acute beds waiting for alternative arrangements because of their poor social conditions.

The most important statement in Dr Donaldson and colleagues' paper was in the last sentence: "These estimates, however, must be interpreted with caution. . . ." Until adequate provisions can be made for the aftercare of patients who needlessly occupy acute beds it would be suicidal to reduce the number of acute beds. This would lead to the total cessation of elective surgery, unnecessary pain and suffering, unnecessary working days lost owing to illness, and a totally unnecessary waste of money. It has been estimated that a single total hip replacement saves £12 500 in the male up to the age of 60; £5000 in a female up to the age of 60; and £1700 per individual aged 60-70.¹

Further studies are required, but the findings will be valueless unless the study includes an examination of all factors that may be responsible for patients unnecessarily remaining in acute hospital beds. Most clinicians are so aware of the acute shortage of beds that it must be very rare for these beds to be used inappropriately without very good reason.

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¹ Taylor, D G, *Proceedings of the Royal Society*, Series B, 1976, **192**, 145.

SIR,—Dr S N Donaldson and his colleagues (24 September, p 799) have undertaken an interesting study into the contentious subject of how many hospital beds are needed, but the statement in their summary that "the current provision of acute beds (2.0-2.5 per 1000 population) exceeds actual need" is not justified by their data for the following reasons.

(1) They have adopted a different definition of "acute beds" from that in current use in NHS planning circles. Theirs may or may not be a better definition, but they have excluded many beds that would usually be called "acute" and included some that would usually be "geriatric."

(2) They have regarded orthopaedics, ophthalmology, and paediatrics as "regional specialties," whereas most districts would expect to provide these services. It is also not clear when the authors refer to "paediatric admissions" whether they mean

child patients of all specialties or just those of medical paediatrics.

(3) In their study of patients not currently in hospital it is not clear whether people in private nursing homes or homes for the elderly were included. District nurses may not be in regular contact with residents of these private institutions.

(4) There is no mention of the number of patients on waiting lists for hospital admission, and this group of patients does represent an unmet demand.

(5) A figure of 1193 patients meriting care in hospital was divided by the catchment population of 300 000 to give a figure of 4 per 1000 population as "a realistic provision." This assumes 100% occupancy, which is unobtainable for acute beds and in any case would not allow for the inevitable peaks and troughs of demand. If an 85% occupancy is assumed 4.7 beds per 1000 population are needed.

(6) There is no mention of how the catchment population of "about 300 000 people" was derived. This is a difficult and imprecise calculation but the figure is fairly critical in deriving rates of provision. For example, if the catchment population was 280 000 bed needs would be 5 per 1000 population at 85% occupancy.

There is a great danger that a summary of this sort of report will become engraved on the tablets as a revealed truth. At this stage the last sentence of the main report is a better representation of the situation when it states that "these estimates . . . must be interpreted with caution. . . ."

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Adverse reactions to intravenous anaesthetic induction agents

SIR,—We would like to comment on the excellent article on this subject by Drs J M Evans and J A M Keogh (17 September, p 735). We also have been collecting data from patients throughout the UK who have shown "adverse" response to a variety of intravenous agents. However, our analysis has included laboratory investigations of the patients' serum in order to determine the mechanism of adverse response. There are three important reasons for so doing: (1) the drug thought to be the culprit of the reaction may not be directly involved; (2) the patient may require further procedures in the future for which the drug(s) in question would otherwise be the first choice; and (3) there may be medicolegal implications if the question arises as to an untoward drug reaction or professional incompetence.

It is unfortunate that the word "adverse" has become almost synonymous with "immune-mediated" in the context of these intravenous drug reactions. Drs Evans and Keogh have certainly not fallen into this trap, but undoubtedly many readers will

make this correlation. The words anaphylactoid and histaminoid (this the authors do use) are similarly misleading when applied to clinical phenomena *alone*. "Anaphylactoid" is often used to describe a particular clinical picture resembling immune hypersensitivity which may not be basophil- or mast-cell-mediated. Equally, "histaminoid reactions" evaluated solely on cutaneous effects are not necessarily due to plasma histamine release (Lorenz, personal communication). Many reactions are possibly pharmacological in nature, perhaps the direct effect of a mixture of drugs on histamine-containing cells or perhaps the result of the speed of administration of the intravenous dose. Other reactions may represent complex reactions set up as a result of the psychological or the physiological trauma, or both, of the operative procedure on a particular patient. Further, our experiments suggest that temporary or permanent disturbances of the patient's immune system may increase the possibility of adverse response.

Our laboratory analysis of the plasma from 58 patients showing adverse reactions to various drugs (see table) covers the same three-year period as that reported by Drs Evans and Keogh. The samples were collected and analysed according to the protocol^{1,2} described in the *British Journal of Anaesthesia*. The distribution of clinical symptoms and operation procedures was similar to that observed by Drs Evans and Keogh.

Sequence studies of plasma are necessary for the full evaluation of the clinical response; some samples were incorrectly taken and some sequences were incomplete and could not be analysed completely. Nevertheless, it is obvious that complement is involved in at least half of the reactions reported (either as a primary or secondary effect) and that Althesin is the most frequently reported anaesthetic agent associated with the adverse response.

Three types of mechanism were recognised, type I reactions involving IgE and the direct liberation of histamine, immune reactions releasing histamine via the classical (C4) complement pathway, and finally the alternate pathway involving direct activation of C3. Differences in complement pathway activation in Althesin reactions may account for the variability in response time and clinical severity observed with these patients. Reactions showing neither IgE nor complement involvement were considered to be pharmacological. Although IgE was involved in some Althesin reactions, a higher incidence of involvement is apparent in the barbiturate responses (multiple exposure patients). Patients responding on first exposure to Althesin appear to do so by alternate pathway activation of complement C3. This is, of course, not an immune-mediated event and there should be no correlation between such complement-mediated reactions and "clinical atopy."

Analysis of adverse reactions to intravenous induction agents

	Cases analysed	C3 C4 anomaly	Sequence studies possible	Complement activation		IgE involved
				Classical	Alternate	
1974						
Althesin	19	9	5	1	4	1/1; 1/4
Thiopentone ..	1	1	0	0	0	0
Methohexitone ..	1	0	1	0	1	1
1975						
Althesin	16	9	7	3	4	1/3; 1/4
Epontol	1	1	0	0	0	0
Thiopentone ..	1	1	1	0	0	0
1976						
Althesin	15	7	12	4	6	0/4; 1/6
Epontol	2	0	2	0	2	0
Thiopentone ..	2	2	2	1	1	1/1; 1/1

We conclude that the factors likely to predispose an individual towards an adverse response are (a) genetic, (b) underlying immunopathological conditions, (c) frequency of exposure to a particular drug, and (d) various combinations of these. Genetic factors include both IgE (atopy) and complement anomalies. Immunopathological conditions involving circulatory immune complexes, such as chronic infection, systemic lupus erythematosus, and rheumatoid arthritis, may "prime" complement systems, making them susceptible to activation by intravenous drugs. Adverse response can thus be attributed to a range of mechanisms and predisposing factors and no intravenous drug can be considered entirely safe.

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¹ Watkins, J, *et al*, *British Journal of Anaesthesia*, 1976, **48**, 457.

² Watkins, J, Thornton, J A, and Clarke, R S J, *British Journal of Anaesthesia*, 1976, **48**, 1118.

Adverse reactions to Althesin

SIR,—Drs J M Evans and J A M Keogh report (17 September, p 735) a high incidence of adverse reactions after the administration of Althesin (alphadolone and alphaxalone in polyoxyethylated castor oil; Bayer) and suggest that the number is unacceptable. I am concerned that the authors should consider that such a retrospective study would be expected to reveal an accurate picture of the number of adverse reactions that occurred. An anaesthetist is unlikely to recall all the untoward events that occur at induction over 3-4 years and would tend to remember events associated with a new drug rather than the routine agent. I cannot remember a single serious adverse reaction associated with my administration of intravenous anaesthetic agents, including Althesin, over the past six years but do not accept that this is evidence that none occurred. Close observation at the time of induction will show that all intravenous induction agents are associated with adverse effects and these are likely to be higher when a new drug is introduced, especially if it is potent. Drs Evans and Keogh attempt to classify the type of reaction that occurred. I doubt that this is possible from the information available in a retrospective study. It is important to differentiate between anaphylactoid reactions and the pharmacological effects of overdose. This requires measurement.

Over recent years I have administered many Althesin anaesthetics, and in many cases cerebral function has been monitored. There is no doubt that patients react very differently to a standard weight-based dose, some developing signs of profound cortical depression. Adequate anaesthesia may be induced with only 1.5-2.5 ml of Althesin yet anaesthetists, used to injecting 12-15 ml of thiopentone, might not have adapted to the smaller volumes required when using Althesin.

Thiopentone was once described as an ideal method of euthanasia in war surgery.¹ Its popularity could well have declined but for a further publication which suggested that it was the technique of administration and the dose of drug used that was at fault rather than the agent itself.^{2,3} We may now be seeing a

similar situation with Althesin. Serious complications are likely to be reduced if induction agents are administered slowly in small doses, if blood pressure, heart rate, and even cerebral function are monitored during the induction period, and if an intravenous infusion is readily available. The incidence of anaphylactoid reactions will have to be worked out from the results of haematological and biochemical measurements. These may ultimately show that Althesin is particularly prone to induce adverse effects but I do not think one can accept the findings of a retrospective study. Meanwhile one can ponder on the mechanism of apnoea lasting 60 minutes after a single dose of Althesin. Someone could not possibly have given the wrong drug, could they?

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¹ Halford, F J, *Anesthesiology*, 1943, **4**, 67.

² Adams, R C, and Gray, H K, *Anesthesiology*, 1943, **4**, 70.

³ Editorial, *Anesthesiology*, 1943, **4**, 66.

Seniority payments and service in HM Forces

SIR,—Dr W A Jerrett in his letter (17 September, p 774) draws attention to a gross injustice in that service in the armed Forces does not count towards seniority in general practice, except as a "senior" medical officer. The Ministry of Defence should now warn all serving and potential medical officers that their five years of service, even if spent in general practice, will not count towards seniority payments. Perhaps they could include this information in the recruiting information they send to medical students who apply to join the armed Forces.

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General practitioner prescribing costs

SIR,—Dr R T A Scott's letters (30 July, p 319, 10 September, p 708) raised two very different questions. His suggestion in the later letter of a limited list of drugs for free or subsidised use is an attractive one and makes the prescribers' decisions much easier. Dr Scott's first question about the teaching of future general practitioners is even more pertinent now that, according to the DHSS's annual report of 1976, English general practitioners' prescribing costs averaged more than £22 000 each. Classes on prescribing costs were begun in the Wessex day-release course in 1974, when a colleague presented the results of a prescription pricing inquiry into his practice. This was followed by exercises in practical prescribing and questions on attitudes to prescribing. These clearly confirmed that "the question of cost plays no part in choosing drug treatments" was a view prevalent among trainees in 1974-5. Since then further classes have been held and the discussion of cost is now seen by trainees as a factor of importance in choosing treatment, especially where doctors try to agree on joint policies. Even so, we recognise that economy in GP prescribing costs can play only a limited part in keeping

down the cost of the health service. A 12% reduction in prescribing (requiring an average of 150 fewer prescriptions per doctor per month) would result in less than 1% change in the total cost of the NHS.

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Costs of prescribing

SIR,—Professor O L Wade recently delivered a lecture to the British Association for the Advancement of Science on "The problems (of the cost) of Drug Prescribing in the National Health Service." In it he evaluates most helpfully many of the problems in financing medication.¹ However, towards the end of the paper he suggests that monetary incentives could be used to curb wasteful prescribing in general practice. "Each doctor might be given a 'drug allowance' of £400 to £500 per 100 patients on his list to meet the cost of his prescribing. If he prescribes in excess of this, other than in exceptional circumstances, the excess would be met from his practice; if he prescribes within his budget the saving would accrue to his practice to be available for other services."

Unfortunately, one of the snags of the pool as a method of payment is that no one group of doctors can be given additional sums of money without this being subtracted at national level from the remuneration of all remaining practitioners. Thus, what sets out to be an inducement turns out to be a penalty, particularly on the conscientious doctor as well as to the body of general practitioners as a whole. I remember the days when I used to dispense for my own rural practice and chose the capitation system (now 52p per patient). I am well aware of the substantial savings available to be made from prescribing what one dispenses—but in urban practice, with independent chemists, the problem is more complex. The outcome of this system of pseudo inducements would lead to deterioration of health care.

At present there are only a few drugs, borderline substances, for which the GP may be asked to undertake the cost of prescribing himself. Legislation of this type would convert *all* drugs to this class in that the practitioner would be at risk to cover the cost of his patients' needs should they rise above an arbitrary limit. With inflation raising the price of drugs, increased reluctance of hospitals to prescribe what they recommend, and inflation massively eroding the doctors' income, that proportion of the cost of the doctors' drug bill which politicians would allow to fall as a debt on our pockets would be bound to increase to save the public purse.

Doctors with a large turnover of mobile patients, those looking after a preponderance of the elderly, those caring for homes for the elderly, subnormal, or mentally handicapped—all these doctors would find themselves penalised in so far as their drug expenses would be disproportionately high for having accepted responsibility for more difficult patients. What doctor would involve himself in screening his practice if the cost of treating identified cases might fall on his own pocket? What chance have ill patients of being accepted by a new doctor if he can see that they are persons liable to require expensive medication? What GP can afford to look after a child who, happening to have phenylketonuria, has come