by the North Hampshire Immediate Care Scheme and similar schemes around the United Kingdom being the fourth service, expected and integrated with the other three emergency services at serious incidents whenever they occur.

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Assessment of a general practitioner accident service

SIR,-Dr Aubrey Bristow (24 March, p 934), criticises my paper on the Mid-Anglia Practitioner Accident General Service (MAGPAS) (3 March, p 690) and refers to the system that has been adopted in Seattle and other North American cities. In this system emergency telephone calls requesting an ambulance are answered by specially trained operators who take the message, despatch an ambulance, and then proceed to explain to the caller first aid measures that might help the victim. This system is used predominantly to encourage the caller to initiate cardiopulmonary resuscitation in patients who have suffered a cardiac arrest before the arrival of the mobile coronary care unit, and it has been lifesaving on numerous occasions.

I suspect, however, that most motorists passing a serious road accident and then stopping at the nearest telephone to call for an ambulance would be reluctant to return to the carnage to render first aid -even if they could remember everything that the operator had told them. In addition, with the long stretches of motorways and the lonely country lanes in our rural area, it would be likely that a doctor from our accident service would be at the accident by the time that the good Samaritan motorist returned.

Dr Bristow did not take great care in reading the paper that he has been so quick to criticise for not only has he misquoted the total number of patients with airways obstruction treated by the doctors (114 and not 63), but he has also missed the essential point that the figure of 63 applies to the patients with airways obstruction whose airways were cleared by the doctors before the ambulance arrived. This figure alone shows the importance of immediate care in rural areas, for many of these patients would have died or suffered irreversible anoxic

brain damage but for the early arrival of a doctor. Dr Bristow goes on to say that "most fundamentally the paper fails to make any contribution to the central question of whether immediate care reduces mortality or morbidity." In fact, it is extraordinarily difficult to prove that immediate care does save lives since it is unethical to withhold emergency medical care to see whether or not the patient survives. The research and data collection committee of the British Association For Immediate Care is currently working, however, to find a method by which immediate care can be objectively assessed. In America a new injury severity score is being developed specifically to evaluate the effectiveness of prehospital emergency medical care.

Unlike Dr Bristow, I am unconvinced that the Dooley Care Index that he refers to in his letter can be used "to prove the benefits of immediate and I am not alone in taking this view.¹ care," Nevertheless, it might be of interest to Dr Bristow that when the index was used in 1980 to assess the effectiveness of MAGPAS, the score was 10.5(norm $2 \cdot 1$), which would indicate that the service was providing extremely good care. The 36% fall in road accident deaths in the area covered by the service by the fifth year of it becoming operational also suggests that lives are being saved.

In presenting the statistics I was not trying to prove that the service had saved lives for I do not think that this is possible at this time. It was simply a statement showing how one particular scheme functioned and what its members had done at the roadside. Perhaps, in the not too distant future we may be able to prove that immediate care does reduce both mortality and morbidity, but until then the volunteer doctors will continue to provide emergency medical care at the roadside.

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¹ Dooley A, Lucas B. The evaluation of emergency care. Ann R Coll Surg Engl 1978;60:451-6.

Pharmacy: an inquiry into its contribution to patient care

SIR,—Professor Paul Turner's question about where retail pharmacy is to go might be applied to pharmacy as a whole (17 March, p 810). Retail pharmacists are losing contact with family doctors and patients because of non-pharmaceutical interests, the wealth of ready manufactured drugs, the anonymity of group family practices, and the failure to maintain a programme of continuing education in pharmacy and clinical practice. This severance from the rest of the health care team could perhaps be remedied by introducing a regular NHS session in a hospital or group practice pharmacy for every retail pharmacist.

A clinical pharmacist appointed to each district general hospital could ensure continuity and understanding, by forming links between the patient and the health care team on drug matters.

Over the past six months in this unit the following contributions have been made by a clinical pharmacist: (1) A tenfold increase in the recognition and reporting of adverse drug reactions. (2) Twenty five interventions by the pharmacist each week during ward rounds, nearly three quarters of which were adopted as policies. They included policies on dose schedules, incompatibilities, the use of alternative drugs, patient inquiries, and counselling. (3) The clinical pharmacist's presence emphasised the educational value of ward rounds to final year pharmacy students.

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¹ Bonati M, Tognoni G. Has clinical pharmacology lost its way? Lancet 1984;i:556-8.

SIR.-I realise Professor Paul Turner cannot be fully aware of what all the hospitals in the NHS have already accomplished with clinical pharmacy, but more is being done than he realises (17 March, p 810).

In the South East Thames region for the past year or so there has been a working party to advise on the basic requirements for hospitals to establish clinical pharmacy. A training programme is in hand for all grades of pharmacists.

At Brighton General Hospital we moved from ward pharmacy to a pharmacist on the consultant rounds in 1977. Having developed such good will on both sides we were able to extend and offer our experience from five different consultant rounds to third year

undergraduates. The aim that the pharmacist see the patient as well as the prescription can lead only to a whole approach to patient care as hoped for by Professor Turner.

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Gynaecomastia in cystic fibrosis

SIR,-Dr S Braude and others (17 March, p 822) describe transient gynaecomastia in four of seven patients with cystic fibrosis who had hypertrophic osteoarthropathy. I report another observation of this association.

A 23 year old man who had cystic fibrosis for two years was admitted because of a four day history of fever, increasing dyspnoea, and production of sputum. Over the weeks before admission his knees and ankles had become swollen. He also complained of rightsided mastalgia. He had finger clubbing, swollen ankle joints, knee effusions on both sides and bilateral gynaecomastia. Pulmonary function tests showed severe airway obstruction. forced expiratory volume in one second: 950 ml (28% of predicted value and 40% of vital capacity). Sputum culture grew Staphylococcus aureus and Pseudomonas aeruginosa, and the patient was treated with intravenous ceftazidim and flucloxacillin. His sputum volume decreased, and his respiratory condition improved as did his arthropathy and mastalgia. Prolactin serum concentration was normal. The patient was not taking drugs known to cause gynaecomastia, and he had no evidence of liver disease or testicular neoplasia.

The mechanisms leading to clubbing and hypertrophic osteoarthropathy are unknown. The pulmonary clearance of certain vasodilator hormones might be impaired.1 Gynaecomastia may result from an imbalance between circulating estrogens and androgens,² and the affected lung might handle these steroids differently.

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¹ Lemen RJ, Gates AJ, Mathé AS, Waring WW, Hyman AL. Relationship among digital clubbing, disease severity, and serum prostaglandins F_aα and E concentrations in cystic fibrosis patients. Am Rev Respir Dis 1978;117:639-46.
² Carlson HE. Gynecomastia. N Engl J Med 1980;303: 795-9.

SIR,-The suggestion by Dr S Braude and others (17 March, p 822) that vasointestinal polypeptide may be responsible for hypertrophic pulmonary osteoarthropathy has no more basis than any of the plethora of chemical candidates suggested over the years.

Periostitis in hypertrophic pulmonary osteoarthropathy is associated with and preceded by an overgrowth of highly vascular connective tissue along the shafts of long bones.1-3 The increased peripheral blood flow that I showed in patients with this disease is restricted to the forearm and calf and relates to increased flow through this vascularised connective tissue and bone but there is not a generalised increase in peripheral blood flow, in particular in the skin, since flow in the hand