May 1971 over 100 cases have occurred among workers in shipyards on the Clyde and other industrial concerns in the west of Scotland. The peak of the outbreak was reached at the end of June and beginning of July, and a decreasing number of cases are still being notified. Those mainly involved are shipyard personnel working on the open decks of ships under construction, and only a few non-industrial workers have been affected. As in previous outbreaks in the Clyde Valley in 1956, 1967, and 1968, adenoviruses 2, 3 and 4 were isolated from the causal virus.1

While we agree that medical personnel are frequently the cause of the passage of the virus from patient to patient, spread also takes place outside the hospital or shipyard ambulance room, probably as a result of such procedures as amateur first aid for foreign bodies. In the present Clyde-side outbreak many of the patients presented with the condition at the ambulance room and had not been to a clinic with another condition. Furthermore, spread within families was a feature of the 1967 outbreak (Taylor, personal communication). However, an inquiry into the spread of the present outbreak had shown that only 6 of 103 patients questioned specifically on this point had another family member suffering from conjunctivitis. This curtailment of family spread may well be due to propaganda given at the ambulance rooms and clinics since the earlier outbreaks to ensure that the patients are punctilious about hand washing, use of personal towels, etc. Autoinoculation by contaminated fingers is probably the reason for some of the cases we have encountered among doctors, nurses, or ambulance-room attendants. The importance of hand washing is being stressed by Wegman and his colleagues in an outbreak in an American hospital, which ceased after the institution of thorough washing of hands and instruments with soap and water.1 We suggest that this measure should be added to the recommendations given by the Bristol workers.—We are, etc.,

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Pregnancy Testing

Sir,—Mr. A. D. Thurse (25 September, p. 769) and Dr. F. W. Winton (30 October, p. 296) draw attention to the "free" pregnancy testing facilities available through National Health Service laboratories. We also, like Dr. Winton, are disturbed over the increasing numbers of these tests. It must be realized, however, that the increasing demand for laboratory examinations is by no means confined to pregnancy tests. This laboratory covers the disciplines of bacteriology, haematology, clinical biochemistry, and toxicology. In 1960, a gross total of 67,018 specimens (not tests) of all kinds were received, of which 1,978 (2.9%) were pregnancy tests. In 1970 the gross total had risen to 134,433, of which 6,221 (4.6%) were for pregnancy testing. According to the 1961 Census, it is calculated that there will be more pregnancy tests performed in this laboratory during 1972 than there will be births in the whole of Scotland. Beaumont Ruchill Hospital, by around 1990, the tests done will equal the total for women (married and otherwise) between the ages of 15 and 44. (These figures have not been calculated by the statistician to the North-eastern Regional Hospital Board.)

To what do we ascribe the increasing popularity of this test? Is it a result of the pill? Is it related to the new legislation governing abortions? Is it an outcome of the greater freedom in premartial sex relationships? Is it just another result of the "free" facilities of the Welfare State?

Just how "free" is it? A total of 6,000 pregnancy tests were done in 1967, 1970, and 1971. Last year this laboratory £1,200 for reagents alone. To this must be added a further £500 because this laboratory operates a prepaid postal package service covering issue and return of specimen outfits. These figures take no account of other expenses, such as the cost of outfits, technicians' time, office time, stationery, etc. In addition to the qualitative tests done in 1970, quantitative tests totalled 111, and each of these cost a minimum of £140 for reagents alone. There is no abatement in demand for these tests. The figures for the first 10 months of 1971 indicate that the total for the year will pass the 7,000 mark.—We are, etc.,

J. BRODIE

I. A. PORTER

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Care of Chronic Psychotics

Sir,—We find it necessary to challenge some of the claims made by Drs. M. Z. Hussain and A. C. Goodacre (13 September, p. 703) about psychiatric services in Saskatchewan.

It is true that Saskatchewan once had the highest ratio of patients in mental institutions in Canada, and also probably one of the worst mental hospitals in the world.1 Also, it is true that during the sixties the Saskatchewan Hospital, Weyburn, showed the sharpest decline in population of any hospital in the western world. However, despite the repeated and numerous claims emanating from Saskatchewan we cannot agree that the effectiveness of so-called community care in that Province has been subverted.

The big discharge began in 1964, and by early 1966 there had been complaints concerning the rate of discharge and the standards and conditions in homes in which the patients were being placed. An ad hoc committee investigated these complaints, and in June 1966 suggested tighter laws and improved standards for placement homes. However, complaints continued about the early discharge of patients who were still symptomatic and also about the standards in approved homes. When a former patient murdered nine people, the concern became so widespread that another inquiry was instituted under Professor Shervert H. Frazier at the end of 1967. The following extracts from the Frazier report2 are of interest.

"Another common theme was that the Weyburn Hospital (Main Building) had adopted a policy of discharging patients no matter what the situation, the patient's condition, etc., several mentioned that a 'statistical approach' had replaced an individual psychiatric evaluation."

"Recommendation 11: We suggest that the practice of early discharge be brought in line with the principles that, so discharge is determined by bed counts, 'statistics,' or attempts to satisfy institutional goals, but by the needs of the patient, his family, and his community."

"Recommendation 23: Outpatient care and especially home-placement should be enriched more therapeutically by allowing as many of the following programs as feasible: sheltered workshops, half-way houses, day treatment centers, vocational rehabilitation programs, additional recreation, social clubs, exercise classes, nutritional guidance, and classes in personal hygiene."

We can see very little change since 1967, although the hard core of presumably non-dischargeable patients has resulted in a leveling off in the residual mental hospital population. This finding is incomprehensible that a hospital which has discharged over 500 patients from 2,600 to less than 400 beds should still retain a ward with over 100 patients. We are by no means in favour of the continued existence of any large mental hospital, but we are concerned at their being phased out without any adequate planning for alternative methods of care.

Drs. Hussain and Khan mention that the readmission rate has dropped dramatically for the Prince Albert unit. As 1970 was the first full year of operation we question the validity of this statement. Reference to the annual report of the Prince Albert unit, we note, shows that a readmission rate of less than 40% in the first year gradually rises to over 70%, together with an average length of stay of 27.5 days by 1969, both of these figures being the highest for any facility in the Province. It seems likely that Prince Albert will reveal a similar trend over the next few years.

Finally, we now view with increasing suspicion all publications which show an obsessive preoccupation with statistics such as beds per 1,000 population, while revealing nothing about what is really happening to the patients contained in these statistics.—We are, etc.,

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N. P. V. NAIRO

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Neurofibromatosis and Childhood Leukaemia

Sir,—Drs. M. W. McEvoY and Jillian R. Mann (11 September, p. 641) describe the association of neurofibromatosis with acute myeloblastic leukaemia in a 5-year-old boy.
In a recent study of 1,263 children with acute leukemia at the Children's Cancer Research Foundation, Boston we observed two patients with undifferentiated leukemia and multiple neurofibromatosis.1 This frequency is not significantly higher than the estimated incidence of neurofibromatosis in the general population (one case per 2,500-3,300 births).2 The association of neurofibromatosis with leukemia has occurred in two other children in the same clinic (not in our series) and has been described in three case reports from France.3,4 In one instance the combination of diseases affected siblings.5 The eight reported cases of neurofibromatosis and childhood leukemia are of great interest, but the evidence is not sufficient yet to indicate that the high cancer risk in neurofibromatosis extends to leukemia.—1 am, etc.,

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Leukaemia on Myeloma

Sir,—We are prompted by the letter of Dr. Judith A. W. Webb and others (23 October, p. 231) to report the case history of another patient who developed acute myeloblastic leukaemia after having been treated for four years with melphalan for myeloma. A 40-year-old man presented with pleuritic chest pain in 1967 having had backache for two years. X-rays showed that there were widespread osteolytic lesions in the ribs, vertebrae, and skull. The blood count and serum protein pattern were normal and there was no Bence-Jones protein in the urine. A bone marrow aspirate contained more plasma cells than lymphocytes and was not diagnosed as myeloma. Biopsy of one of the rib lesions demonstrated a plasma cell tumour. Therapy with melphalan was started in April 1967, and he had a short course of radiotherapy to his spine. (In May 1967 he developed asymptomatic hypercalcaemia which was treated with prednisolone. The prednisolone was stopped in March 1968.) He continued to take melphalan until August 1971. During the intervening period he was relatively well and his only other medication was analgesics to control lumbar back pain. X-rays showed no extensions of his osteolytic lesions and he did not develop an abnormal plasma protein.

In August 1971 he developed a 'flu-like illness and a routine blood count demonstrated that the majority of the white cells were blasts. The total white cell count was 7,500/mm³.

He was admitted to hospital one week later, when the only abnormal physical sign was purpura over both scapulae. The platelet count was then 66,000/mm³ and the white cell count had risen to 33,000/mm³. A bone marrow aspirate contained numerous hypercellular fragments and over 90% of the nucleated cells were typical and atypical blast cells which were periodic-acid Schiff negative.

There were no plasma cells. A diagnosis of acute myeloblastic leukaemia was made. The melphalan was stopped and treatment with daily intravenous cytosine arabinoside and oral thioguanine was substituted. There was an initial fall in the blast count but after two weeks of therapy he died of bronchopneumonia.

This case history is similar to a number of others recently reported,1,2 where patients with myeloma have responded well to treatment usually including melphalan and finally have developed acute myeloblastic or monoblastic leukaemia. Whether this is merely the natural history of the disease or related to the treatment remains to be decided.

We should like to thank Dr. P. H. Sanderson and Dr. M. Hulbert for allowing us to report this case.

—We are, etc.,

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Undesired Effects of Clioquinol Therapy

SIR,—I would like to draw your attention to the possible misquotation of a reference by Dr. O'Sullivan and myself on two occasions in the article by Dr. S. I. Terry (25 September, p. 745). Our article was a review of patients who had suffered from thalidomide neuropathy and there was no mention of clioquinol. Dr. Terry quotes this article as describing the clinical picture of clioquinol intoxication. This is very misleading. I would be grateful if you would draw the author's and your readers' attention to the error.—I am, etc.,

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** We showed Dr. Le Quesne's letter to Dr. S. I. Terry, who writes: "I am grateful to Dr. Le Quesne for the opportunity to clarify two statements in my original article. My aim had originally been to draw attention to the similarity between the marked dysaesthesiae but lack of muscle weakness reported by Dr. O'Sullivan and herself in thalidomide toxicity (with its associated abnormal neuropathy) with the symptoms and signs described in the case of clioquinol toxicity that I reported. Also, Sobue and others described the marked leukocytosis and elevation of the erythrocyte sedimentation rate in the acute phase as a feature of their series of clioquinol toxicity. Unfortunately, in condensing information for the original article the credit for these separate observations was transposed. I apologize for the inconvenience this may have caused."—Ed., B.M.J.

1 Sobue, L. et al., Neurology (Minneapolis), 1971, 31, 168.

Aluminium and Chronic Renal Failure

Sir,—Dr. V. Parsons and others (30 October, p. 273) have shown that bone biopsy specimens of patients with chronic renal failure occasionally have a high aluminium content. This is discussed in the context of the hypothetical toxicity of aluminium hydroxide and aluminium-containing resins in chronic failure.1 It is perhaps relevant that the patients with the highest aluminium/calcium ratios did not receive thalidomide medication nor did they manifest osteomalacia.

Unfortunately this work throws no light on the crucial clinical problem—the toxicity of aluminium in renal failure. Aluminium-containing compounds are administered in order to lower the serum phosphate level by the formation of insoluble aluminium-phosphate complexes. Since inorganic and organic compounds are of fundamental importance in every viable body tissue, phosphate depletion is likely to produce adverse sequelae. This has been shown in both humans2 and experimental animals3 subjected to anorexia or prolonged aluminium ingestion. Such results cannot logically be extrapolated to the situation where aluminium is trapped as an innocent bystander or where aluminium hydroxide is administered to reduce hyperphosphataemia.

We are currently examining this problem by administering food containing aluminium hydroxide to weaning rats. Normal rats show very little bone aluminium even at the end of a month of such treatment. Nevertheless, growth is impaired and rickety changes produced in the bones. Animals on a similar diet, to which disodium hydrogen phosphate is added to grow and maintain normal bone tissue, have shown that their bone aluminium content is three times normal. Animals with chronic renal failure also show elevated bone aluminium levels. It appears probable that aluminium deposition in bone is a result of some abnormality. It is interesting to observe that patients from different centres have different bone concentrations of aluminium may well be related to either differing intakes of phosphate or different serum phosphate levels.—We are, etc.,

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Trimethoprim/Sulphamethoxazole Mixture in Pregnancy

Sir,—The trimethoprim/sulphamethoxazole mixture sold under the names of Septrin and Bactrim seems to be fulfilling its early promise as an antibacterial agent, and is being widely used. Since the combination is very little absorbed by the kidney, it will eliminate many strains of proteins as well as Escherichia coli, it naturally curies a higher proportion of patients with urinary infections than would a sulphamamide used alone. On the face of it, trimethoprim/