In this Group wards are already closed for lack of nurses, which inevitably causes considerable hardship to the local population. Considerable local efforts are being made to recruit more nurses and to improve the training of nurses in this Group. Now our efforts are to be wasted and we are not even to be allowed to replace a vacancy when a nurse leaves. Furthermore we are not allowed to advertise for nurses to train for the future. To illustrate the problem as it already affects us in this area, may I quote the two following examples?

At routine out-patient clinics I frequently see patients who need an operation, but at the same time I realize that due to the shortage of nurses there is no prospect of these patients being admitted for the treatment they require. In all honesty I am compelled to advise these unfortunate people and their doctors to seek admission in some other area where the nursing shortage is less acute. A few of these patients can be admitted to hospitals elsewhere, but a large proportion of them under existing circumstances cannot hope for admission to hospital for the operation which they require. There are just not sufficient beds to accommodate them or nurses to look after them.

Patients who require a manipulation or similar minor operation with a general anaesthetic as day patients are taken into a ward where other patients already in the ward are got out of bed to make room for them. The linen is changed and the day patients are accommodated for a few hours until they are ready for discharge in the late afternoon. The original patients are then allowed to get back into their own beds.

If public funds cannot be provided to pay the salaries of the nurses we so urgently need, let us be honest and tell the public of our predicament. Let them be told that patients are turned away for lack of staff and lack of accommodation. The Health Service can no longer be truly comprehensive as the political parties would have us believe.—I am, etc.,

Eastbourne, Sussex.

S. A. JENKINS.

****** We understand that some other regional hospital boards have also asked hospital management committees for the time being to stop recruiting nurses, including student nurses, because the committees have overspent their revenue allocations for the current year. Overspending by management committees is said to have been in part due to increased fuel costs owing to the cold summer, to the high price of vegetables, and to the engagement of staff for whose salaries insufficient provision had been made.—ED., *B.M.J.*

Albright's Syndrome

SIR,—Dr. P. Hall's report of a patient with Albright's syndrome (polyostotic fibrous dysplasia) recorded a normal chromosome complement in the peripheral blood (November 3, p. 1159). We had previously carried out chromosome studies in a typical patient which confirm the findings in the peripheral blood and which also demonstrated that the affected areas of skin have a normal karyotype.

The patient, aged 8, was referred to Professor C. E. Dent by Dr. Fourman, of Cardiff. The diagnosis was first suggested in 1955 when he was x-rayed on account of a limp. He has sustained several fractures of his right femur, tibia, and forearm. The diagnosis was confirmed by biopsy in 1959. Examination shows a large pigmented naevus over the right buttock and thigh. X-rays, taken in Cardiff, showed cystic changes in the right femur, tibia, and ulna and also in the left tibia.

Because of the characteristic involvement of one side of the body and the distribution suggesting an origin in a primitive somite, a somatic mutation was considered as a possible mechanism by which the features of the disease were produced. To explore this possibility, the patient's chromosomes were studied in cultures of peripheral blood and skin obtained by biopsy of the naevus. Thirty cells in the blood culture and 32 in the skin culture were counted. All contained 46 chromosomes and the karyotype was that of a normal male.

These findings suggest that mosaicism involving the whole or a visible part of a chromosome is unlikely to be the cause of the disease. An abnormal gene is most likely to be responsible. This is in keeping with the findings in other congenital abnormalities involving the skin, including epiloia and neurofibromatosis.—We are, etc.,

A. W. JOHNSTON.

KUSUM P. LELE.

RUTH MARSHALL.

London W.C.1.

Persistence of Haemoglobin F

SIR,—The effects of inheritance of the gene for high levels of foetal haemoglobin (Hb F) in adult life are now well understood. Heterozygous subjects are haematologically normal, but Hb F can be found in the blood after infancy to the extent of 12–28% of the total haemoglobin, the actual figure tending to be constant among the affected members of one family. Fixed blood films treated by the method of Kleihauer *et al.*¹ reveal that every erythrocyte receives approximately the same complement of Hb F, just as it receives its share of Hb S in the sickle-cell trait. The situation can be explained by the absence or failure of one normal gene for the formation of the β -polypeptide chain of Hb A, and its replacement by a gene for the formation of the γ -chains of Hb F (the "high-F gene").

On other occasions when Hb F appears in measurable amount in the blood after infancy, as in thalassaemia, sickle-cell anaemia, leukaemia, and pernicious anaemia, its amount is variable and its distribution throughout the red-cell population is irregular; and its presence is generally regarded as compensatory for a failure to synthesize adequate amounts of Hb A.

In the course of examining children for variations in the normal disappearance of Hb F, an apparently new type of inherited persistence of foetal haemoglobin was discovered. An English child of 9 years had an attack of acute thrombocytopenic purpura which followed a normal course; his paternal uncle had been similarly affected. In the child's blood film after acid lysis, however, were some 4% of recognizably foetal cells. These were of varying content of Hb F, and in addition to them were others with trace amounts of Hb F. The picture was indistinguishable from that usually seen in a baby of about 5 months.² It was confirmed in the patient on many occasions, and persisted unchanged after his recovery. It has not been seen in other children with thrombocytopenic purpura. A haemolysate of the child's blood was found to contain 5.3% of Hb F.³

The mother's blood showed no foetal cells, and her Hb F level was 0.8%. The father's blood after acid lysis showed a degree of haemoglobin retention identical with that in his son's blood, and 4.5% of his haemoglobin was Hb F. The only sib, a brother aged 10, showed the same picture, and his Hb F level was 7.3%. The Hb F in the blood of the father and children was

separable by resin chromatography at pH 6.0. Paper electrophoresis at pH 8.9 in tris buffer showed, for all members of the family, no abnormal adult haemoglobin variant, and no increase of Hb A₂. None of the subjects showed any anaemia or any morphological changes in the red cells, or any alteration in red-cell fragility (tested against 0.35% NaCl). Thalassaemia was therefore reasonably excluded.

There can be little doubt that the present family demonstrates an inherited tendency to produce, in health, excessive amounts of Hb F. The amount produced is much less than in subjects with the "high-F gene," and its distribution in the erythrocytes is quite different. The only resemblance that this family bears to one possessing the "high-F gene" is that the amounts of Hb F detected in three members of two generations were approximately equal. Since the partition of Hb F among the red cells resembles that seen in some cases of thalassaemia minor, it might be thought that this hereditary syndrome has more claim to be regarded as "non-microcythaemic thalassaemia" than has the "high-F" anomaly. At present, however, there are no grounds for regarding it as a single gene effect. It may reasonably be regarded as an inherited failure to complete the normal change-over from foetal to adult erythropoiesis, with arrest at about the stage reached by a child of some 3 to 5 months of age. The genetic defect required to produce this must be located in the regulator gene or genes concerned with the maturation of haemoglobin synthesis in early infancy.—I am, etc.,

Department of Pathology,
The Royal Infirmary,
Bristol 2.

ALAN B. RAPER.

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- ² Fraser, I. D., and Raper, A. B., *Arch. Dis. Childh.*, 1962, **37**, 289.
- ³ Betke, K., Marti, H. R., and Schlicht, I., *Nature (Lond.)*, 1959, **184** (Suppl. 24), 1877.

Flame Burns

SIR,—Ten years ago review of the cases of thermal injury admitted to the Derbyshire Children's Hospital over twelve months indicated that nearly half were due to flame burns and were correspondingly severe. I wrote to the press warning of the dangers of unguarded flames and inflammable clothes, and included a photograph of a recent nightdress burn. Although this picture was not published the parents of the child who had permitted the use as a warning to others received hostile criticism from neighbours who knew of the case.

Recently I reviewed the burns again. I hope the following statement will be of interest to the profession and a guide to the public, while being sufficiently vague and delayed to avoid uninformed criticism of involved parents by their neighbours.

During a six-month period this year 19 children, 10 under 3 years of age, were admitted to the Derbyshire Children's Hospital for burns and scalds. Of these, four were flame burns, three of whom required transfusion. Two children fell on or through the fire-guard.

All injuries occurred in the home, and of the 15 scalds, six occurred during washing day when children fell into or spilt the hot-water buckets over themselves. Other five resulted from the child pulling a hot teapot or other food pan over itself. Seven cases required one or more grafting procedures.

More care on washing day, particularly of the toddlers, seems very necessary; particularly around Christmas flame burns must be thought of, and non-inflammable garments worn by children at parties where there are candles, and in nightclothes by the fire.—I am, etc.,

Derby.

R. LUNT.

Medical Centres in Hospital

SIR,—There have been several references in the medical press to the recent opening of the impressive medical centre at Kingston Hospital. We should certainly welcome all ventures of this kind, but it is unlikely that any management committee will be able to match the £42,000 so generously donated to Kingston by King Edward's Hospital Fund. In view of present financial stringency in the hospital service it does seem important to report an earlier centre, opened in March, 1962, at St. Albans City Hospital.

The St. Albans centre, less ambitious than that at Kingston, was built and furnished from Exchequer funds for just over £5,000. It includes conference room, library, reception area, light refreshment bar, and cloak-rooms. Projection equipment was kindly donated by Messrs. Smith Kline and French, and we are greatly indebted to the Nuffield Provincial Hospitals Trust for providing the salary for a secretary/librarian during the next three years. The centre has now been in use for nine months and forms a valuable meeting-place for hospital staff and general practitioners.

It is highly desirable that similar centres should be developed in other hospitals. We hope that the St. Albans centre, which has been accommodated in a relatively inexpensive prefabricated building, may encourage the medical staff in other areas to press for the development of comparable facilities.—I am, etc.,

London W.1.

REGINALD S. MURLEY.

POINTS FROM LETTERS

Appendicitis

Dr. T. D. S. HOLLIDAY (Chester Royal Infirmary) writes: The bacterially inflamed appendix is probably the consequence of vascular changes in an organ made turgid by various causes, among which virus infections of the lymphoid tissue are probably frequent. Some of the histologically normal appendices may well have been removed in the pre-suppurative stage, with benefit to the patient, who has thereby been spared the increased dangers attendant upon operation in the suppurative stage.

Pleuropneumonia-like Organisms

Dr. C. M. HEANLEY (Salisbury, Rhodesia) writes: Your review of a monograph on pleuropneumonia-like organisms (November 3, p. 1169) reminds me that during the 24 years preceding 1930 I found many times that if a calf developed this disease within the four days following vaccination then no lymph could be got from it. It makes me wonder whether the pleuropneumonia organism contains an anti-biotic which can control the pustulation of smallpox. The possibility of associated rinderpest could never be excluded.

Polythene Gloves

Dr. E. S. ARMSTRONG ASHE (Meols, Cheshire) writes: I am finding that cases of arthritis of the small joints of the hands are improved by a local application such as ung. methyl. sal. and a polythene glove with an elastic wrist band. The glove is worn continuously night and day, and it is found that there is greater absorption of any medicament applied in this way than formerly. I would be interested to hear of other doctors who have used this method of treatment.