appears to affect the very young and the very old, and most of the patients here fall into these two categories.

The results of this small series may perhaps be taken as an indication that correctly managed medical therapy has something to contribute in the acute case of colitis. The measures taken will be bound to improve the general condition of the patient. Even if surgery is later decided upon, the risks of operation will be reduced by the adequate correction of infection and deficiencies. Adrenocortical suppression should not have occurred, but in any case operative and post-operative steroids should be given to guard against this. It is suggested that the combined steroid regime be tried in all fulminating cases, but not persisted in if there is no favourable response in a few days, or abandoned should the patient's condition seriously deteriorate on treatment. Patients with surgical complications such as perforation or haemorrhage will of course require special surgical consideration. The results with combined intramuscular A.C.T.H. and intrarectal prednisolone are possibly superior to other reported regimes of steroid therapy.

Summary

The results of treating 12 cases of fulminating ulcerative colitis are reported. Combined intramuscular A.C.T.H. and prednisolone enemata assisted recovery in nine cases, with appropriate additional measures for correction of anaemia, shock, electrolyte imbalance, sepsis, etc. A possible synergic mechanism is suggested for the effectiveness of the steroid therapy.

I thank the physicians and surgeons of the Royal Hospital for their co-operation and help, and permission to publish cases; the pathology department for much help; and Glaxo Laboratories Ltd. for generous supplies of "predsol" enemata.

REFERENCES

Brooke, B. N. (1956). *Lancet*, **2**, 532.
—— (1959) Ibid., **2**, 745.
—— and Slaney, G. (1958). Ibid., **1**, 1206.
Lennard-Jones, J. E., and Vivian, A. B. (1960). *Brit. med. J.*, **2**, 96.
Matts, S. G. F. (1960a). *Lancet*, **1**, 517.
—— (1960b). *Proc. roy. Soc. Med.*, **53**, 650.
—— (1961a). *Brit. med. J.*, **1**, 165.
—— (1961b). *Quart. J. Med.*, **30**, 393.
—— and Gaskell, K. H. (1961). *Brit. med. J.*, **2**, 614.
Nugent, C A., Eik-Nes, K., and Tyler, F. H. (1959). *J. clin. Endocr.*, **19**, 526.
Truelove, S C. (1956). *Brit. med. J.*, **2**, 1267.
—— (1957). Ibid., **1**, 1437.
—— (1958). Ibid., **2**, 1072.
—— (1960a). Ibid., **1**, 464.
—— (1960b). Ibid., **2**, 102.
—— and Witts, L. J. (1955). Ibid., **2**, 1041.
—— —— (1959). Ibid., **1**, 387.
Veldstra, H. (1956). *Pharmacol. Rev.*, **8**, 339.
Watkinson, G. (1958). *Brit. med. J.*, **2**, 1077.

A new British Standard specification (B.S. 3462: 1962) for spectacle frames made predominantly of gold-filled material or nickel silver has just been published. The standard deals with those essential requirements for metal frames which are independent of individual dimensional requirements, and incorporates a mechanical test for the frames most likely to be used by children—those made of nickel silver. The object of this test is to ensure that the bridge is of such strength that serious misalignment through rough handling is less likely to occur. Copies may be obtained from the British Standards Institution, Sales Branch, 2 Park Street, London W.1 (price 5s., postage extra to non-subscribers).

SEPSIS IN THE HOME*

BY

CLIFFORD R. KAY, M.D.

General Practitioner, Manchester

In spite of the vast number of published reports on staphylococcal infection many aspects of the epidemiology of the *Staphylococcus aureus* remain obscure. A large proportion of these publications are based on observations in hospitals, where the environment is both specialized and highly complex. The present investigation was designed to study the relationship of *Staph. aureus* to man in the far simpler environment of the home and the family.

In the remainder of this paper, when "staphylococcus" is used without qualification the species *Staph. aureus* is implied.

Material and Methods

The investigation was conducted in a small single-handed general medical practice situated in a pleasant residential area on the south side of Manchester, a large industrial city with a high rate of atmospheric pollution.

Study A

Throughout the year 1958 all septic lesions presenting in the practice were recorded. In addition, as many of the following observations were made as were practicable in each case: (1) a swab was taken from the lesion and sent for culture, (2) an anterior nasal swab was taken from the patient with the lesion, and (3) an anterior nasal swab was taken from other members of the family.

Definition of Sepsis.—An attempt was first made to describe shortly the type of sepsis it was thought desirable to study—but without success. Finally, it was decided to make a classified list of all the lesions that were to be recorded. Two criteria were applied in the selection of this list from all purulent processes: (1) the lesion should be easily accessible, and (2) it should be known to be commonly due to *Staph. aureus*. For both these reasons vaginal and urethral discharges were excluded. Throat infections were also omitted because of their complex aetiology and the great difficulty in defining them. The final list is given in Table I, and these were the lesions recorded.

TABLE I.—*List of Septic Conditions Recorded in Study A*

Infections of the skin
Follicular: Pustules. Furuncles. Carbuncles
Diffuse: Impetigo. Secondarily infected dermatoses
Traumatic: Secondarily infected skin wounds

Infections of subcutaneous tissues
Abscesses draining through the skin

Infections of special organs
Hands and feet: Palmar and plantar space infections. Pulp space infections of digits. Perionychia
Eyes: Conjunctivitis. Styes. Blepharitis. Dacryocystitis
Ears: Otorrhoea—acute and chronic. Otitis media. Otitis externa

Study B

This study is described in outline here as only brief reference will be made to the results. A full description of the investigation will be given in a separate communication.

Between March, 1957, and May, 1959, 37 families were investigated for periods ranging from 12 to 24

*Based on part of an M.D. thesis presented to the University of Liverpool, December, 1960, and part of the essay awarded the Sir Charles Hastings Clinical Prize, 1961.

months. Anterior nasal swabs were taken from all members of the households every six weeks—a purely arbitrary interval—while any intercurrent staphylococcal lesion was also recorded and swabbed.

Seventeen of the families were selected because one of their members had presented with a staphylococcal infection. These families are here called "septic families." The remaining 20 families were selected as controls, and matched the septic families fairly closely in respect of numbers, age structure, and social status.

Bacteriological Examinations

Almost all the swabbings were done personally by me. For the nasal examinations the swab was first freshly moistened in sterile normal saline solution, then firmly rotated round the periphery of each anterior naris before being replaced in a stoppered sterile tube. All the technical procedures were carried out by Dr. M. T. Parker and his staff in the Manchester Public Health Laboratory. The swabs were sent to the laboratory as quickly as possible, usually by hand on the day of collection. Some, however, which had to be collected in the evening or at week-ends were sent by post.

A report was made on any organism cultured from the swabs taken from lesions, and where appropriate the sensitivity of the organism to antibiotics. In the case of the nasal swabbings a positive report was given only when *Staph. aureus* was cultured.

The sensitivity of all staphylococci was determined to the following antibiotics—penicillin, streptomycin, chloramphenicol, chlortetracycline, and erythromycin. All staphylococci were typed by the bacteriophage method.

In this investigation the main purpose of phage-typing the staphylococci was to decide whether or not strains isolated from different members of one family, from different sites on one person, or from one person on different occasions, were the "same"—that is, members of one clone.

It is relatively rare to have complete identity of phage pattern in two cultures thought to be the same (Williams and Rippon, 1952). Thus an attempt had to be made to define how much the phage pattern of two cultures must vary before regarding them as different, and this analysis of the phage-typing was made by Dr. Parker.

Results

Table II summarizes the main findings of the year's study. Nearly 80% of the total of 118 lesions were swabbed and 80% of the swabs showed significant bacterial growth, the remainder giving no growth or only growth of a commensal. In 58 lesions (62.4%) the pathogen was *Staph. aureus*, including six lesions in which it was associated with another pathogenic organism. Sixteen (17.2%) of the lesions were due to a variety of other pathogens. Forty-five per cent. of all staphylococcal lesions were penicillin-resistant—an unexpected finding outside hospital which is discussed below. One lesion was due to a strain resistant to penicillin and streptomycin, and one to a strain resistant to penicillin and the tetracyclines.

TABLE II.—*A Year of Sepsis (Study A)*

	No.	%
Lesions recorded	118	100
Lesions swabbed	93	78·8
Swabbed lesions showing bacterial growth	74	79·6
Swabbed lesions growing *Staph. aureus*	58	62·4
Staph. aureus penicillin-resistant	26	44·8
Swabbed lesions due to other organisms	16	17·2
Staph. aureus plus other infection	6	6·5

During the year of study A the practice grew rapidly relative to the numbers present at the start. To avoid complicated adjustments only those patients who were in the practice throughout the year were included in the calculations of incidence.

Incidence of Sepsis (Table III).—Logan (1953) analysed morbidity encountered in eight general practices during 12 months. He gave no single figure for "sepsis," but by adding the results of septic lesions classified under different headings a close approximation to the range of conditions recorded here can be obtained (Table I). Eleven per cent. of the patients in the practices that Logan studied suffered from sepsis during the year. The general practitioners concerned were not specially interested in sepsis, so that it is not surprising that the incidence in the present study (14%) was rather higher. Logan's finding that 13% of the population reported with the common cold during the year gives some perspective to these figures. Gould and Cruikshank (1957) found that 5% of the patients in the latter's general practice had staphylococcal lesions during the year. This should be compared with the estimate of 9% recorded here.

Sex Incidence.—The results in this study show no significant difference in the incidence of sepsis between the sexes (males 15.6%, females 17.6%). Logan's figures show a higher incidence of sepsis in males. This is probably due to the effects of minor trauma on a population containing a higher proportion of manual workers than in my practice.

Age Incidence.—Table IV shows a higher incidence of sepsis in childhood and an unexpected second peak between 30 and 40 years of age. The latter finding may be due to the contact of the parents in this group with their young children. Other investigators agree that there is an increased incidence in childhood (Logan, 1953; Spence *et al.*, 1954) while the neonate is especially susceptible to staphylococcal infection, at any rate in hospital (Wysham *et al.*, 1957; Forfar and Maccabe, 1958; Simpson, Tozer, and Gillespie, 1960). Otherwise there is a fairly even spread of infection over adolescent and adult life.

TABLE III.—*Incidence of Sepsis (Study A)*

	No.	%
Total practice population	509	100
Individuals with sepsis	67	13·8
Septic lesions	85	16·7
Staphylococcal lesions (estimated)	53	10·4
Individuals with proved or presumed staphylococcal lesions	45	8·8
Individuals with recurrent staphylococcal lesions during year	9	20·0*

Note.—The practice population on which these calculations are based consists of those individuals who remained in the practice throughout the year of observation and excludes those who joined or left the practice during the year. The proportion of staphylococcal lesions in this series is assumed to be the same as in the swabbed lesions shown in Table II.

* This is an underestimate of the risk of recurrence, since the period of follow-up ceased at the end of the year.

TABLE IV.—*Incidence of Sepsis. Age Analysis (Study A) (See Note to Table III)*

Years	No. of Lesions	Total Population	%
0–	24	103	23·3
10–	8	54	14·8
20–	9	85	11·2
30–	26	100	26·0
40–	6	66	9·1
50–	7	50	14·0
60–	4	34	11·8
70–	1	16	6·3
80–	0	1	0·0

Incidence of Infections due to Penicillin-resistant Strains

The incidence in this study is recorded in Table II. There are not many previous records of the incidence of infections due to penicillin-resistant staphylococci (as distinct from nasal strains) in populations outside hospital. Forbes (1949) found 12% of staphylococci from infections in out-patients to be resistant. Summers (1952) also recorded an incidence of 12% in out-patients. Between 1950 and 1953 Gould and McKillop (1954) found an incidence of 14%. Roodyn (1954) found 23% in general practice.

Parker (1958) analysed material sent to a public health laboratory from cases outside hospital during the years 1953–6. He divided the staphylococci into those from "deep" lesions and those from "superficial" lesions. Deep lesions included boils, carbuncles, and paronychiae. Thus they were comparable with most of the lesions in this study. Parker found that only 19% of staphylococci from his deep lesions were penicillin-resistant. But in the present series, recorded in 1958, the incidence was as high as 45%. A study of staphylococcal infection in general practice in Australia (Johnson *et al.*, 1960), at the same period, revealed that 64% of lesions were due to penicillin-resistant strains. This suggests that the incidence recorded here is not atypical of the present position, and must cast doubt on the advisability of using penicillin for the treatment of staphylococcal infection in general practice unless the sensitivity of the organism has been determined.

Recurrence of Infection in Individuals

It is well known that a patient who has a staphylococcal infection is likely to suffer from a recurrence, but it is interesting to try to assess the risk statistically. Table III shows that 9 out of 45 infected patients (20%) had one or more further attacks during the year of observation. The risk of recurrence is obviously much higher if the length of follow-up is increased. Roodyn (1954) likewise found a recurrence rate of 20%, but this was during a period of two years' observation.

In this study, therefore, it was found that if a patient develops a staphylococcal infection the chances of a further attack were more than double the 8.8% incidence in the general population (Table III).

Recurrence of Infection in Families

Staphylococcal infection tends to spread to several members of a household. The chance of further lesions developing in the family is assessed in Table V from the material of Study B. Recurrence rates in the "septic families" are compared with the incidence of lesions in the "control families." The figures indicate that once staphylococcal infection has appeared in a family the chance of another lesion developing in one of its members is increased fourfold.

Spread of Infection within the Family.—Study B was primarily concerned with 17 septic families and 20 control families. During the period of observation at least one member of seven of the control families developed a staphylococcal lesion, giving a total of 17 plus 7, or 24 families with lesions. In no fewer than 13 of these 24 families two or more members of the same household were affected. Thus after any individual first developed a staphylococcal lesion, spread of the infection to other members of his family occurred in about half the cases. Material for culture was not available from all lesions, but where the phage patterns of the lesion strains were known it was usually found that a single strain was the cause of all the lesions in that family. This was the case in 22 out of the 24 families in which infection occurred. An example of how infection spreads within a family to cause a "family epidemic" is shown in the diagram, where the findings in Family 30 are charted. In this and one other family the "epidemic" strain of staphylococcus was almost certainly brought into the household from the maternity hospital by the newborn baby.

TABLE V.—*Recurrence of Sepsis in the Family (Study B)*

	Control Families	Septic Families
No. of families	20	17
,, ,, individuals	83	63
Average No. of individuals per family	4·1	3·7
Total No. of family—year's observation	24	21
No. of septic lesions subsequent to initial swabbings	10	36
No. of lesions per family-year	0·42	1·7
Ratio of lesions—control: septic	1·0	4·1

	FATHER (30)	MOTHER (27)	SON (7)	SON (6)	SON (born Sep. 4, 57)
1957 MAY	(1)	○	○	(4)	
JUL.	(1)	○	○	○	
AUG.	(1)	○			
OCT.	(1)	(1)	○	(4)	(2)
NOV.	(1)	(2)	(3)	(3)	(2)
DEC.	(1)	(2)	○	○	(2)
1958 JAN.			[2]		
FEB.	○	(2)	(2)	○	(2) [2]
MAR.	○	(2)	(2)	(2)	(2)
MAR.					[2]
MAY	○	(2)	○	○	(2) (3)

LESION [2] 52A/79 Conc. phage, penicillin resistant

NASAL SWAB
- ○ No staphylococcus grown
- (1) 71 Penicillin resistant
- (4) 3B/55/71 Penicillin sensitive
- (2) 52A/79 Conc. phage, penicillin resistant
- (3) 7/54 Penicillin sensitive

Chart of Control Family No. 30.

Conclusions

Staphylococcal infections are often so trivial that many patients do not need treatment from their doctor. Therefore even the high rates found in this study must be an underestimate. Staphylococcal infection is therefore a very common illness in the community, with a high rate of recurrence in the individual and a marked tendency to spread to affect other members of the family. It occurs at all ages although it is commoner in childhood. There are no marked differences between the sexes. Almost half the infections in this practice in 1958 were due to penicillin-resistant organisms.

Relationship of Sepsis to the Staphylococcal Carrier State

It is now generally agreed that the anterior naris frequently provides the reservoir of staphylococci which

are the cause of lesions in the carriers and in their contacts (Dolman, 1935; Williams, 1946; Roodyn, 1954; Tulloch, 1954; Gould and Cruikshank, 1957; R. E. O. Williams *et al.*, 1959). In recent years attention has been drawn to the existence of other potential carrier sites, but the relative importance of these and of the frequency of auto-infection and cross-infection has remained obscure.

The following observations, made during the present study, may help to clarify the situation.

Nasal Carriage in the Patient.—Table VI presents the relationship of the lesion to nasal carriage in the same individual. In 92 instances from studies A and B swabs had been taken both from the lesion and from the patient's nose. In two-thirds of these cases the nasal strain was identical with the lesion strain, while in the remaining third there was no growth from the nasal swab or the strain was different. In Table VII the distribution of the lesions on the body is compared between these two groups. The results reveal that there is a very strong association between lesions on the head and neck and nasal carriage. On the other hand, with lesions in the axilla and on the lower limb the association with nasal carriage is significantly weaker. This suggests that axillary and lower-limb sepsis may be associated with carrier sites other than the nose. Upper-limb lesions (mainly on the hands) and eye lesions are equally distributed between the two groups.

TABLE VI.—*Relationship of Staphylococcal Lesion to Nasal Carriage (from Studies A and B)*

	No.	%
Total number of paired swabs (lesion and patient's nasal swabs)	92	100
Number in which lesion and nasal strains identical	63	68·5
" " " nasal swab negative or different from lesion strain	29	31·5

TABLE VII.—*Distribution of Lesions in Individuals Carrying the Lesion Strain in the Nose (Group X) Compared with Individuals Not Carrying the Lesion Strain in the Nose (Group Y) (From Studies A and B)*

	Head and Neck	Eye	Axilla	Upper Limb	Trunk	Lower Limb	Total
Group X: Number	32	12	2	11	2	4	63
Group Y: Number	4	7	5	4	0	9	29
Group Y: Expected number (approx.)	12	6	2	5	0	4	29

Note.—The expected numbers are calculated on the assumption that there is no difference in the distribution of lesions between the two groups. The differences between the actual and the expected numbers suggest that there is a strong association between lesions on the head and nasal carriage, and that lesions on the axilla and lower limb are weakly associated with nasal carriage and are therefore probably associated with a different carrier site.

Nasal Carriage in the Patient's Family.—The state of nasal carriage in the patient's family was known in 58 instances, and another member of the family was found to be carrying the lesion strain in the nose in half the cases whether or not the patient with the lesion was a nasal carrier of the strain. In only 8 (13.8%) of the 58 lesions was no nasal carriage of the lesion strain found in either the patient or the family.

Discussion

These results show that, in the home, the patient carries the lesion-producing strain in the nose in only two-thirds of cases. But consideration of the patient alone gives an incomplete picture because another member of the family is a possible source of infection in half the cases. This agrees with the finding of Tulloch *et al.* (1960). Further, there is good evidence (Table VII) that other carrier sites beside the nose must be considered.

Carriage on the Skin

It has been suggested that the whole of the skin may "carry" staphylococci (Miles *et al.*, 1944; Williams, 1946; Moss *et al.*, 1948), but the mere presence of staphylococci on the skin may be due to contamination, and it does not necessarily indicate true colonization by the organism there, although Devenish and Miles (1939), and Roodyn (1960) believed that this does occur.

Staphylococci are ubiquitous, so that it is important to bear in mind the distinction between mere contamination and true colonization by the organism, and the term "carrier site" should be applied only to a situation where active multiplication of the organism is proceeding in the absence of infection.

Other Carrier Sites

Hare and Ridley (1958) found a laboratory worker who had a heavy perineal colonization by staphylococci, and Ridley (1959) showed that 22% of male adults were perineal carriers. J. R. B. Williams *et al.* (1959) also observed perineal carriage among nursing staff and found that carriage in the axilla was almost as frequent. Tulloch *et al.* (1960) were satisfied that the perineum was a frequent source of staphylococcal infection.

Valentine and Hall-Smith (1952) thought that the eye may occasionally provide a reservoir of staphylococci.

There is no doubt that in the newborn baby the umbilical stump is a most important source of infection (Jellard, 1957), and it is fortunate that this site does not persist.

It seems likely, therefore, that only three positions on the human body normally become carrier sites—the anterior nares, the axillae, and the perineum. It may be pertinent that these sites are so similar physiologically in that each provides warm, moist, hairy skin.

It must be emphasized, however, that the anterior naris of the patient or of another member of the family was a possible source of infection in 86% of all lesions encountered in this study, so that the nose must still be regarded as the most important of the carrier sites in family infections.

Relationship of Site of Lesion to Carrier Site

The figures shown in Table VII indicate that lesions in the axilla and on the lower limb are poorly correlated with a nasal carrier site. This supports the view that the axilla and the perineum may act as carrier sites independently of the nose and that the perineum may be the source of infection for lesions below the waist as suggested by G. I. Watson (personal communication) and by Tulloch *et al.* (1960).

Treatment of Carrier Sites

No attempt was made in this investigation to study the treatment or prevention of staphylococcal sepsis by the application of antibiotics to the carrier sites. The observations recorded here could help to make such treatment more rational in the future, though complete investigation of a family would be a formidable procedure.

Summary

This paper describes an investigation of sepsis in the family and its relationship to staphylococcal nasal carriage. About 9% of individuals per year suffered

from staphylococcal infections, 45% of which were frequent in children, but there was no significant difference in incidence between the sexes. The chance of recurrence of staphylococcal infection in an individual was double the incidence in the general population, while the risk of a recurrence in any members of the same family was four times the rate experienced by a family with no previous infection. Infection in one individual later spread to other members of the family in no fewer than half the cases. It was nearly always found that a single staphylococcal strain was the cause of all the lesions in a family.

Comparing the staphylococcal strains found in the lesion and in the anterior nares, these were found to be identical in the individual with the lesion in two-thirds of the instances, while in half the cases another member of the patient's family was carrying the lesion strain, whether or not the patient was a nasal carrier of this strain.

Evidence is presented for the belief that the axilla and the perineum must be regarded as carrier sites in addition to the nose, and that there may be an association between the position of the lesion and the site of carriage of the causative staphylococcus.

I am indebted to Dr. M. T. Parker, lately Director of the Public Health Laboratory in Manchester. Dr. Parker was responsible for all the bacteriological services required in this study, and he has given me encouragement, advice, and most valuable constructive criticism. I am grateful also to Dr. K. W. Cross, of the department of medical statistics, Queen Elizabeth Hospital, Birmingham, for his assistance with the statistical aspects of the study; to the staff of the department of medical illustration, Manchester Royal Infirmary, for the presentation of the tables and graphic material; and to the Research Committee of Council of the College of General Practitioners for advice and encouragement in planning the investigation.

REFERENCES

Devenish, E. A., and Miles, A. A. (1939). *Lancet*, **1**, 1088.
Dolman, C. E. (1935). Ibid., **1**, 306.
Forbes, G. B. (1949). *Brit. med. J.*, **2**, 569.
Forfar, J. O., and Maccabe, A. F. (1958). Ibid., **1**, 76.
Gould, J. C., and Cruikshank, J. D. (1957). *Lancet*, **2**, 1157.
—— and McKillop, Elizabeth J. (1954). *J. Hyg. (Lond.)*, **52**, 486.
Hare, R., and Ridley, M. (1958). *Brit. med. J.*, **1**, 69.
Jellard, Janet (1957). Ibid., **1**, 925.
Johnson, A., Rountree, Phyllis M., Smith, Katherine, Stanley, N. F., and Anderson, K. (1960). National Health and Medical Research Council Special Report, Series No. 10. Canberra.
Logan, W. P. D. (1953). *Studies on Medical and Population Subjects*. No. 7. H.M.S.O., London.
Miles, A. A., Williams, R. E. O., and Clayton-Cooper, Barbara (1944). *J. Path. Bact.*, **56**, 513.
Moss, Brenda, Squire, J. R., and Topley, Elizabeth (1948). *Lancet*, **1**, 320.
Parker, M. T. (1958). *J. Hyg. (Lond.)*, **56**, 238.
Ridley, M (1959). *Brit. med. J.*, **1**, 270.
Roodyn, L. (1954) Ibid., **2**, 1322.
—— (1960). *J. Hyg. (Lond.)*, **58**, 1, 11.
Simpson, K., Tozer, R. C., and Gillespie, W. A. (1960). *Brit. med. J.*, **1**, 315.
Spence, J. C., Walton, W. S., Miller, F. J. W., and Court, S. D M. (1954). *A Thousand Families in Newcastle upon Tyne*. Ch. XII, p. 86. Oxford Univ. Press, London.
Summers, G. A. C. (1952). *Lancet*, **1**, 135.
Tulloch, L. G. (1954). *Brit. med. J.*, **2**, 912.
—— Alder, V. G., and Gillespie, W. A. (1960). Ibid., **2**, 354.
Valentine, F. C. O., and Hall-Smith, S. P. (1952). *Lancet*, **2**, 351.
Williams, J. R. B., Talbot, E. C. S., and Maughan, E. (1959). *Brit. med. J.*, **1**, 1374.
Williams, R. E. O. (1946). *J. Path. Bact.*, **58**, 259.
—— Jevons, M. P., Shooter, R. A., Hunter, C. J. W., Girling, J. A., Griffiths, J. D., and Taylor, G. W. (1959). *Brit. med. J.*, **2**, 658.
—— and Rippon, Joan E. (1952). *J. Hyg. (Lond.)*, **50**, 320.
Wysham, D. N., Mulhern, Marie E., Navarre, G. C., LaVeck, G. D., Kennan, A. L., and Giedt, W. R. (1957). *New Engl. J. Med.*, **257**, 295.

FRACTURES OF BASE OF FIFTH METATARSAL

BY

J. B. PEARSON, M.B., F.R.C.S., D.Obst.R.C.O.G.
Resident Surgical Officer, Children's Hospital, Birmingham

It was decided to compare the effectiveness of different forms of treatment of a relatively minor fracture, that of the styloid process of the base of the fifth metatarsal. This fracture is usually sustained during acute inversion of the foot, when peroneus brevis is stated to be pulled off the base of the fifth metatarsal, together with the styloid process to which it is attached (Jones, 1921). Attempts artificially to produce the fracture on cadavers were unsuccessful. While Watson-Jones (1955) states that "strapping is usually sufficient treatment though a walking-plaster may have to be worn for six weeks," no record can be found of a critical assessment of these two methods, or of the use of local infiltration of the fracture site with procaine or hydrocortisone.

Diagnosis is based upon the finding of tenderness at the lateral aspect of the base of the fifth metatarsal, both on local pressure and on inversion of the foot. *X*-ray examination confirms the presence of a fractured styloid process of the base of the fifth metatarsal, though congenital anomalies of ossification in this region have to be borne in mind (Holland, 1921). In this series the clinical signs were always supported by confirmatory *x*-ray films, and there was no doubt that the appearances were those of fracture and not of an os vesalianum or unfused epiphysis. In children there is often a juxta-epiphysial fracture of the tubercle rather than a true diaphysial fracture.

Methods

Between September, 1958, and September, 1960, 146 fractures of the styloid process of the fifth metatarsal were seen in patients attending the casualty department of the General Hospital, Birmingham.

Sixty-five patients were treated in plaster-of-Paris. The usual regime was that the plaster was applied as a "back slab and U" from the level of the tibial tubercle to the metatarsal heads, and after a week this was enclosed and a walking-block applied so that the weight-bearing could be resumed. The plaster was removed after a variable time and usually a supportive bandage ("cellanband") was applied for a week or more after the plaster had been removed.

Twenty-six patients were treated by infiltration of the fracture site with 2% procaine, and 27 were treated by infiltration of the fracture site with hydrocortisone. In the case of the procaine infiltrations, from 2 to 5 ml. was injected into the fracture haematoma and the effectiveness of this was demonstrated by confirming that forcible pain-free inversion of the foot could be performed after the injection. The patient was immediately encouraged to walk with no support. In the case of the hydrocortisone injections, a bleb of procaine was first injected into the skin in order to make the directing of the needle less painful to the patient. A fresh needle was then passed into the fracture site, which was opened by holding the foot in inversion, and from 25 to 75 mg. of hydrocortisone was injected, the patient then being encouraged to walk.

Twenty-eight patients were treated by activity from the start, a crêpe bandage being supplied as the only