antigen tests. In neither case did aspiration prevent further pus formation, so that open drainage was clearly required and was highly successful in terminating the course of the infection, though lymphadenitis must have persisted in a low-grade form for some time thereafter. There was no beneficial effect from the antibiotics, but this was as expected, gross suppuration being already obvious at the time of admission a month after the onset.

Spaulding and Hennessy (1960) record that 26 of their 83 cases progressed to gross suppuration between 12 and 40 days after the onset. They advise closed aspiration every day or on alternate days, and found that in only 6 of the 26 cases was aspiration required more than once. They regarded incision as inadvisable, owing to the risk of producing persistent sinuses. These views are not consistent with our experience in these two cases, as it became obvious that the abscesses would not have subsided with any other treatment than surgical drainage; this was successful and the incisions soon healed up.

Most writers agree that broad-spectrum antibiotics have no influence in preventing suppuration though they may reduce the degree of fever and, after surgical drainage, may promote healing. In mild cases which do not progress to the stage of suppuration conservative treatment is best employed, the infection subsiding within three to four weeks without the aid of any antibiotics.

Summary

Two cases of cat-scratch disease, both in children aged 13, are discussed. The clinical features were remarkably similar and both responded to treatment by surgical drainage and cleared up satisfactorily. No appreciable benefit was noted as a result of the employment of courses of demethylchlortetracycline in Case 1, or of oxytetracycline in Case 2, and suppuration occurred in all groups of infected lymph nodes in both cases. Various aspects of treatment of the disease are briefly discussed.

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REFERENCES

Brand, T. A., and Finkel, K. C. (1956). *Brit. med. J.*, **1**, 88.
Daniels, W. B., and MacMurray, F. G. (1954). *J. Amer. med. Ass.*, **154**, 1247.
Debré, R., Lamy, M., Jammet, M. L., Costil, L., and Mozziconacci, P. (1950). *Bull. Soc. méd. Hôp. Paris*, **66**, 76.
Greer, W. E. R., and Keefer, C. S. (1951). *New Engl. J. Med.*, **244**, 545.
Lancet, 1960, **2**, 532.
Lyon, R. L. (1956). *Lancet*, **2**, 555.
Spaulding, W. B., and Hennessy, J. N. (1960). *Amer. J. Med.*, **28**, 504.
Winship, T. (1953). *Amer. J. clin. Path.*, **23**, 1012.

Preliminary Communications

Secretor Status of Streptococcus Pyogenes Group A Carriers and Patients With Rheumatic Heart Disease or Acute Glomerulonephritis

Prompted by the studies of Glynn *et al.* (1959) on the possible relationship of the secretor status of patients to the occurrence of acute rheumatic fever, on which further data were published by Clarke *et al.* (1960) and briefly by Buckwalter *et al.* (1962), we began a study in 1961 on this and related points.

Although the study is not fully completed, some points have arisen which seem worthy of mention, especially as it is difficult to obtain enough material in one research centre, and extension of the survey to other centres would seem to be necessary.

The possible causal relationship between the secretor status of the patient and the occurrence of rheumatic fever may be explained in three ways: (1) a genetically determined higher attack rate of rheumatic fever after group A streptococcal infection in non-secretors; (2) a genetically determined higher infection rate with group A streptococci in non-secretors compared with secretors with the same attack rate for rheumatic fever in both groups; and (3) a combination of the two factors.

Preliminary data from our study tend to show that the second point may be of real importance. The carrier rate for group A streptococci was determined by taking throat swabs, which were cultured anaerobically on 5% sheep-blood-agar plates and in sheep-blood broth, and from which, after 48 hours, subcultures were made on blood-agar plates. This method produced the same result as when Pike's enrichment medium is used. The secretor status was determined with the haemagglutination-inhibition reaction, using extract from the *Ulex europaeus* as anti-H.

During a study of the carrier rate for beta-haemolytic streptococci group A in a random sample of about 11% of the total population of a village in Holland with 4,700 inhabitants, there was a definite preponderance of non-secretors among the carriers. Persons younger than 6 years of age were excluded from the sample. Table I gives the data for three consecutive monthly examinations of the same sample.

Of the 405 secretors and 97 non-secretors investigated three times, 102 secretors (25.2%) and 38 non-secretors (39.2%) had had group A streptococci on one or more occasions during these examinations ($P=0.025$). The apparent discrepancies in the number of patients in the three different investigations shown in Table I and the number of patients seen three times in these investigations are due to the fact that not all persons were seen three times.

TABLE I.—*Throat Carriers of S. pyogenes Group A in Secretors and Non-secretors in a Random Sample of the Total Population Above the Age of 5 of a Village in Holland*

Investigation	Total Series			Carriers of *Str. pyogenes* Group A		
	Total No.	Non-secretors		Total No.	Non-secretors	
		No.	%		No.	%
First ..	532	103	19·4	103	29	28·1
Second	547	107	19·6	83	24	28·9
Third ..	511	98	19·3	89	26	29·2

The blood-group distribution in this sample did not differ significantly for secretors and non-secretors, nor from the overall distribution for the people living in the Netherlands (Nijenhuis, 1961).

A further study on this point was made on military recruits on the day of their enlistment and after one month of training at the recruiting centre. The data are given in Table II. Again there is a preponderance of carriers of streptococci in the non-secretor groups in both samples, but the difference is significant only at a 5% level at the moment of enlistment. How far crowding in the barracks and the acquisition of new types influenced the difference

found at the two examinations could not be decided owing to the small number of carriers.

TABLE II.—*Throat Carriers of Str. pyogenes Group A in Secretor and Non-secretor Military Recruits, at Enlistment and After One Month of Military Training*

Investigation	Total Series			Carriers of *Str. pyogenes* Group A		
	Total No.	Non-secretors		Total No.	Non-secretors	
		No.	%		No.	%
First ..	1,503	332	22·1	131	39	29·8
Second	1,495	317	21·2	175	45	25·7

Of 262 patients with mitral stenosis, 15.3% were non-secretors. Patients with other than mitral defects were excluded from this investigation. As these patients came from all over the Netherlands and some from abroad, we did not succeed in obtaining a matched control group. But since 15.3% is lower than the percentage of non-secretors in the sample from the village and from the military sample mentioned above, whereas Clarke *et al.* (1960) and Buckwalter *et al.* (1962) found a higher percentage of non-secretors in their patients, this could indicate a difference in the composition of the various groups of patients examined. To investigate this possibility we divided the patients with mitral stenosis into various subgroups.

The blood-group distribution of the mitral-stenosis patients showed an excess of blood group A (54.1%) and a shortage of blood group O (40.2%) compared with normal controls (42.9% for group A and 45.3% for group O as described by Nijenhuis (1961)). This relative shortage of group O and relative excess of group A in patients with rheumatic carditis was also shown in the investigations of Clarke *et al.* (1960) and Buckwalter *et al.* (1962). Since the secretor/non-secretor ratio is about the same for persons with group O as for those with group A (Clarke *et al.*, 1956), this excess of group A or shortage of group O cannot in itself explain the secretor/non-secretor ratio in the different groups of patients.

The degree of fibrosis in 170 cases and the degree of calcification of the mitral valves in 161 cases, as felt by the exploring finger at operation, were noted. All patients who had other defects besides their mitral stenosis were excluded from the group who were treated surgically for the latter.

The distribution of these findings over secretors and non-secretors is shown in Table III. These data strongly suggest that the greater the calcification and fibrosis the lower the percentage of non-secretors. However, because of the large number of patients on whom data on calcification (46) and on fibrosis (37) are missing, and of whom eight and four respectively were non-secretors, it is not possible to come to a definite conclusion with regard to the calcification. For fibrosis, however, the chance that the group with unknown fibrosis will make the difference insignificant at a 5% level is under 5%.

To our knowledge, investigations regarding the possible correlation of acute nephritis and secretor status have never been reported in the literature. We therefore investigated 179 patients with this disease and 280 healthy school-children as controls: 43 (24.0%) of the patients and 66 (23.6%) of the controls were non-secretors. These preliminary figures do not indicate any real difference in the secretor/non-secretor ratio between our patients and our controls.

TABLE III.—*Condition of Mitral Valves Found During Commissurotomy for Mitral Stenosis in Secretor and Non-secretor Patients*

	Total No.	Non-secretors		P
		No.	%	
Calcified	55	4	7·3	<0·05
Non-calcified	106	23	21·7	
Fibrosis	130	16	12·3	<0·001
No fibrosis	40	15	37·5	

Summarizing, it seems possible that a non-secretor is not only liable to have *Str. pyogenes* group A in his throat more frequently, but when he develops a mitral stenosis there will be less tendency to fibrosis and calcification than in the secretor patient.

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REFERENCES

Buckwalter, J. A., Naifeh, G. S., and Auer, J. E. (1962). *Brit. med. J.*, **2**, 1023.
Clarke, C. A., Edwards, J. W., Haddock, D. R. W., Howel-Evans, A. W., McConnell, R. B., and Sheppard, P. M. (1956). Ibid., **2**, 725.
—— McConnell, R. B., and Sheppard, P. M. (1960). Ibid., **1**, 21.
Glynn, A. A., Glynn, L. E., and Holborow, E. J. (1959). Ibid., **2**, 266.
Nijenhuis, L. E. (1961). "Blood-group Frequencies in the Netherlands, Curaçao, Surinam, and New Guinea." Thesis, Amsterdam.

Medical Memoranda

Agranulocytosis During Administration of "Atromid"

"Atromid," an I.C.I. preparation containing ethyl-α-*p*-chloro-phenoxy-isobutyrate (clofibrate), has recently been introduced as a means of lowering high levels of serum cholesterol and triglycerides in patients with atherosclerosis. The only side-effects reported have been nausea and abdominal discomfort in a small minority of patients, and a potentiating effect on anticoagulants (Oliver *et al.*, 1963). The case here described is of a patient on phenindione anticoagulant therapy who developed agranulocytosis during administration of atromid and in whom normal granulopoiesis returned after withdrawal of this drug.

CASE REPORT

A woman of 57 was admitted as an emergency case in May, 1963, with the history of a sudden onset of dyspnoea and chest pain radiating into the left arm. The past history was not noteworthy, but there was a family history of hypertension in both sides of the family. Myocardial infarction was confirmed on the E.C.G. and estimation of serum transaminase. Her haemoglobin was 14.2 g./100 ml., white cells 12,000 per c.mm. (neutrophils 90%, lymphocytes 10%), E.S.R. 44 mm./hour (Wintrobe), blood urea 25 mg./100 ml., serum cholesterol 325 mg./100 ml.

The patient was immediately anticoagulated with phenindione and sedated with phenobarbitone 30 mg. t.d.s. and butobarbitone 200 mg. o.n. In view of the family history and high serum cholesterol, four weeks after admission atromid 0.5 g. t.d.s. was administered. It was observed that the dosage of phenindione required to keep the prothrombin time within the therapeutic range was halved after atromid administration. Six days