II). At this time also the early morning urine was of low and fixed specific gravity. After potassium repletion both these findings disappeared.

Denton, Wynn, McDonald, and Simon (1951) have reported two cases in which a similar amino-aciduria occurred during potassium depletion. Neither had glycosuria. In one of their two patients' treatment with potassium abolished the amino-aciduria; the other patient died while still depleted of potassium.

In his recent review Stanbury (1958) draws attention to the fact that amino-aciduria has resulted from severe and acquired renal tubular disturbances and that it has on occasion been reversible. We feel that the amino-aciduria in our patient was caused by a renal tubular disturbance for which the potassium deficiency was responsible. This interpretation is supported by the normal liver-function tests, by the low and fixed specific gravity of the urine at the time of amino-aciduria, and by the disappearance of the urinary abnormalities on correction of the potassium depletion. In the patient with nephrotic syndrome and both amino-aciduria and potassium depletion, reported by Stanbury and Macaulay (1957), treatment with potassium had no effect on the amino-aciduria. In their patient potassium depletion had been present for much longer than in our patient or those reported by Denton *et al.* (1951).

It is submitted that the possibility of potassium depletion and/or intracellular acidosis should be considered in every case of amino-aciduria and also that amino-aciduria should be looked for when potassium depletion is suspected.

Summary

A case is presented in which potassium depletion was accompanied by significant amino-aciduria, which disappeared with potassium therapy.

Attention is also drawn to the difficulties attendant upon a definitive diagnosis of potassium depletion, and it is again emphasized that normal serum potassium levels can be misleading.

Possible implications of the findings are discussed.

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PROPHYLACTIC USE OF ORAL PENICILLIN IN RHEUMATIC FEVER, CHOREA, AND CARDITIS

BY

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We have attempted to show the actual occurrence of rheumatic manifestations, intercurrent respiratory infections, and laboratory evidence of streptococcal infection in children suffering from heart disease and given penicillin prophylactically. At the St. Joseph's Children's Heart Hospital, Rainhill, under the devoted care of nursing and teaching sisters, their treatment and progress have careful and close surveillance. Three physicians regularly attend the hospital, so that each child is usually seen once in the course of each week and more often when necessary. The 40 beds are occupied mainly by children suffering from rheumatic heart disease, but there are also a few children who have congenital heart lesions. No attempt at a statistical study or a comparison of the effects of penicillin V with penicillin G has been made because the series is too small.

For the first year of the trial prophylaxis had not been given and the results are analysed under the heading of "control period." In the second year penicillin V tablets, 60 mg. b.d., were used; these tablets were taken on an empty stomach with the sister present at the bedside. A similar procedure was followed throughout the third year, penicillin G tablets, 200,000 units b.d., replacing the penicillin V.

The clinical progress of each child was commented on independently by each physician. Throat swabs were taken from every child at fortnightly intervals, and these were cultured, typed, and grouped at the Royal Southern Hospital. The delay between taking the swab and culturing it was between one and a half and two hours, but we first proved to our own satisfaction that under the particular circumstances the streptococcus group A will survive for much longer periods than this.

Blood was also taken from every child at fortnightly intervals and estimations were made of the erythrocyte sedimentation rate and the anti-streptolysin O (A.S.O.) content. With the kind co-operation of Distillers Company Ltd., the majority of specimens of blood were also forwarded to their laboratories, where the penicillin content was estimated.

Clinical Findings

TABLE I.—Manifestations of Acute Rheumatism

	No. of Cases	Rheumatic Fever	Chorea	Carditis	Total
Control period	38	5	3	0	8
Penicillin V ..	40	4	2	1	7
„ G ..	39	1	1	1	3

Rheumatic Fever

The cases of rheumatic fever occurring during the three years (Table I) were considered under two headings: group A, those who fit the Jones criteria (modified) as reported by the Royal College of Physicians (1957)—that is, showing two major or one major and two minor manifestations; and group B, those who, while not fulfilling the criteria, were considered to have rheumatic fever by three physicians who each saw the case independently.

Control Period.—During this phase there were three cases of group A type. One had been in hospital for some years, the others had been admitted 14 weeks and eight weeks previously, but as they were quiescent for some time before transfer from another hospital we regarded them as showing fresh infection and not as relapses. In addition, two cases were classified under group B, both of whom had been under our care for some months prior to the recrudescence.

Penicillin V Period.—One child had rheumatic fever of group A type as shown by fever, joint pains, pallor, malaise, and the appearance of a pericardial rub. This child had been well and in St. Joseph's for the previous 15 months. Three children had rheumatic fever of group B type. The first (J.B.) had been well for 18 months. She developed low-grade pyrexia, malaise, pallor, a raised A.S.O titre (1,250), and a raised E.S.R., the episode lasting four weeks. The second (K.M.) had been well for seven months. She developed fever, arthralgia, and swelling of one ankle-joint; three months later a relapse occurred with fever, occasional joint pains, sweating, and a raised E.S.R. The third (S.H.) had been well for six months. She had fever, erythema marginatum, pallor, sweating, a raised E.S.R., and a raised A.S.O. She remained in this state of ill-health for five months in all. Two further cases require brief mention at this point. Both of them were suspected of continuous low-grade activity throughout the year of observation, but their relapse had begun in the control period and we feel that, in view of the doubt about them, inclusion was not warranted in this analysis.

Penicillin G Period.—There was one case of rheumatic fever (group B). This patient (D.G.) had been well for 19 months. Her illness lasted for two and a half months, during which she was pyrexial and complained of vomiting, epistaxis, and joint pains. It should also be mentioned that shortly after the year's observation ended, and while the children were still on penicillin G, there was a further case of rheumatic fever (group A), but this has not been included in the present analysis.

Chorea

Control Period.—During this phase there were three cases of chorea.

Penicillin V Period.—Two cases of chorea occurred during this time. One child (B.O.C.) had been well for the previous 23 months and then developed moderately severe chorea which lasted for one month. The other (G.N.) had been admitted towards the end of the control period with chorea, which subsided shortly after admission. Three and a half months after the beginning of penicillin V he had a mild and short-lasting relapse.

Penicillin G Period.—A boy admitted during this period was well on admission. Two and a half months later he had a mild and short-lasting attack.

Rheumatic Carditis

For reasons given elsewhere (Chamberlain *et al.*, 1958) we have been in the habit of separating patients in whom the sole manifestation of rheumatic activity was carditis from those suffering from rheumatic fever or chorea with or without carditis. It is the former group with whom we are concerned in this section.

Control Period.—There were no cases during this year.

Penicillin V Period.—One child (V.H.) had been well for the previous 18 months. She had malaise, pallor, a rise in A.S.O titre, and an episode of congestive cardiac failure in the absence of any evidence of pulmonary infection or other cause for the failure.

Penicillin G Period.—One child (G.P.) had not had any active rheumatism in the previous two years, but had established aortic and mitral valvular disease. She had been in congestive failure seven months previously during a bout of bronchitis. She again developed congestive cardiac failure, this time accompanied by erythema marginatum and epistaxis and with no evidence of respiratory infection.

Intercurrent Infections

Respiratory infections, chiefly bronchitis, are prone to occur in children with established rheumatic heart disease, not uncommonly precipitating congestive cardiac failure, and sometimes death occurs in consequence. We therefore analysed the figures with regard to these episodes in the three years.

Control Period.—Six children had severe bronchitis—one child on three occasions, two on two occasions, and the remaining three only a single bout. Three of the children developed congestive failure, which we believe to have been precipitated by the bronchitis.

Penicillin V Period.—Six children had bouts of bronchitis, two of them having two attacks in the year. In two cases congestive cardiac failure appeared as a consequence of the illness. Two other children were in congestive failure when they developed superimposed bronchitis and so were extremely ill; fortunately both recovered in due course.

Penicillin G Period.—Three children had bronchitis—one child on two occasions. In two cases the attacks were mild and none of these children developed cardiac failure.

Special Investigations

Erythrocyte Sedimentation Rate.—Most of the children suffering from rheumatic fever, carditis, or bronchitis showed a rise in the E.S.R., but this was not an invariable finding. During the three years of our study several other children showed a prolonged and definite rise in the absence of any manifestation of rheumatism. It would thus seem that this investigation is of minor value only, and we have come to regard it as an unreliable guide in modifying the child's routine.

Throat Swabs.—Despite great care in taking, transmitting, and examining throat swabs a group A streptococcus (and this penicillin-resistant) was identified in only one child in the three years. This patient had satisfactory blood levels from penicillin G. On the other hand, many streptococci of other groups were isolated. Though complaint was often made of "sore throat," no typical case of tonsillitis was seen in the series.

TABLE II.—A.S.O. Titres

	October	November	December	January	February	March	April	May	June	July	August	September
<i>Penicillin V Period, 1955-6</i>												
M.H.												500
J.F.		250	350	250								250
T.R.											1,250	900
J.R.				350	500	350			250	250	350	500
E.R.		350	500	250	350	620	620					620
P.M.					350	350						
P.Me.			180	500	250			620				
G.T.												
<i>Penicillin G Period, 1956-7</i>												
M.H.	620	900	620	500	500	500	350 (Dis- charged)					
J.F.	350	500	180	350	500	500	500					
T.R.	900	900	620	620	900	500	500	350	620	620	(Dis- charged)	
J.R.	350	900	500	500	500	500	250					
E.R.	1,250	900	250									
F.M.								620	900	900	1,250	900
G.N.											500	620
S.H.	1,250	900	620	620	900	900	620	500	620	900	900	1,250
*G.W.	1,250	1,250	500	900	900	900	1,250	900	1,250	900	900	1,250
*J.D.	1,250	1,250	1,250	1,250	1,250	620	1,250	1,250	1,250	1,250	500	1,250
*D.J.	350			500	620	620	620	500	620	500	500	620

* These cases were not included in Table I, as they were suffering from active rheumatism on admission.

Anti-streptolysin O Titre.—Table I shows the number of children suffering from rheumatic fever, carditis, and chorea. These children have been omitted from Table II, as we wish to discuss the significance of an elevation of the A.S.O titre in the absence of any evidence of rheumatic activity. *Control Period:* Two children showed a rise in titre; in one case it varied from 500 to 620 for a period of eight months; in the other there was a rise to 350 for a period of two months.

Blood Penicillin Levels.—Since blood penicillin levels were estimated on all the children under observation at fortnightly intervals it is obviously impossible to reproduce all the figures here. We have therefore chosen at random the first four girls and the first four boys who were in St. Joseph's throughout the entire two-year period, and who are in fact representative of the remainder. By altering the time of giving the tablets and the times of taking blood we were able to obtain a guide to the varying level of the blood penicillin at different periods after taking the antibiotic. We have arranged Table III so that this is clearly shown rather

so we have calculated the mean level. The number of minutes between the taking of the tablet and the subsequent taking of blood is referred to as the dose-sampling time. For completeness a small number of children suffering from congenital heart disease were given penicillin V, 240 mg., and the blood levels so obtained were equivalent to those obtained with penicillin G, 200,000 units.

Discussion

Blood Penicillin Levels

Before discussing our clinical results it is necessary to consider whether the dosage of penicillin could be regarded as adequate. In a study of the prophylaxis of the recurrence of rheumatic fever (Kohn *et al.*, 1953) it was repeatedly found that the group A strains of the β haemolytic streptococcus were sensitive within the range of 0.001 to 0.08 Oxford unit of penicillin per ml., of these 80% were sensitive within the range of 0.01 to 0.02 unit.

Table III shows that, in the case of penicillin V a level much greater than 0.08 was reached in all cases between 60 and 90 minutes after taking the tablet, and was maintained for a minimum period of 90 minutes. Despite this, the clinical results were poor. In consequence, for the year of trial with penicillin G the dose was raised to 200,000 units b.d. With this dose the required blood level of 0.08 unit was reached within 30 to 60 minutes and maintained for longer than three hours in some cases; the maximum levels were also very much higher.

Since our study began, the Rheumatic Fever Committee of the Royal College of Physicians of London (1957) has suggested that oral penicillin G, 200,000 units b.d., or alternatively penicillin V, 120 mg., b.d., is effective when given over prolonged periods for the prevention of group A β haemolytic streptococcal infections in children and adults. Massell *et al.* (1957) suggest that oral buffered penicillin G, 200,000 units b.d., is effective, but that there is "still room for improvement of presently used regimes."

Clinical Findings

Previous literature has indicated that, in general, oral penicillin is effective in reducing the risk of relapse in rheumatic fever.

TABLE III.—Blood Penicillin Levels in Units Per ml.

Dose-Sampling Time in Minutes									
	30	60	90	120	150	180	210	240	
<i>Penicillin V, 60 mg. Tablet</i>									
J.R.	0	0.08	0.16	0.16	0.15	0.06			
E.R.	0	0.10	0.21	0.22	0.14	0.21	0.10	0	
D.G.	0.021	0.23	0.24	0.09	0.09	0.07		0.04	
V.H.	0	0	0.13	0.24	0.13	0.15			
B.O.C.	0	0.15	0.18	0.06	0.11	0.04	0.05		
G.T.	0	0	0.17	0.13	0.07	0.02			
G.N.	0	0.23	0.19	0.15	0.15	0.06	0.05		
P.N.	0	0.15		0.14	0.09	0.17	0.09		
<i>Penicillin G, 200,000 units Tablet</i>									
J.R.	0.40	0.65	0.26	0.56	0.12				
E.R.			0.23	0.26	0.76	0.16			
D.G.	0.75	0.72	0.50	0.75	0.28	0.36			
V.H.		0.98	0.28	0.54					
B.O.C.			1.11	1.02	0.64	0.32	0.22		
P.N.		0.60	0.71	0.23	0.30				
G.T.			1.02	0.75	0.35	0.06			
G.N.			0.087	1.02	0.56	0.17	0.01		

than reproduce the figures week by week for each child over a period of two years. Naturally it has followed that over such a long period of observation each child would have his blood taken at the same number of minutes after his tablet on more than one occasion. The resultant blood levels were closely similar in the vast majority of such occasions, but when it has not been

Pitt Evans (1950) used 100,000 units of calcium penicillin, given in 5% glucose, once daily before breakfast. This trial was carried out over two years on children admitted to a convalescent home for a maximal stay of six months, and they could not therefore be followed up long enough. There were no relapses of rheumatic fever in 155 cases, as against four in a control group.

Brick *et al.* (1950) used 50,000 units of oral penicillin twice daily in their 38 patients followed at monthly intervals for two years. Three had rheumatic recurrences as against six in a control series of the same number.

Kohn *et al.* (1953) gave their cases 200,000 units of oral penicillin four times a day for the first seven days of each month from October to June. In the third year of their study there were no relapses in 48 cases, in the fourth year one child relapsed out of 45 (this child had large infected tonsils), and in the fifth year one out of 40. Two other children also relapsed, but this was attributed to poor co-operation. Massell *et al.* (1957), using oral buffered penicillin, 200,000 units b.d., noted 2 relapses in 114 rheumatic subjects observed over 6,545 "patient weeks."

In our own series 7 children out of 40 relapsed within a year of prophylaxis with penicillin V. We have been unable to find a series in the literature with which to compare our results, but it is evident that, at least on the dose we used, they are unsatisfactory; we are at the moment continuing the trial on a schedule of 240 mg. b.d.

During the year on penicillin G, 3 children relapsed out of a total of 39, which might be taken as showing improvement on the two previous years, though it is not statistically significant. On the other hand, these results must be regarded as much less successful than those obtained by previous workers, and it is difficult to explain this discrepancy. It is conceivable that had our cases not been under close observation the case of chorea could have been missed, but this cannot be said of the other two, nor of the third child, whose relapse occurred shortly after the year under analysis. Whatever the reason, our figures leave no doubt that the schedule of treatment we used is clearly open to improvement.

An apparently more encouraging feature was the comparatively infrequent and mild bouts of chest infection occurring while the children were on penicillin G as compared with previous years.

Laboratory Evidence of Streptococcal Infection

Pitt Evans (1950) found that seven of the children receiving penicillin carried the group A streptococcus in the throat for two consecutive weeks and four for three consecutive weeks; there was one doubtful case of tonsillitis. None of the patients on penicillin showed a rise in A.S.O. titre though these were estimated at monthly intervals.

Brick *et al.* (1950) took monthly throat swabs and obtained a single positive swab from each of three children.

Kohn *et al.* (1953) studied the organisms grown from the throat in detail. Unfortunately it is not clear how many of the 110 positive swabs in the fourth year and the 47 positive swabs in the fifth year were from the children on penicillin, and how many from the controls. They do, however, comment that they have observed

positive cultures in young adults receiving from 100,000 to 600,000 units of penicillin daily. In no case did they find a penicillin-resistant organism. These workers did not undertake A.S.O. studies.

Massell *et al.* (1957) prepared throat cultures and estimated the A.S.O. titre every four weeks on each of the 114 rheumatic subjects attending their clinic and also at additional times when they attended these patients' homes in the event of illness. They regarded a positive culture or a rise in A.S.O. titre as evidence of infection. In seven children these were accompanied by clinical manifestations in the throat, 19 others were asymptomatic; of these latter the rise in A.S.O. titre was the only manifestation in seven.

Table II reveals that eight otherwise healthy children showed a marked rise in A.S.O. titre during the year 1955-6, and that four of these maintained this rise for several months during the subsequent year. Three further children showed a rise in A.S.O. titre in the year 1956-7, while three children admitted in this year maintained a high level for 12 months despite the early subsidence of any evidence of rheumatic activity.

Thus our figures bear a close resemblance to those of Massell *et al.*, though we were unable to support our findings with positive throat cultures except in one case.

Massell *et al.* have suggested from their work that it is unlikely that group C or G streptococci were responsible for the A.S.O. rise in their patients. We were consistently unable to isolate group A streptococci from the throats of our patients before or during a period of rising titre of A.S.O., despite considerable effort.

In view of this it seems an open question whether the A.S.O. rise in these cases was due to a streptococcus other than group A, or whether the group A streptococcus was harboured by the patient in some inaccessible region, and this despite penicillin prophylaxis.

If this latter suggestion is correct it would follow that children susceptible to rheumatic fever and carditis may harbour the streptococcus for a prolonged period and show antibody reaction to it without apparently suffering a relapse of their rheumatism. This would indicate that, in this phase, either some other requisite factor for the development of rheumatism is not present or some inhibiting factor is at work.

Summary

A study has been made of the prevention of manifestations of rheumatic disease in susceptible children by the use of penicillin V, 60 mg. b.d., and penicillin G, 200,000 units b.d. Neither regime proved completely effective. It is hoped that larger doses may prove more successful, and investigation along those lines is proceeding.

It has also been shown that many children on these prophylactic measures develop a high anti-streptolysin O titre in the absence of evidence of rheumatic disease, and the significance of this finding is discussed.

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ACCEPTANCE OF PSYCHIATRY BY THE MEDICAL STUDENT*

BY

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The attitudes which students acquire during their childhood, their school, and their preclinical training determine to a considerable extent their acceptance or rejection of psychiatry. But these attitudes are liable to reinforcement by their experience of clinical medicine and by their first contacts with psychiatry itself. It is difficult for the student to approach this subject with the same objectivity which he can give to other subjects. He is in danger of uncritical acceptance or of uncompromising rejection, and either attitude is equally undesirable. Many explanations have been offered for this. Among them is the fact that in most schools psychology and sociology are not taught adequately in the preclinical period and the place of psychiatry in the curriculum is relatively small.

Moreover, when psychology has been taught, it is not infrequently of the dry-as-dust academic variety, which the student cannot relate to the personal problems he meets in his patients and in everyday life. There is then the alleged hostility to psychiatry of clinical teachers in other fields of medicine and the undoubted tendency of the young student to identify himself with the attitudes of those whom he admires and respects. An excessive emphasis on psychopathology and on psychological determinism has also been blamed; for this is foreign to his thinking, and the impact, if it is felt at all, arouses anxiety in the student himself. More significant perhaps is the fact that there is as yet no body relevant to aetiology and treatment which is generally acceptable and accepted. Psychiatry is still cloven by the false antitheses of the psychosocial and organic approaches to the subject.

Teleological Aspect of Behaviour

No doubt all these and many other reasons play their part in shaping the student's attitudes. It is suggested, however, that the basic reasons lie deeper and are related to the explanatory hypotheses which each of

us must hold about the nature of man and about his behaviour. For 150 years biological science, and hence medicine, has tried to reach understanding of living phenomena in terms of physics and chemistry. A strict aetiology has implied a search for physical causation for all forms of disease and behaviour. The great successes of the anatomico-pathological concept of disease has abolished teleological explanations for biological events. Every event in nature is viewed retrospectively in terms of a chain or net of causative factors which determined it. Every endeavour has been made to narrow the concept of biology as science to the causal-mechanical methods of physics. It is indeed only reasonable and profitable to think about the functions and dysfunctions of a tissue, an organ, or an organ-system in terms of aetiological causation, but it has never been possible to think of human behaviour at its most integrated level in these terms. Indeed, it is only possible to do so when the concept of purpose or teleology is invoked.

We cannot avoid this forward-looking teleological aspect of behaviour, and this, which has been so unpopular and almost heretical among scientists, has raised the cry that psychiatry is "not scientific." Yet in the development of science teleological explanation probably always precedes and provides the ground for the discovery of causal mechanisms. It is a necessary stage in the development of science, and it is not intrinsically "unscientific." Indeed, science is every logical connexion of facts in thought whether it is aetiological or teleological.

The modern medical student, in contrast to his predecessors in other centuries, is little burdened by philosophy. He has escaped from preoccupation with mediaeval dualism—the mind-body problem—but he shares the prejudices of his cultural setting and he dislikes doubt and uncertainty. He is offered an immense array of technical knowledge, but he needs above all a satisfactory *explanatory hypothesis* about human behaviour with which he can work and into which he may fit, as best he can, all the data of human disease and disorder which he will meet. The great successes of aetiological medicine in the present century, based on the explanatory hypothesis of biology, are plain to all and are accepted without question. The failure of this hypothesis to cover all the facts of human behaviour and particularly those of psychological illness is, however, the threat which the student meets as soon as he encounters his first patients.

Determinism

The chief explanatory hypothesis of medicine rests upon physical determinism, the logic of which is that all physical events in the universe, and in the case of medicine human diseases, are determined by a necessary antecedent chain of physical causation. Physical determinism, however, while it is an entirely satisfactory explanatory hypothesis for biology, for macrophysics, and for much of medicine, is not so when it is applied to either normal or abnormal human behaviour. Strictly speaking, physical determinism is mechanism, and logically means that every event, including human behaviour, is completely explicable in terms of its antecedents. Thus, given complete knowledge of conditions, one would have complete knowledge of precisely how a person will, indeed *must*, act. The

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