factors are missing from one article of diet they will probably be supplied in others. It is by no means so sure that this remains true in the case of the child population. I would ask you here to give your attention to certain matters of fact, hoping that you will not think them trivial because they concern foodstuffs that are so familiar.

First consider our bread supply. The ancient controversy concerning the relative merits of brown and white bread has been tainted by prejudice. We have had enthusiasm without knowledge on the one side, and, to some extent at least, we have had the influence of trade interests on the other side. I hold no brief for whole-meal bread. I think the well-fed and well-to-do public may be justified, now the war is over, in insisting upon returning to its very white loaf, if this loaf suits its tastes and appetite better than any other. Other articles eaten will surely supply any deficiency. It is well to face the facts, however. The elaborate milling, the "refining" which the wheat grain suffers before it appears in the form of our white flours, deprives it entirely of its water-soluble vitamine. If our pure white wheaten flour were in this country to form so large a proportion of the whole diet as rice does in the East, beri-beri would with certainty appear among us. Indeed, in certain limited communities it is said to have followed the use of white bread. The wheat grain, no less than the rice grain, shows that an essential nutritive factor may have an unequal and limited distribution in a natural product, and illustrates the danger of "fractionating" natural foods to which I referred in my opening remarks.

It is clear that the danger of any deficiency in bread becomes the greater the more it bulks as a constituent of the whole diet. In the case of the poor—and especially the children of the poor—it undoubtedly bulks largely. Consider again the question of margarine as a substitute for butter. If the margarine is made wholly of vegetable fats, it lacks the fat-soluble vitamine. It is not by any means a trivial point to remember that brown bread and butter is, from the standpoint of vitamine supply, an excellent combination, while white bread and margarine may be a desperately bad one. Speaking in parenthesis, I may say that I happen to be aware from personal knowledge that the margarine industry in this country is quite alive to the significance of the facts. Given full liberty of action, the trade is, I believe, both willing and able to turn out products free from the reproach of this deficiency.

We must remember, again, that polished rice contributes not a little to the table of the poor, especially in some districts, and is often eaten plain boiled. Many of the advertised proprietary breakfast foods and invalid foods are practically devoid of vitamine.

In connexion with the antiscorbutic vitamine in particular, let it be called to mind that tinned foods are largely used in poor households—for one thing, because they often need no cooking. In these very households salads are rare, and during those periods of the year when fruit is dear extraordinarily small amounts of fresh foodstuffs are consumed.

Yet, it may be said, in spite of all this, beri-beri or pelagra or any disease like them is unknown in this country; and scurvy is, to say the least of it, very rare. But disturbances in health may exist without arriving at the extreme stage when the appearance of a severe set of symptoms leads to the recognition of actual disease. In particular is this true of conditions of malnutrition, and, unless the considerations I have put before you are wholly baseless, malnutrition may arise irrespective of the quantity of food eaten. It may depend not upon insufficient food but upon badly treated foods or upon an ill-chosen combination of foods. There is no point in all that I have said that I would like to emphasize more than this: Absence of an essential vitamine from the food means disease; but no more than a relative deficiency of a vitamine may mean bad nutrition. In my own opinion at least the diets of the poor—and once again it is the children who are chiefly concerned—often display such deficiency. In connexion with a point such as this a study of national supply, or even of family budgets, gives only partial information. Even when the total family food-budget is satisfactory, ignorant and ill-judged feeding of the child members is both possible and common.

The worst of it is that at present the facts are not generally recognized, even by the medical profession, and the ill health with which such deficiencies as we have been considering are associated is, in my belief, misunderstood and wrongly treated.

The case of rickets, however, stands out. Rickets is very common in this country. True rickets is a disease which, though crippling, is not fatal in itself. It is, nevertheless, a factor of supreme importance to the country because of its indirect effect upon the infantile death-rate. The child who is rickety is much more prone to other ailments—much more vulnerable in all respects—than a normal child. I will not dogmatize with regard to its origin. The mere suspicion that it may be due to a specific dietetic error, such as a deficiency in the supply of the fat-soluble vitamine, should awaken instant interest. Once we are convinced of the primary importance of this factor it will become of national importance to look to the question not alone of the quantity, but of the quality, of the fat in the diets of infants and of nursing mothers.

Mention of the nursing mother brings me to my last point. Evidence has accumulated to suggest that vitamins are not made in the animal body. The animal depends ultimately upon the plant for a supply. They accumulate in the tissues of herbivorous animals, and there, as well as in the plant, they are available for human consumption. But if vitamins are not made in the body at all, this means, in the case of the nursing mother, that if she consume a diet deficient in vitamins her milk may ultimately become deficient—to the complete detriment of the nutrition of her child. Animal experiments have already revealed such deficiencies in milk. It will be seen that the whole question of infant feeding is one in which a knowledge of these special accessory food factors is particularly important. The deficiencies which we have been considering are indeed of more urgent importance in connexion with the nutrition of the young than when adults are concerned. Hence what I believe to be the national importance of the subject. Whether this importance prove ultimately to be less than some of us believe, or whether—for this is quite possible—it may prove to be of as yet unsuspected importance, it should in any case awaken public interest. We are yet at the bare beginning of its study.

A Collective Investigation OF TEN THOUSAND RECRUITS WITH DOUBTFUL HEART CONDITIONS.

Conducted at the National Hospital for Diseases of the Heart by C. CHAPMAN GIBBES, R. O. MOON, S. RUSSELL WELLS, P. HAMILL, FREDERICK W. PRICE, and J. STRICKLAND GOODALL.

REPORT III,*

COMPILED BY

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THE ETIOLOGY OF AORTIC REGURGITATION.

It has been found extremely difficult, on account of the large amount of material with which we have to deal, and the number and variety of the problems that present themselves, to treat the different questions in logical order. In many cases the questions at issue are so intermixed that when the solution of any particular problem is attempted it is found necessary to solve another first. In our last report we dealt with the incidence of certain general diseases in our whole 10,000 cases, and arrived at the conclusion that there was a slight but definite correlation between a history of rheumatic fever and chorea, but no correlation between a history of rheumatic fever and either a history of "rheumatism," growing pains, or tonsillitis. We stated that the importance of these disorders as

* Part I was published in the *BRITISH MEDICAL JOURNAL*, May 18th, 1918, p. 556; Part II, September 7th, 1918, p. 248.

efficient causes in producing valvular lesions "could only be determined by a detailed examination of the etiological factors in the different cardiac disorders." Before undertaking such an investigation it was necessary to classify our material according to the various valvular and other defects. Diagnosis during life of cardiac lesions, however, is a question of opinion and not necessarily of fact; therefore before dealing with the etiology of aortic regurgitation, for instance, we should first discuss in detail the data on which the diagnosis was made, and this discussion would again lead us down side-paths which need not now be particularized. The result would be that all continuity of thought would apparently be lost in the presentation of our conclusions; we therefore propose first to discuss the etiology of certain affections of the heart, as far as our cases are concerned, reserving the reasons for our diagnoses for later communications, though some of these communications are already written. While asking the reader to suspend judgement for the time being, we desire to record the fact that the diagnoses have in all cases been arrived at as the result of physical examinations only, and that the etiological factors, such as a history of acute rheumatism and the like, have in no case been taken into consideration in arriving at a decision. Since physical signs alone enable a diagnosis of aortic regurgitation and mitral stenosis to be made with a considerable degree of certainty, we shall treat of the etiology of these lesions first; moreover, since it is generally admitted that the diastolic murmur over the base and down the sternum, which is characteristic of aortic regurgitation, and the apical presystolic and apical diastolic murmurs of mitral stenosis are as a rule associated with structural lesions of the valves, and hence are not likely to be confused with the so-called functional murmurs, there is a distinct advantage in investigating the etiology of these affections before proceeding to more debatable ground.

AORTIC REGURGITATION.

Etiology.

In dealing with the etiology of the cases of aortic regurgitation it must be pointed out in the first place that the cases dealt with were all between the ages of 16 and 42. Consequently, those causes which act late in life necessarily will not figure as prominently as those which occur earlier. It may also be suggested that, since we are dealing with a selected population, the medical boards may have sent a preponderance of a particular class. This, however, is hardly likely, for the War Office instruction was that cases only should be sent in which a doubt as to the classification for military purposes had arisen, and there is no reason to suppose that cases of aortic regurgitation due to any particular cause are likely to furnish a larger proportion of doubtful cases than those due to any other cause. The causes to which aortic regurgitation has been assigned are very numerous, including a great variety of microbic infections, but it is generally recognized that acute rheumatism is one of the commonest. Chorea, and the vaguer and slighter forms of rheumatism described as "growing pains" and the like, are credited with producing it in many cases. An increasing volume of opinion seems to exist which credits syphilis with being the efficient cause of a large proportion of cases; it has been stated that as many as 41 per cent. of aortic regurgitants show evidence of the presence of syphilis. The gonococcus is likewise suspect, while many consider that severe and continuous muscular strain is a common cause of the affection. The arterial changes grouped as atheroma are undoubtedly found, *post mortem*, to be the immediate cause of aortic regurgitation in many cases, particularly in later life, but this is a pathological phenomenon and a proximate rather than an original or true cause, and the question still remains—What was the cause of the atheroma?

Out of the 10,000 recruits examined 307 cases were considered to be definite examples of aortic regurgitation. In all of these a diastolic murmur was audible over the base or down the sternum, though the point of maximum intensity was not always over the aortic area, and in practically every instance other signs suggestive of aortic regurgitation were found. In many cases there was reason to believe that other lesions were present as well. The

following table shows the distribution of the 307 cases according to the diagnoses arrived at.

TABLE I.—Cases of Aortic Regurgitation.

Diagnosis.	No. of Cases.
Pure Aortic Lesions:	
Aortic regurgitation alone	94
Aortic regurgitation and aortic stenosis	14
Aortic regurgitation and doubtful aortic stenosis	16
Mixed Lesions:	
Aortic and mitral regurgitation	112
Aortic and mitral regurgitation and aortic stenosis	5
Aortic regurgitation and mitral stenosis	15
Aortic and mitral regurgitation and mitral stenosis	33
Aortic and mitral regurgitation and aortic and mitral stenosis	4
Aortic, mitral, and tricuspid regurgitation	1
Aortic regurgitation and old pericarditis	8
Aortic and mitral regurgitation and old pericarditis	2
Aortic and mitral regurgitation, mitral stenosis, and old pericarditis	3
Total	307

With a view to elucidating the influence of the various suggested factors in the production of aortic regurgitation in cases of men up to 42 years of age, the histories of the recruits have been analysed.

TABLE II.—Giving a Comparison between a History of Various Antecedent Diseases and a History of Strain in the whole 10,000 Recruits on the one hand, and like Histories in the 307 Aortic Regurgitants on the other.

Total 10,000 Recruits.			307 who showed Definite Signs of A.R.	
Possible Etiological Factor.	No. of Cases.	Percentage of Total 10,000.	No. of Cases.	Percentage of 307 A.R.'s.
Rheumatic fever*	1,921	19.2	162	52.8
Chorea	265	2.6	20	6.5
"Rheumatism"	1,613	16.1	55	17.9
Growing pains	2,649	26.5	84	27.3
Tonsillitis... ..	2,210	22.1	60	19.5
Scarlet fever	2,185	21.8	71	23.1
Diphtheria	728	7.3	22	7.2
Pneumonia	537	5.4	20	6.5
Influenza	5,629	56.3	142	46.3
Syphilis	200	2.0	11	3.6
Gonorrhoea	596	6.0	16	5.2
Strain	2,711	27.1	113	36.8

A.R. = Aortic regurgitation.

* No case was credited with rheumatic fever unless there was a clear history of pain and swelling in the joints, fever, and an illness necessitating confinement to bed over a period of some weeks. Some recruits naturally gave a history of more than one of the diseases recorded above.

It will be seen that the aortic cases give a strikingly higher percentage of histories of rheumatic fever than the average, and that they also show higher percentages of histories of chorea and strain. The absence of marked differences of percentages in the other groups makes us doubtful whether a history of these ailments in the cases of aortic regurgitants is not merely accidental.

The percentage method of investigation is open to so many sources of error in numbers like these, that while drawing attention to the figures for rheumatic fever and strain, we do not wish to base any conclusions on the table just given alone. There is, however, another method by which the problem may be investigated. We can calculate the correlation coefficient for aortic regurgitation and each of the factors which may be in etiological relation with

it.* One hundred and sixty-two cases gave a history of rheumatic fever and symptoms of aortic regurgitation, 145 aortic regurgitants gave no history of rheumatic fever, while 1,759 cases had a history of rheumatic fever but no evidence of aortic regurgitation, and 7,934 cases no signs or history of either. The coefficient of correlation comes out at 0.499, the limit of probable error being ± 0.015 , while $\chi^2 = 229$. This is very definite correlation, and may be defined as from moderate to considerable.

TABLE III.—Showing the Numbers for the Various Diseases or Possible Causes Investigated, with the Correlation Coefficient, Limits of Probable Error, and Value of χ^2 in Every Case.

A.	B.	Number of Cases.				Coefficient of Correlation.	Limit of Error.	χ^2 .
		History of A. and B.	History of A. only.	History of B. only.	No History of A. or B.			
A. R.	Rh. fever ...	162	145	1,759	7,934	0.499	0.015	229.83
"	"Rheumatism" ...	55	252	1,558	8,135	0.039	0.020	0.75
"	Growing pains ...	84	223	2,565	7,128	0.016	0.020	0.123
"	Chorea ...	20	287	245	9,448	0.179	0.019	18.34
"	Tonsillitis ...	60	247	2,150	7,543	0.053	0.020	1.20
"	Scarlet fever ...	71	236	2,114	7,579	0.025	0.020	0.30
"	Diphtheria ...	22	285	706	8,987	0.004	0.020	0.006
"	Pneumonia ...	20	287	517	9,176	0.045	0.020	0.82
"	Influenza ...	142	165	5,487	4,206	0.161	0.019	12.96
"	Syphilis ...	11	296	189	9,504	0.107	0.020	4.05
"	Gonorrhoea ...	16	291	580	9,113	0.032	0.020	0.317
"	Strain ...	113	194	2,598	7,095	0.164	0.019	15.07

A. = Aortic regurgitation. B. = Etiological factor under consideration—for example, rheumatic fever, chorea, or the like.

The value of χ^2 in the above table is so small in the case of "rheumatism," growing pains, tonsillitis, scarlet fever, diphtheria, pneumonia, syphilis, and gonorrhoea, as strongly to suggest that the system is not really a random sample from material in which the two variates are independent, but that, on the contrary, the two variates are correlated with each other in various degrees. χ^2 , however, is sufficiently high in the cases of rheumatic fever, chorea, influenza, and strain, practically to exclude a chance distribution. Before accepting the correlation figures for chorea, influenza, and strain, we have to remember that many cases giving a history of these illnesses also gave a history of rheumatic fever. In view of the well marked correlation of 0.499 ± 0.015 between aortic regurgitation and rheumatic fever, and the fact that no less than 52.8 per cent. of cases of aortic regurgitation gave a definite history of rheumatic fever, it is obviously dangerous to attribute aortic regurgitation to any other cause, such as influenza, strain, or syphilis, where there is also a history of rheumatic fever.

We therefore propose now to discuss in detail the cases of aortic regurgitation giving histories of the various diseases with which we are concerned, and shall compare the percentage of cases in any group giving a history of rheumatic fever with the percentage of the whole 307 cases that gave this history. It is obvious that if rheumatic fever and any other disease, such as syphilis for example, are both causes of aortic regurgitation, we should expect to find among a random sample of aortic regurgitants giving a history of syphilis a smaller proportion giving a

history of rheumatic fever than among aortic regurgitants as a whole; or, put in other words, there would be more without a history of rheumatic fever than among a sample of cases of aortic regurgitation selected without regard to the syphilitic history; while, if syphilis were not a cause of aortic valvular trouble, we should expect the proportion of rheumatic cases to be as big as or bigger than in the whole class. While we have shown in Report II that there is no correlation to be found between a history of rheumatic fever and either one of "rheumatism" or growing pains, and while the percentage and correlation figures just given lend no support to the view that they are efficient causes in producing aortic regurgitation, it is possible that such histories in some of these cases may refer to slighter attacks of the same ailment as that which we have dealt with under the head of rheumatic fever, though the evidence we have already brought forward makes it improbable that they do so as a rule. We have therefore thought it wise to include a heading of "History of 'Rheumatism,' Growing Pains, or Chorea"; the reasons for including chorea will be given under the head of that disease.

There is yet another method by which we can approach the problem. As it is well known that the damage caused by acute rheumatism is by no means confined to one set of valves, and that it frequently produces multiple lesions, we can see if, in cases of aortic regurgitation, there is a great excess of either simple or multiple lesions in those cases giving a history of any particular disease beyond what we should expect if true rheumatism were the cause.

Rheumatic Fever.

One hundred and sixty-two of the 307 cases of aortic regurgitation, or 52.8 per cent., gave a history of this disease, as against 19.2 per cent. of the 10,000 cases examined. The correlation coefficient between a history of rheumatic fever and aortic regurgitation was 0.499 ± 0.015 , while $\chi^2 = 229$. These facts are powerful arguments in favour of rheumatic fever being an important cause of aortic regurgitation. In comparing the ratio of the pure aortic lesions to mixed lesions we thought it wise, in the first instance, to divide our cases into three groups, shown in Table IV:

TABLE IV.—Group 1, a Clear and Definite History of Rheumatic Fever. Group 2, a Rheumatic History such as "Rheumatism," Growing Pains, or Chorea, but no Definite History of Rheumatic Fever. Group 3, no History Suggestive of any form of Rheumatism.

Group.	Total	Pure Aortic Lesions.	Mixed Lesions.	Ratio of Aortic to Mixed Lesions.
1. With history of rheumatic fever	162	70=43.21%	92=56.79%	0.76
2. With history of "rheumatism," growing pains, or chorea	73	29=39.72%	44=60.27%	0.66
3. With no rheumatic history	72	25=34.72%	47=65.28%	0.53
Total ...	307	124	183	0.68

It will be seen that the ratio between aortic and mixed lesions steadily falls as we pass from Groups 1 to 3, and that in those cases showing no history of any form of rheumatism the largest proportion of mixed lesions occurs. A reference to the table already given of the diagnoses will reveal the fact that in most cases where a mixed lesion occurred mitral regurgitation was present, and as the diagnoses were founded on physical signs alone, and largely on murmurs, this may have been either due to structural damage of the valves or to relative incompetence from stretching of the mitral ring or other causes.

"Rheumatism."

Fifty-five of the 307 cases gave a history of "rheumatism"; 17 of these also gave a definite history of rheumatic fever either before or after the "rheumatism." In the remaining 38 no clear account of rheumatic fever, as we have defined it, could be elicited, though in some there was a history of growing pains or chorea as well as rheumatism.

* The formulae used were—

$$Q_s = \sin \frac{\pi}{2} \frac{1}{\sqrt{1 + K^2}}$$

$$\text{where } K^2 = \frac{4abcdN^2}{(ad-bc)^2(a+d)(b+c)}$$

Q_s being taken as a sufficiently near approximation to r (see Pearson, *Phil. Trans.*, 1900, series A, vol. 195).

$$(Poc)\chi^2 = \frac{N(ad-bc)^2}{(a+b)(c+d)(b+d)(a+c)}$$

$$\text{Error: } \pm 2.02347 \times \frac{1-r^2}{\sqrt{N}}$$

TABLE V.—Comparison of the 307 Aortic Regurgitants with the 55 giving a History of "Rheumatism," as regards the Incidence of Rheumatic Fever.

	No. of Cases	With History of Rh. Fever.	Without History of Rh. Fever.
Total Ao.R.	307	162=52.8%	145=47.2%
Ao.R. and "rheumatism" ...	55	17=30.9%	38=69.1%

The fact that a smaller proportion of cases with a history of "rheumatism" (30.9 per cent. against 52.8 per cent.) give a history of rheumatic fever than the whole 307 do, suggests that "rheumatism" may have been the cause of the aortic regurgitation in some of these cases; or, what would come to the same thing, that some cases of the disease we have called rheumatic fever were included in the "rheumatic" group.

Of the 38 "rheumatic" cases without a history of rheumatic fever, 14 were diagnosed as instances of aortic lesions, and 24 as mixed lesions—a ratio of 0.58 as compared with 0.76 for rheumatic fever.

Growing Pains.

Out of the 307 aortic regurgitants, 84 gave histories of growing pains; 48 of these gave a definite history of rheumatic fever as well, and of the remaining 36 some 7 gave a history of either "rheumatism" or chorea in addition to growing pains.

TABLE VI.—Comparison between the Incidence of Rheumatic Fever in this Group with its Incidence in the whole 307 Cases.

	No. of Cases.	With History of Rh. Fever.	Without History of Rh. Fever.
Total Ao.R.	307	162=52.8%	145=47.2%
Ao.R. and growing pains ...	84	48=57.14%	36=42.85%

The close agreement between these figures obviously makes it unsafe to attribute aortic regurgitation to growing pains. Of the 36 cases without a history of rheumatic fever 15 were classed as pure aortic and 21 as mixed lesions; a ratio of 0.71, which is practically the same as the 0.76 found in the cases with a history of rheumatic fever.

Chorea.

In 20 cases there was a history of chorea, 10 of these giving also a history of rheumatic fever, 6 of chorea with either growing pains or "rheumatism," and 4 a history of chorea without any other rheumatic manifestation.

TABLE VII.—Comparison between the Rheumatic History of those Cases showing Chorea and the Rheumatic History of the Total Number of Aortic Regurgitants.

	No. of Cases.	With History of Rh. Fever.	Without History of Rh. Fever.
Total Ao.R.	307	162=52.8%	145=47.2%
Ao.R. and chorea	20	10=50.0%	10=50.0%

The question arises whether those cases in which there is a history of chorea should be classified definitely with true rheumatism, and put in the same group as rheumatic fever, or not. In favour of such a classification are the facts that chorea is a definite illness, persisting for a long time, and hence it is comparatively easy, from a careful inquiry into the history of the case, to make sure of it having occurred. Since at the present time there seems to be a growing tendency to regard true chorea and acute rheumatism as different manifestations of the same disease, and since there is, as shown in our last report, a certain degree of correlation between a history of chorea and rheumatic fever, we were at first inclined to classify them together, but, on further consideration, it was thought wiser to adopt a more conservative attitude, and to put the chorea cases together with the more doubtful rheumatic manifestations such as "rheumatism" and growing pains, and only to credit to rheumatic fever those cases where there was a history of true rheumatic fever as well as of chorea. One of the considerations which induced us to do this was the fact shown in the above

table, that the proportion of chorea cases giving a history of acute rheumatism—namely, 50 per cent.—was practically the same as the proportion of total cases of aortic regurgitation giving a similar history, which was 52.8 per cent.

Of the ten cases without a history of rheumatic fever, three were considered to be examples of pure aortic lesions, and seven of mixed lesions—a ratio of 0.43. The numbers are, however, too small to be worth considering.

Tonsillitis.

Tonsillitis is considered by some to be an affection of rheumatic origin; others, while not admitting this, yet consider that it may be the cause of valvular affections. Confining ourselves for the moment entirely to aortic regurgitation, we may say at once that our figures for these cases afford but little support of these views. Sixty cases in all—that is, 19.5 per cent. of the 307—gave a history of tonsillitis, but in 40 there was also a history of rheumatic fever, and in 7 one either of "rheumatism," growing pains, or chorea. In other words, in 78.3 per cent. there was a possible rheumatic history, and in only 21.7 was this absent.

TABLE VIII.—Tonsillitis.

	No. of Cases.	With Rh. Fever.	With Rh., G.P., or Ch.	Without Rh. History
Total Ao.R.	307	162=52.8%	73=23.8%	72=23.4%
Ao.R. and tonsillitis ...	60	40=66.6%	7=11.7%	13=21.7%

G.P. = Growing pains; Ch. = chorea.

The ratio between the cases showing pure aortic lesions and those with mixed lesions was 0.58, as against 0.76 for rheumatic fever; while of the 20 without a history of rheumatic fever 6 show pure aortic lesions and 14 mixed—a ratio of 0.43, so that here there is a preponderance of mixed lesions.

Scarlet Fever.

Scarlet fever has been regarded as a not uncommon antecedent of valvular lesions; but, as this fever is not infrequently followed by a fever bearing a close resemblance to acute rheumatism, many have thought that the valvulites following scarlet fever are really of rheumatic origin. The figures for these cases give a slight support to this view. In 9 out of the 45 cases where rheumatic fever and scarlet fever both occur, the rheumatic fever followed immediately after the scarlet fever—that is, in 20 per cent. of the cases there was a close connexion between them. In the remaining 36 cases the two diseases were separated by some years.

Seventy-one out of the 307 cases—that is to say, 23.1 per cent.—gave a history of scarlet fever. In 45, as mentioned already, there was a history of rheumatic fever; in 13 a history of either "rheumatism," growing pains, or chorea; in 13 there was no history of any rheumatic affection.

TABLE IX.—Contrasting the Total of Aortic Regurgitants with those giving a History of Scarlet Fever, showing a Slight Preponderance (63.4 per cent. as against 52.8 per cent.) giving a History of Rheumatic Fever where Scarlet Fever had also occurred; in the Scarlet Fever Group there is rather a Smaller Proportion with no Rheumatic History.

	No. of Cases.	With Rh. Fever.	With Rh., G.P., or Ch.	Without Rh. History
Total Ao.R.	307	162=52.8%	73=23.8%	72=23.4%
Ao.R. and scarlet fever ...	71	45=63.4%	13=18.3%	13=18.3%

Ratio of Pure to Mixed Lesions.—In 27 cases lesions of the aortic valve alone were diagnosed, in 44 the mitral valve was also affected—a ratio of 0.61 against 0.76 for the cases with a history of rheumatic fever. Of the 26 cases without a history of rheumatic fever, 8 had pure aortic lesions and 18 mixed—a ratio of 0.4, so here, as in the case of tonsillitis, mixed lesions predominate.

These results do not in themselves lend any support to the view that scarlet fever is an efficient cause of aortic regurgitation.

Diphtheria.

Of the 307 cases of aortic regurgitation 22, that is, 7.2 per cent., gave a history of diphtheria.

TABLE X.—Showing the Distribution of Cases with a History of Diphtheria and a Comparison between them and the Total Aortic Regurgitants with regard to their Rheumatic History.

	No. of Cases.	With Rh. Fever.	With Rh., G.R., or Ch.	Without Rh. History
Total Ao.R. ...	307	162=52.8%	73=23.8%	72=23.4%
Ao.R. and diphtheria ...	22	15=68.2%	5=22.7%	2= 9.1%

A comparatively large proportion of these cases were pure aortic lesions, namely, 13 as against 9 with mixed lesions, the ratio here being 1.44 instead of the ratio 0.76 shown by the total number of aortic cases with a history of rheumatic fever. Of the 7 cases without a history of rheumatic fever, 4 were pure aortic lesions and 3 mixed—a ratio of 1.3.

These figures cannot be said to lend any support to the idea that diphtheria is a cause of aortic regurgitation, but the large proportion of pure aortic cases in this group is interesting, though it may well be accidental.

Pneumonia.

The proportion giving a history of pneumonia is not large, 20 cases in all, that is, about 5.9 per cent. of the total 307 cases. Twelve of these gave histories of acute rheumatism, 2 of some rheumatic affection, and 6 no rheumatic history. Here again, as the table shows, there is no evidence from the figures that pneumonia is in itself a cause of aortic regurgitation.

TABLE XI.—Pneumonia.

	No. of Cases.	With Rh. Fever.	With Rh., G.P., or Ch.	Without Rh. History
Total Ao.R. ...	307	162=52.8%	73=23.8%	72=23.4%
Ao.R. and pneumonia ...	20	12=60.0%	8=40.0%	6=30.0%

Since an attack of pneumonia is apt to affect the muscle of the ventricles, it is not surprising that these cases of aortic regurgitation with a history of pneumonia show a larger proportion of mixed lesions than is shown by the 162 cases with a history of acute rheumatism; the ratio of pure aortic to mixed lesions being 0.43 where pneumonia had occurred, as against 0.76. The cases without a history of rheumatic fever comprised 2 cases of pure aortic lesions and 4 of mixed, a ratio of 0.5.

The analysis of these cases is against pneumonia being a cause of aortic regurgitation.

Influenza.

As judged by these cases, influenza is but of little importance in the production of aortic regurgitation. Of the 307 cases, 142 (46.3 per cent.) gave a history of one or more attacks of influenza, but in 82 of these there was also a history of rheumatic fever, and in 31 a history of chorea, "rheumatism," or growing pains—that is, in 79.6 per cent. there was a possible rheumatic history.

TABLE XII.—Influenza.

	No. of Cases.	With Rh. Fever.	With Rh., G.P., or Ch.	Without Rh. History
Total Ao.R. ...	307	162=52.8%	73=23.8%	72=23.4%
Ao.R. and influenza ...	142	82=57.7%	31=21.8%	29=20.5%

The ratio of pure aortic to mixed lesions was 0.73, which is very similar to that of the total number of aortic regurgitants with a history of rheumatic fever. Of the 60 cases without a history of rheumatic fever, 21 were pure aortic cases and 39 were instances of mixed lesions, the ratio being 0.5, against 0.76 for the whole of the cases of aortic regurgitation with a history of rheumatic fever.

(To be continued.)

A PLEA FOR THE HOMOGENOUS NERVE GRAFT.

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In the majority of cases of complete nerve division that have resulted from gunshot injury the nerve ends are capable of approximation for suture when operation is performed for this purpose. But there are quite large numbers in which approximation is not possible owing to the destruction of a considerable segment of nerve trunk, together with extensive tissue loss. Many such a case has been operated upon, and the surgeon who has been confronted with the problem of how to make good the loss has failed to proceed further or has resorted to one of various illogical expedients, without, in my experience, any good result.

As surgical specialist to the Pensioners' Resurvey Boards at the Duke of York's Head Quarters I have had ample opportunity of observing these results, and I have seen no instance of recovered nerve function where bridging with foreign material has been done, or where the nerve ends have been connected by a wrapping of fascia, Cargile membrane, or vein. If Van Lair's experiments are to be relied upon, it is inconceivable how anatomical union can by such means occur.

He found, as a result of nerve trunk excisions in animals, that where a space of 4 cm. was left between the nerve ends no recovery occurred; with an excision of 2 cm., recovery did not occur for thirty months; with a 1 cm. excision, recovery of function was delayed to fourteen months; whereas in the case of approximation and suture, recovery occurred in eight months.

These experimental operations were aseptically performed so that there would be absence of scar tissue and the circumneural areolar and fatty tissue left would offer but little obstruction to growth, if any, furnishing rather an admirable conducting sheath to support and direct the growing nerve fibres in a correct course were this all that was required.

But it is clear that something more than a conduit is necessary. It is recognized that the neurilemma elements are essential to sustain and nourish nerve fibres in their growth. Where interrupting scar is present, as it invariably is in gunshot injuries, it is doubtful if the smallest breach is passed. Wherever a nerve has been wounded and divided, completely or in part, palpation will reveal the presence of a neuroma, which means a mechanical and functional block, and my experience is that until this block is removed and direct approximation of healthy elements obtains, function is not restored, in spite of massage, electricity, and other futile means wastefully expended over a period of years. It is not reasonable to expect growing fibres, after being contorted in scar, to traverse such a bewildering obstacle and reach their destination. Thus excision of a lateral scar the result of a lateral notch is to perform a recognized operation.

In view of the large number of pensioners still languishing with disabilities that are the direct result of sectional nerve lesions it is fortunately proving by accumulating evidence that lapse of time should be no deterrent against suture. The longest period that has elapsed between section and suture of the cases I have observed that have been followed by recovery is two years and seven months. This was a case of complete ulnar nerve division.

The results in general of nerve suture as they appear at the Resurvey Boards are remarkably good, and the degree of restoration of function is found on palpation to be in inverse ratio to the development of a neuromatous swelling where junction has been effected. Conformity at the site of union approximates the normal when coaptation is exact and restoration of function is complete; whereas if operation results in failure, marked neuromatous swelling occurs, which is a help in deciding for reoperation without undue delay. "What is anatomically correct will not be physiologically wrong" is a phrase that in nerve surgery may be taken as a guide if operative results are to give satisfaction. It is not possible to make certain of establishing perfect histological union, but the oval contour and striation of a nerve does permit of coaptation that approximates morphological perfection, so that torsion can be avoided and nerve fibres in their growth are likely on the whole to