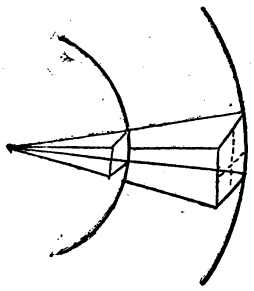
If a sphere dilate, therefore, to twice its diameter or radius, it would have an internal surface area four times as large as before, and its walls, to cover that greater surface, would necessarily become four times as thin. It may be objected that the heart is not a sphere; the same principle, however, applies, so that if the heart were dilated to double its diameter its inner surface would be approximately four times as large, and the heart walls four times as thin. For this reason alone it would be roughly four times as difficult for the heart to contract on its contained blood, there being four times the area of blood to contract on, or four times the number of units of area of blood to resist its contraction.



This, however, is not the whole difficulty, for the heart's power of exerting a pressure on its contents becomes halved when its diameter is doubled, so that while the work to be done is quadrupled, the working power is halved, or, in other words, the heart's embarrassments are increased eightfold.

The proof that the working power of the heart is thus lowered may be presented as follows: The force with which a hollow rubber or muscular sphere can contract on its contents is merely the force with which one-half of the sphere can pull the other half towards itself, and is measured by the pull across any circumference.

If p = the pull of a unit of muscle;
,, c = the length of circumference, which is $2\pi r$, where r is the radius;
,, t = the thickness of muscle at the circumference;
then pct = the pull across the whole circumference drawing the two halves of the hollow sphere together, and so compressing its contents.

Now pct is the same as $p2\pi rt$, but I have already pointed out that t, the thickness, becomes four times less when r, the radius, is doubled.

When, therefore, the sphere is doubled in radius or diameter the pull across the circumference is doubled in virtue of the circumference being double as long, but it is one-quarter in strength in view of the fact that the muscular material is only one-quarter as thick. On the whole, therefore, the pull is halved.

If the sphere were further dilated, say to three times its former radius, the pull would amount to $p\ 2\pi\ 3r\frac{t}{9}$, or, in other words, it would gain threefold by the increase in circumference, and lose ninefold by the decrease in thickness, since the thickness decreases inversely as the square of the radius, as already shown.

Though the heart is not a true sphere it is a hollow roundish viscus, and, in its measure, is subject to the same physical principles. When enlarged it has more units of surface of blood, increasing, more or less, as the square of the diameter to resist its pressure; and it has less power to contract, the power changing in inverse ratio to the diameter.

From these two causes combined, the compression the heart can exert on its contents would decrease eightfold if the diameter of its chamber were doubled, and twenty-seven-fold if the diameter were trebled — that is, it varies always inversely as the *cube* of the diameter or radius.

As applied to the heart or any hollow viscus in the body, such as the bladder or uterus, these figures must not be taken as of any definite value, other than showing a physical principle to which they are subject, and the immense mechanical drawback of dilatation.

This consideration shows why "breaking the waters" when delivery is well advanced is followed by such powerful contractions on the foetus. It is quite likely that the contractions are no more powerful than before, but merely more efficient. The surface to contract on is smaller, and the two halves of the uterus can be drawn towards one another with more force, simply because the uterus is thickened. With less to contract on and a thicker contracting wall no increased muscular effort is required to produce an enormously increased expulsive effect, the pressure on the foetus could, more or less, rise inversely as the cube of the decrease in diameter.

A dilated heart may be greatly relieved by blood-letting, because it allows the distended ventricle to contract; and, once contracted, the muscle wall may, at least for a while, prevent its chamber from becoming over-large again, for it is much easier to keep a chamber small than to reduce it when once large.

I have endeavoured to show in many papers in the BRITISH MEDICAL JOURNAL and elsewhere, in the past, that the same physical principle applied to the auricle probably explains most that is puzzling in the behaviour of the heart in mitral stenosis. The presystolic crescendo murmur and its running up to and into the first sound, the absence of ventricular regurgitation through an incompetent mitral valve, the presence of regurgitation and a systolic murmur replacing the presystolic when the auricle later on dilates and allows regurgitation into it, and so on.

The important thing, however, to first realize is that the size of the cardiac chamber at any time determines, more than is readily imagined possible, the power of the heart to empty itself, the heart weakening, as it dilates, inversely, more or less, as the *cube* of the dilatation.

A STUDY OF THE PNEUMOCOCCUS AND STREPTOCOCCUS GROUPS IN THEIR RELATION TO INFLUENZA.*

BY

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THE object of this investigation was a study of the pneumococci and streptococci from cases of epidemic influenza, firstly in their relation to the earliest stage of illness, when the infection was presumably a comparatively pure one; and, secondly, with regard to their part in the more advanced cases, when pulmonary complications had set in.

The preliminary portion of the inquiry was directed to questions of technique. It was necessary to define precisely what constitutes a pneumococcus and what a streptococcus, and what criteria may be adopted for distinguishing various types of these organisms from one another. It was concluded that, while there are various properties which, if taken together, may serve to distinguish the pneumococcus and streptococcus groups from each other, there is only one test which, if used as a single test, can serve to differentiate the two groups—the bile test. The solubility of pneumococcus in bile, apparently an empirical reaction, is nevertheless an indication of a specific chemical constitution of the pneumococcus, which it does not share with the streptococcus group. This test was then adopted as the invariable means of distinguishing the two groups, while the other tests in common use—inulin fermentation, presence of capsule, type of colony, morphology, virulence to mice, power of growth in certain media, homogeneity or granularity of growth in plain broth, etc., were used only as secondary confirmatory tests.

The methods adopted for differentiating the different types of organisms within these two groups were for the pneumococci serological, agglutination tests with the Rockefeller Types I, II, and III serums being carried out; for the streptococci, biochemical, fermentation and haemolytic tests being used.

The cases studied may be taken as typical of those occurring in the Edinburgh district during the second and third waves of the epidemic, that is, during those outbreaks which reached their height in November, 1918, and in February, 1919. One hundred and eight cases were studied completely from the point of view of their

*Advance reports on this subject have already been communicated as follows: (1) Preliminary Report to the Medical Research Committee, April, 1919; (2) Paper read before the Edinburgh Medico-Chirurgical Society, May 14th, 1919, and published by the *Edinburgh Medical Journal*, July, 1919; and (3) Paper read before the Pathological Society of Great Britain and Ireland, July, 1919; the Report (4) of which the present paper is an abstract was sent to the Medical Research Committee in April, 1920, and will be published in the *Edinburgh Medical Journal*.

pneumococci and streptococci; seventy-five were civilians, the remainder soldiers.

Specimens from cases of "pure influenza" were usually taken during the first three days of illness; from cases with pulmonary complications naturally, as a rule, at a later stage, varying from three days upwards. From the former group of cases nasopharyngeal swabs were usually employed; from the latter, sputum. Forty of these specimens were plated out direct on media suitable for pneumococcus; in the remaining sixty-eight a preliminary passage through mice with subsequent plating from heart blood and peritoneum was used along with direct plating of the specimen on suitable media.

The Incidence of Pneumococci and Streptococci in the Cases.

Pure Influenza Cases.—In this group of cases pneumococcus was isolated from 6 out of 44 cases, or from 13.6 per cent.; while streptococci were obtained from 44 out of the same 44 cases, or from 100 per cent.

Cases of Influenza with Pulmonary Complications.—From the second group of cases pneumococci were isolated from 50 out of 64 cases, that is, from 78.1 per cent.; streptococci were isolated from all the 64 cases, or 100 per cent.

It was therefore possible to draw the conclusion that the pneumococcus group played little part in the cases of pure influenza, and could therefore be excluded from the list of suspected initiating agents; that its great increase in number in the cases suffering from pulmonary complications, having in mind the known pathogenicity of many pneumococci, suggested an important rôle in these complications. It was further shown that streptococci were invariably present in both simple and complicated cases.

The next step was therefore to ascertain if these pneumococci in the secondary complications were of one type, and similarly if the streptococci of the simple and complicated cases differed on the one hand from each other, and on the other hand from the streptococci normally inhabiting the mouths of human beings.

Types of Pneumococci Present.—Fifty-one strains of bile soluble diplococci from 34 cases of influenza were typed out, using Rockefeller I, II and III serums. Of these 34 cases three yielded Type I pneumococcus, three Type II pneumococcus, two Type III pneumococcus, and 26 pneumococci of Group IV. An attempt was made further to subdivide the Group IV pneumococci with the object of determining whether those present in influenza were or were not the same as those sometimes obtained from healthy throats. The attempt was unsuccessful.

Types of Pneumococci in relation to Types of Clinical Case.—The eight strains of Types I, II and III pneumococci were, with one technical exception, derived from cases with well-marked pulmonary involvement, in each case with consolidation, shown either clinically or at *post-mortem* examination. The exception was a case of "pure influenza," showing no pulmonary symptoms at the time a Type I pneumococcus was obtained; she manifested pulmonary symptoms within a day or two and died within a week of pneumonia. One of the Type II pneumococci was from a lobar pneumonia in which the existence of influenza was doubtful.

The Group IV pneumococci were derived from cases of pure influenza; from cases of influenzal bronchitis; from cases of influenzal bronchopneumonia; and from cases in which, as shown both clinically and by *post-mortem* examination, extensive consolidation was present. They were isolated from cases in which the illness was trivial, from cases in which a very chronic chest condition supervened, and from cases with a rapidly fatal termination.

Incidence of Haemolytic and Non-Haemolytic Streptococci.

For the purpose of differentiation the action on blood agar was employed.

One hundred and nine strains of streptococci derived from 57 cases were subjected to the test. From 28 cases of pure influenza haemolytic streptococci were not once obtained; of 29 cases with pulmonary complications, 8 yielded haemolytic streptococci, or 28 per cent. of these complicated cases.

The Haemolytic Streptococci.—The same conclusion was thus arrived at with regard to the haemolytic streptococci as had been come to concerning the pneumococci; there was evidence that they played no part in the production of pure influenza, but they were apparently active as secondary invaders of the lung in more than a quarter of the cases with pulmonary complications, as compared with the figure of three-quarters of the cases obtained for the pneumococci.

The Non-Haemolytic Streptococci.—These were obtained from all the cases examined save two. In these two, haemolytic streptococci were obtained in the culture, in one from lung substance (*post mortem*), in the other from empyema pus. Holman's classification for the non-haemolytic streptococci was adopted. Ninety strains, derived from 46 cases, were tested in this way. Seventy-seven of these were grouped as follows: *faecalis*, 0; *non-haemolyticus I*, 1; *mitis*, 44; *salivarius*, 26; *non-haemolyticus II*, 1; *non-haemolyticus III*, 0; *equinus*, 5; of the remaining 13 strains, 9 were either *mitis* or *salivarius*; 4 either *faecalis* or *non-haemolyticus I*. The 90 strains may therefore be grouped as follows: 79 were either *mitis* or *salivarius*, while 11 were of other varieties. As regards their case incidence, either *mitis* or *salivarius*, or both, were obtained from 21 out of 22 cases of pure influenza; while either *mitis* or *salivarius*, or both, were obtained from 22 out of 24 cases with pulmonary complications.

Assuming Holman's tests to be adequate for the differentiation of these organisms, one would be able to conclude that their presence in cases of influenza is not significant of an infection from without, as such types are met with in normal mouths. Evidence will be produced, which cannot be given in detail here, to show that Holman's tests are not adequate for this purpose. Taking other factors into consideration, however, the conclusion was arrived at that it was in the highest degree improbable that organisms of this group were responsible as initiating agents in the epidemic; while as to their rôle in the secondary complications, there was little doubt that some members of this group, possibly even the saprophytic varieties, played a part in the complicated cases.

Summary of Conclusions.

In this brief summary of the results obtained proof of many of the statements has necessarily been omitted; these proofs will be included in the detailed paper. Here I may add some of the main conclusions. Only the pneumococcus and streptococcus groups are dealt with.

It was shown that pneumococci and aerobic streptococci could be cleared of suspicion of acting as initiating organismal causes in the epidemics; that the pathogenic organisms of these groups which were associated with the production of pulmonary complications were of a variety of types; that many of these were definitely infections from without, while with the remainder limitations of technique prevented exact proof as to their origin being brought forward; that the presence of saprophytic streptococci and of presumably saprophytic pneumococci in the lungs of many advanced cases may be explained as a late invasion of the already diseased lung, either by the patient's own mouth organisms or by the saprophytic mouth organisms passed on from another patient, while their presence in the sputum is, in many cases, due simply to the contamination of the specimen with organisms from throat and mouth.

Both from the point of view of the history of the individual case and from that of the history of the epidemic itself, one may divide influenza into three stages: a primary stage, when the unknown infecting virus, whether it be Pfeiffer's bacillus or some other organism, is apparently *acting* alone, though presumably frequently accompanied by other organisms which may or may not later be able to develop a like activity; a secondary stage, when pathogenic pneumococci and streptococci are active in producing pulmonary complications, either alone or along with other pathogenic organisms; and a tertiary stage, when saprophytic mouth organisms may invade the already diseased lung.

It was shown that the type of pulmonary complication is to a considerable extent dependent on the type of pathogenic pneumococcus or streptococcus present; that pneumococci of Types I, II and III were associated, as was to be expected, with pneumonias showing definite consolidation, as were also certain of the Group IV pneumococci; that others of the Group IV pneumococci and the haemolytic streptococci were associated with complications of the bronchopneumonic and bronchitic types.

The presence of complex infections in epidemics of this kind does not imply that the original bacterial cause at the beginning of the outbreak was not a single type of micro-organism; nor does proof that the important organismal varieties are always types foreign to normal mouths and throats—are in fact infections from without—imply that the real primary agent in such epidemics may not be a temporary susceptibility, individual or general, due to causes at present unknown.