A Clinical Lecture

ON

PYOPERICARDIUM IN CHILDREN UNDER TWELVE YEARS OF AGE.

DELIVERED AT THE HOSPITAL FOR SICK CHILDREN,
GREAT ORMOND STREET.

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I HAVE taken the subject of pyopericardium in young children because it is an interesting and also a most difficult clinical disease. Fortunately for our reputations it is not a common event, but it is a very fatal one, and for this reason we require to have a fairly clear idea of the chief conditions under which it may occur, the difficulties in its diagnosis, the striking clinical features, its course, and the present aspect of its treatment. In a general hospital a student may possibly never see a case, although by a curious coincidence one occurred in the University College Hospital wards recently which showed the very unusual symptom of pericardial friction. Even in large children's hospitals it is only an occasional event, so it is not improbable that any conception of pericarditis may be founded upon the rheumatic type, which is, I think, for those who have had a reasonable opportunity of studying it, a condition not very difficult to detect.

Pyopericardium in young children may occur in a general pyaemia, as a sequence to a local infection, complicating, for example, suppurative otitis media, osteomyelitis, or septic wounds. In such cases we must be prepared to find multiple abscesses in various regions of the body.

Pericarditis is under these circumstances a deadly complication, but, as can readily be believed, it is only one factor in a mortal illness; and, although it is well to have made the diagnosis, it can seldom be that any treatment of this one suppurating focus will save the patient. It is, of course, possible that a septic wound of the pericardium might produce a primary suppurative pericarditis, but such an occurrence is too rare to detain us now.

The first point to appreciate in the study of pyopericardium is that in young children under 12 years of age it is almost invariably associated with pulmonary disease, and that this is most commonly pneumococcal in origin.

The following is a brief description of a case:

A female, aged 2½ years, had suffered for several weeks from whooping-cough, which was complicated, as so often happens among the poor, by pneumonia. When she came under observation she was exceedingly ill, cyanosed, and dyspnoeic. Respiration was panting, and there was irregular fever. In both pleurae there were signs of effusion, and later in the illness the cardiac dullness was noted to be increased and the cardiac sounds to be faint. Death occurred rapidly. Bilateral empyemata were diagnosed, and suppurative pericarditis was suspected. At the necropsy 1½ oz. of pus were present in the pericardium, with pus in both pleurae. The pneumococcus was isolated from the effusion.

ETIOLOGY.

The condition is met with in males and females equally, and thus sex has no bearing upon its occurrence. The age incidence is, however, remarkable. I analysed 100 fatal cases from the Great Ormond Street and other records, and found 83 per cent. occurred before the fourth year of life was completed, and two-thirds between the ages of 1 and 3 years, making it, in fact, almost a disease of infancy.

Primary pyopericardium is, however, excessively rare, and I could only find one case in my series in which it was really the predominant feature, although in some others it was the only acute lesion, the lungs and pleurae showing signs of previous though extensive disease. About 60 per cent. are associated with empyema, and about 40 per cent. with pneumonia or acute pleurisy. A few are associated with acute pulmonary tuberculosis.

As predisposing causes measles, whooping-cough, and, I think, influenza may be cited.

PATHOLOGY.

The pathological conditions that are found after death in the pericardium vary considerably, and are of considerable practical importance. In some cases the signs are slight, a few flakes of fibrino-purulent exudation and a little fluid which is scarcely turbid alone bear witness to a virulent infection.

In others there is an exudation very much like the fibrino-plastic exudation in rheumatism, but softer in consistency. In others there is a creamy pus, sometimes in large amount. And, lastly, there may be remarkable thickening of the pericardium, with some inspissated pus and evidence of partial organization of the exudation.

Later in my remarks I shall point the bearing of these facts.

SYMPTOMS AND DIAGNOSIS.

In former times, when empyemata were left to Nature, or at most aspirated, this sequence of events was noticed: First an empyema, then a pleuro-pericarditis, and then suppurative pericarditis. In such cases the empyema had directly infected the pericardium. Now we seldom see this, and I do not think we can be at all sure, when suppurative pericarditis is discovered in association with empyema, that it has been a direct secondary infection, for it may occur with a left or right, or bilateral empyemata, and with pneumonia irrespective of empyema. Again, in cases with empyemata, these may be small and far away from the pericardium; and, lastly, in some cases of great virulence, death may be sufficiently rapid to prove that in such a case an early empyema and early suppurative pericarditis have arisen by the simultaneous infection of the two membranes.

It is, then, probable that in more cases, perhaps, than we may suppose the suppurative pericarditis has arisen independently of the pleural infection by a simultaneous infection or secondary blood infection rather than by direct continuity.

Clinically it is practically impossible, in the present state of our knowledge, to ascertain the time of commencement of pyopericarditis. When I add that in only 6 of the 100 cases the diagnosis of suppurative pericarditis was confidently made, and that in 2 of these pericardial friction was present, it is clear that to date its commencement is an even more difficult undertaking.

For this reason I have departed from the routine method of presenting the disease, and interpolate a few remarks upon the difficulties in diagnosis.

The first one is the usual absence of pericardial friction. It is most interesting to discover the amount of reliance we place upon that physical sign for the detection of pericarditis. Take it away, and we are all—experienced and inexperienced—blundering about in uncertainty.

The poisons leading to pyopericardium have, I imagine, a more peptonizing or digestive power than the rheumatic; and so the exudations are softer and more creamy in consistence and less harsh, hence friction is so much less frequent. It would be an error, however, to make the occurrence of pericardial friction diagnostic of rheumatic pericarditis, for it certainly occurs now and again in pyopericardium.

Another great difficulty in diagnosis is the early age of the patient. Accurate observations in small children are frequently very arduous undertakings.

Then there are almost invariably signs of severe pulmonary disease, such as pneumonia, pleurisy and empyema, which is sometimes bilateral. All these distract our attention from the heart, and, further, without any pericarditis these tend to excite the action of the heart and further confuse us. Lastly, there is the absence of endocarditis—only 1 in the 100 cases showed endocarditis; and though I do not mean to infer that endocarditis is a sign of pericarditis, its occurrence naturally helps to compel our attention to the condition of the heart.

We have, then, to diagnose pneumococcal pericarditis among other severe thoracic lesions, and we may have even more difficulties to contend with than these, for in 15 cases in the series there was also suppurative meningitis, and in 8 suppurative peritonitis.

I think, then, the difficulty of this diagnosis is reasonably explained, and with this goes also the explanation of the statement previously made, that it is practically impossible to date the duration of a fatal pyopericardium with accuracy.

CLASSIFICATION OF CASES.

To a great extent we have to judge of its course from a study of the illnesses with which it is associated, from the occurrence of some very suggestive clinical signs, and from the pathological appearances after death.

Now the illnesses in which the fatal pericarditis is discovered vary much in duration, and so for the purpose of description I have grouped them into three classes:

1. The acute, running their course in about four weeks, and including 20 per cent.
2. The subacute, lasting from four weeks to six months, and including 50 per cent.
3. The chronic, with insidious onset lasting from six months to over a year, and including 17 per cent.

I do not imply from these figures that the pericarditis lasted over these long periods, but, taken with the evidence of *post-mortem* examinations, I think it points to the probability that some cases of suppurative pericarditis may run a much longer course than we might suspect from the dangerous character of the complaint.

In the acute cases the onset of the illness is usually abrupt and pneumonic in type. In the subacute and chronic cases, cough, pleural pain, and wasting are prominent. In some cases vomiting and diarrhoea have been urgent; and lastly, in the most chronic, only gradual failure of health and strength with wasting have been recorded.

The majority of these symptoms are in no sense localizing. When, however, we come to consider those which have attracted especial attention as unusual and not quite like those we should expect in severe pulmonary disease we find the following: Extreme illness with lividity; panting respiration and excessive dyspnoea; curious irregular rises of temperature, accompanied by a rapid and feeble action of the heart; orthopnoea and syncopal attacks. The last two appeal to me as the most suggestive if in addition the pulse is strikingly rapid and excited.

When a diagnosis has been arrived at with confidence one of two events has probably occurred. Either pericardial friction has been detected, or there has supervened a large pericardial effusion. In small children both events are exceptional.

It is an interesting point that dilatation of the heart, so important a feature in rheumatic pericarditis, is not equally prominent in this form, and accordingly, when in these cases the cardiac dullness is increased, it is more suggestive of an effusion than is a similar occurrence in rheumatism.

The detection of a considerable pericardial effusion in small children is not at all easy, particularly if the heart is displaced by a simultaneous empyema, and one is very liable to go wrong from making a false start. I mean by this that when the case first comes under observation as a case of pneumonia or empyema, oblivious of the possibility of suppurative pericarditis, the exact position of the heart, its area of cardiac dullness, and the accurate character of the sounds are not thoroughly fixed in one's mind, and then there is no basis from which to work when that complication arises.

If, on the other hand, the exact condition of the heart is recorded at first, it is possible in some cases to trace every stage in the development of an effusion.

Among the most important signs are:

1. Progressive muffling of the heart sounds synchronous with enlargement of the cardiac area, together with marked percussion dullness over the pericardium and sometimes posteriorly in the interscapular region. Tubular breathing or absent breath sounds may also be noted in the interscapular region.
2. A rapid and extensive increase of the cardiac dullness upwards towards the left clavicle. I have seen a case in which there was absolute dullness reaching to the left clavicle, and the *post-mortem* examination showed that the upper lobe of the left lung was collapsed and pushed backward by the distended pericardial sac.
3. The pear-shaped outline of a distended pericardium.
4. The abrupt transition from the dullness of fluid to resonant lung tissue.
5. A wavy and diffuse pulsation to the left of the sternum.

When there is much fluid round the heart the pulse becomes rapid, small, and sometimes very irregular, and almost running in character.

Yet these cases are exceptional in the very young, and even if there is a considerable effusion, this may be missed from the absence of any pronounced physical signs, or more usually from the presence of misleading signs in the lungs.

What are the most common errors in diagnosis?

1. They are chiefly those of mistaking the half truth for the whole—suppurative pericarditis never entering one's consciousness. Thus, we may stop short at empyema, or pneumonia and empyema. If the empyema has been drained, a loculus or some retention of pus is suspected. In case of bilateral empyemata it is very human to be content with having detected both abscesses and to attempt an explanation of all the symptoms by their presence.
2. Another pitfall in diagnosis is tuberculosis. This is particularly liable to be the case in the marasmic child who has had a subacute pneumonia. The failure in recovery, the wasting and breathlessness, together with the signs in the lungs, are all ascribed to tuberculous pulmonary disease and the consequent toxæmia.

I have commented elsewhere on the great danger that there is in ascribing obscure pulmonary or other diseases in childhood to tuberculosis. It is comparable to a tendency in adult life to fall back upon syphilis or influenza under similar difficulties.

It is to be hoped that some perfected tuberculin reaction may help us in the future in cases of pyopericardium; but the more closely we consider the complexity of vital processes the more cautious we become in trusting the "pathognomonic" sign, whether clinical, bacteriological, or bio-chemical.

A very puzzling condition, of which I have only had one experience, has been the occurrence of a left empyema between the lung and pericardium, combined with a suppurative pericarditis. It will be readily understood that an empyema alone in such a situation may lead to much difficulty in diagnosis, for such an empyema may partially cover the pericardium and so mask the heart sounds, and, further, push the heart to the right, and lead one to believe that the dullness thus caused to the right of the sternum is not the result of a displaced heart but of a distended pericardium. In such cases much attention must be directed to the audibility of cardiac sounds and to the character of the pulsation to the right of the sternum. If the sounds are clear and the pulsation definite the diagnosis of a displaced heart is favoured.

There is another possibility in the diagnosis of this lesion. The fluid may be hemmed in by adhesions at the back of the pericardium and push the heart forward. In such a case the sounds are clear and the pulsation definite. I think that the acme of clinical skill has been reached when in an infant the diagnosis is made from the study of the physical signs in the interscapular region.

How are we to distinguish between an imperfectly-drained empyema and an empyema complicated by a suppurative pericarditis? Experience only too clearly shows the slender reliable evidence that is available. Each individual case must be dealt with on its own merits. There will rightly be a hesitation in diagnosing two lesions, and every attempt will be exhausted to explain the illness as the result of the empyema. If, however, this is obviously straightforward and well drained, that explanation must fail. On the other hand, if the empyema is not in an ordinary position, or the lung has not expanded, and if, further, there are no strong indications of cardiac disease, seeing the great gravity of suppurative pericarditis and the difficulty of any certain diagnosis, it would be wiser to investigate the empyema region afresh than risk an exploration of the pericardium.

The discovery of a loculus from an empyema may mean rapid and complete recovery, the exploration of a doubtful suppurative pericarditis is a much more hazardous and uncertain procedure.

The last points to which I wish to draw attention are the remarkable clinical distinctions between rheumatic and pneumococcal pericarditis. It is a point of some theoretical interest also, for this reason. If we look upon

acute rheumatism as an infection, we must be prepared to face the question whether it is a peculiar infection or an attenuated septic infection the result of diverse causes. If we have the treatment of that terrible disease at heart, we cannot be content to rest with the vague assertion that acute rheumatism is an attenuated septicaemia, and, if there is any hope in the serum and vaccine methods, it is clearly of increasing importance that we determine the real position of acute rheumatism. Now, if it is merely an attenuated septicaemia, we must surely admit it may be sometimes pneumococcal, for everything points to that infection as producing a septicaemia. Yet what do we find when, leaving theory, we come to clinical and pathological facts?

In the first place we find that pneumococcal pericarditis is at its maximum in frequency under 4 years of age, whereas rheumatic pericarditis is rare under 5.

Endocarditis is almost invariable in rheumatic pericarditis, most exceptional in the pneumococcal form. Dilatation is invariable in the rheumatic form, it is not considerable or apparently frequent in the pneumococcal.

Empyemata, pneumonia and pleurisy are the rule in the pneumococcal type. Empyemata never occur with rheumatic pericarditis.

Lastly, we have all grades of pneumococcal pericarditis—the acute and virulent haemorrhagic type, the grossly purulent, the fibrino-purulent, and the chronic adhesive.

In rheumatism also we have the virulent, the fibrino-serous, and the chronic adhesive forms.

Throughout the history of pneumococcal pericarditis we notice the tendency to pus formation and the absence of pericardial friction; throughout the rheumatic the tendency to resolution and the presence of friction.

The micrococcus obtained from the pneumococcal pericarditis is a lanceolate, encapsuled, delicate diplococcus; that obtained from the rheumatic form is smaller, not encapsuled, and resistant in its growth, though prone to lose its virulence.

Rheumatic arthritis is exceedingly frequent with rheumatic pericarditis; pneumococcal arthritis, though well recognized, is exceptional.

If there is any meaning in the term "disease," then, I think, we have two different processes here, and if that is so, then we must admit that if acute rheumatism is an attenuated septicaemia, it is not an attenuated pneumococcal septicaemia.

Now let us turn to the rare cases of other forms of purulent pericarditis in childhood, those secondary to the *Staphylococcus aureus* in osteomyelitis for example. In these we find multiple abscesses.

Again, in those septic infections we may find an abscess in one place and clear fluid in other places. For example, pus in a knee-joint and clear fluid in one pleura. Surely, then, in rheumatism when we find clear fluid in one place, we ought to find now and again, if it is an attenuated septic infection, abscesses elsewhere. Such an occurrence is beside my experience, and I hold that acute rheumatism is as special a disease as is a staphylococcal or pneumococcal disease.

PROGNOSIS AND TREATMENT.

The prognosis of suppurative pericarditis in small children is exceedingly grave, and one cannot help feeling that unless a serum can be obtained which will combat these general pneumococcal infections an improvement in our powers of diagnosis will not greatly help our methods of treatment.

Surgical intervention at present offers the best chance of success, and there are a certain number of cases on record of recovery. The best results are to be expected in children older than those with which this lecture has been chiefly concerned, and in which there is a considerable effusion of liquid pus. I feel sure that in many of the cases in infants, even if the pericardium is opened, the relief afforded would be but comparatively slight, on account of the difficulty in the drainage of the tenacious exudation and the multiple associated lesions. Yet pathological investigation amply shows that there is sometimes a remarkable vitality exhibited by children suffering from this form of pericarditis, and that great natural efforts are made for recovery; this encourages us to persevere in aiming at an early diagnosis.

Paracentesis of the pericardium will of course be required

in these cases; and if there is sufficient evidence of a purulent effusion, it is better to proceed at once to open the pericardium after the paracentesis rather than to first explore and then operate at a later date.

There has been a good deal of difference of opinion expressed as to the best site for exploration. Dr. Samuel West has recently advocated the left lower margin of the cardiac dullness—that is, in the region of the impulse.

In uncomplicated cases of pericardial effusions few, I imagine, would doubt the wisdom of this ruling. In the condition under consideration, however, we must be prepared to adapt ourselves to circumstances, and choose the fourth or fifth intercostal space close to the sternum, or the left costo-xiphoid angle, passing the needle inward and upward, behind the lower end of the sternum.

There is no doubt that a needle has been passed into the heart on occasions, and as a rule without ill effects, nevertheless it is an anxious occurrence.

Lastly, inflammatory changes in the anterior mediastinum and great thickening of the pericardium itself make it sometimes difficult to tell when the pericardial cavity has been opened, and it is not surprising that now and again a pericardial cavity has been thought to be opened and a drainage tube introduced into the mediastinal anterior tissues.

SOME CONSIDERATIONS REGARDING PNEUMONIA IN CHILDREN.*

DRAWN FROM AN ANALYSIS OF 500 CASES.

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THAT pneumonia in children is a very interesting, a very common, and a very fatal disease will, I venture to think, be generally admitted. During the period I have had charge of a ward in the Edinburgh Sick Children's Hospital 3,300 cases of all varieties of disease have been treated, and of these over 500, or nearly one-sixth, have been cases of pneumonia, clearly proving, if proof were needed, how largely it bulks amongst the serious diseases of the children of the poor, necessitating treatment in hospital. Much has been written on the subject, and it has been so thoroughly discussed in all its phases, and the field so carefully gleaned, that I fear my contribution will not add very materially to what is already common knowledge regarding the disease. Still, I consider that when one has had a large experience it is desirable that that experience should be critically digested and summarized, and it is more with the object of ascertaining what I myself have learned than with the view of contributing any new facts of material importance that I have ventured to write this paper. I was confronted at the threshold of my inquiry with the consideration of the method to be adopted in the grouping and classification of the cases—a question bristling with many difficulties and pitfalls, whether we approach it from a bacteriological, a clinical, or a pathological standpoint.

Broadly speaking, pneumonia in children may be divided into cases of bronchopneumonia and those of the lobar variety; but owing to the many anatomical and physiological differences in the respiratory system of the child compared with the adult, the discrimination between the two conditions is frequently puzzling and sometimes impossible, and the difficulties assume wider proportions as one's knowledge of the subject increases. Doubtless in the majority of cases the process is absolutely typical of one or other variety, but there are cases, occupying a position on the borderland, which are very difficult to place, and others again of a mixed variety which seem to defy classification, whether we investigate them clinically or pathologically.

Clinically we recognize a case as belonging to the lobar type of pneumonia by its abrupt onset, its lobar distribution, its continuous high temperature, terminating by crisis, and its speedy resolution; while we characterize another as bronchopneumonia by its slower onset frequently following bronchitis, by the marked remissions of temperature which terminates by lysis, and after a prolonged

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