FIBRINOUS DEPOSITS WITHIN THE HEART IN DIPHTHERIA AND IN SOME OTHER DISEASES OF CHILDREN.

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My object is to show, by a short summary, in what diseases among children fibrine is most liable to become deposited within the heart's cavities, and under what conditions. My observations have reference only to what are usually recognised as *ante-mortem* clots.

The following data are derived from notes of about 200 *post-mortem* examinations made at the Children's Hospital, Great Ormond Street, during two years and a half. In fourteen cases of phthisis, four showed considerable fibrinous deposits, of which two were cystiform; whilst in the remaining ten cases fibrine was either absent or present in small amount. Of the cases of tuberculosis (including tubercular meningitis, but excluding phthisis), three only out of a total of thirty-three presented fibrinous deposits of any size. Very marked fatty changes in the muscular walls were present in two of these three cases. Of twenty-three *post-mortem* examinations on diphtheria-cases, more than one-half showed marked fibrinous deposits (probably all *ante-mortem*). They were chiefly in the right cavities, though to some extent the left cavities also suffered in most instances. Moreover, in seven out of the twenty-three the right cavities were nearly filled with firm decolorised fibrine. Fibrinous deposits also existed in the remainder of the diphtheria-cases, but they were of small size, though probably *ante-mortem*, except in one case, where the child died with symptoms suspicious of diphtheritic paralysis commencing. This last case was the only one in which a distinct *bruit* was present during life.

A few other cases presented well-marked fibrinous deposits, namely, two of morbus coxae, where death resulted a few days after amputation at the hip-joint; one case of osteotomy of the femora, death resulting within thirty-six hours, accompanied by symptoms of shock; one case of tracheotomy for papilloma of larynx, followed by pneumonia and death in about three days; two cases of chronic and one of acute nephritis (non-scarlatinal); two out of seven cases of diphtheritic paralysis; and one case each of simple purulent meningitis, otitis (with pneumonia), pyelo-nephritis, and diabetes mellitus. There were also two cases of morbus cordis, with old fibrinous deposit; and one of adherent pericardium, with softening fibrinous deposit in the right auricular appendix, giving rise to pneumonia.

Microscopic examinations of seventeen of the twenty-three cases of diphtheria proved that the muscular fibres were undergoing a fatty change, more or less general. In many cases it was accompanied by atheromatous patches and scroll-like buffy markings about the root of the aorta and bases of the mitral and aortic cusps. But these appearances were observed in various other diseases.

Hypostatic pneumonia or congestion was associated, in most cases, with deposits of fibrine within the heart's cavities of both sides, though the right cavities in more than half the number of cases contained a much larger amount than the left. Collapse and emphysema were present in nearly every case of diphtheria.

In the only three cases in which fibrine was deposited in the right cavities alone, there was collapse of the bases and posterior parts of the lungs, pneumonia and congestion being absent in them—namely, two cases of diphtheria and one of amputation at the hip-joint for morbus coxae. This circumstance is strongly in favour of the view that extensive fibrinous deposits in the right cavities lead to collapse of the lungs; but I think the evidence *post-mortem* also points to pneumonia and congestion being secondary to collapse thus produced. The deposits in the left cavities were, as I remarked before, much smaller usually than those in the right, but they were so disposed usually as to block the mitral orifice, whilst in the right cavities they appear first to form in parts removed from the direct current, such as the auricular appendix, which is often distended.

The course of events would seem to me to be briefly as follows: 1. Deposition of fibrine in some part of the right cavities removed from the direct blood-stream. 2. Partial blocking of the pulmonary artery by extension of the deposit. 3. Collapse of the remote part of the lungs, with emphysema of the anterior parts. 4. Feebleness of the circulation in the left cavities induced thereby. 5. The last condition leads to separation of threads of fibrine in the left auricle, which become entangled with the chordæ tendinæ of the mitral valve. 6. This deposit, once begun, tends to spread, so that processes extend up the aorta on the one side, and into the left auricle on the other.

7. From the deposit in the left auricle secondary prolongations are extended into the auricular appendix and into the branches of the pulmonary veins, where they have rounded ends. 8. Hence the hindrance to the return of blood from the lungs and the hypostatic congestion of those parts previously collapsed.

One can understand how a slight obstruction at the mitral orifice might, in a short space of time, induce considerable congestion of the lungs if we reason as follows: Supposing one minim of blood fail to pass the mitral orifice at each contraction of the left auricle, then if the heart's contractions equal 120 per minute, there will be two drachms of blood thrown back upon the pulmonary circulation per minute, or fifteen fluid ounces per hour.

The circulation in the remoter parts of the lungs must necessarily be very slow, for at least three reasons: 1, the small amount of blood entering the lungs goes to be distributed over a large area; 2, the obstruction which exists to the return of the blood by the pulmonary veins; 3, the weakness of the left auricle consequent on its dilatation from mitral obstruction.

The symptoms of fibrinous deposit are not easy to differentiate from those due to fatty heart, so frequently present.

With regard to treatment, one should be careful not to adopt too active measures in those diseases in which fibrine is liable to become deposited, notably in diphtheria and nephritis. Cardiac depressants, such as antimony in the form of an emetic, are dangerous. Too active purgation and diaphoresis, by increasing the relative amount of fibrine in the blood, should be avoided in the treatment of nephritis. Any depressing nervous influence should, of course, be guarded against.

I have thought it possible that ether may have had some influence in bringing about the separation of fibrine, as it was administered, contrary to the usual custom, in the four operation cases quoted above.

My thanks are due to the members of the staff of the Children's Hospital for allowing me to make use of the cases, and also to Dr. Penrose, the present registrar and pathologist, for helping me to continue my investigations.

PLASTERS IN THE TREATMENT OF SKIN- DISEASES.

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Of late years one of the most generally used methods of local treatment of skin-diseases, especially eczema, has consisted in enveloping the affected portions of the skin in pieces of soft material such as muslin, thickly spread with diachylon ointment, over which soft rags are placed to prevent the melted ointment soaking through; the whole dressing being held in position by a firmly applied bandage. Previous to the application of the ointment as described, the affected part may have been treated by free use of soft soap or other stimulating measures. This dressing was to be renewed morning and evening, and it was a point of the first importance that the ointment should be thickly spread, and that the pieces of rag covered with it should be applied evenly and in close contact with the skin, and that there should be no delay between the use of the soap, if employed, and the subsequent application of the ointment. I need scarcely call to mind that diachylon ointment is made by boiling litharge with oil in the presence of water.

When I first employed this method of treatment, I encountered considerable difficulties. In the first place it was not easy to obtain the ointment uniformly made. Sometimes it was deficient in cohesion and apt to crumble, or it was so hard that it would not spread unless partially melted, or it was so soft that with the heat of the body it rapidly liquefied and soaked away, often saturating the clothing of the patient, and penetrating through the rags and bandages which had been placed over it. Again, patients and their attendants did not like the troubling of spreading the ointment, and they were apt to put it on either too thickly or too thinly, since it requires some mechanical skill to spread ointment uniformly.

It seemed to me that many if not all these difficulties would be removed, and some additional advantages gained, if it were possible to obtain the ointment ready spread on some suitable substance and of such a consistence that it would not liquefy at the temperature of the body, and at the same time possess some adhesive qualities. After experiments in this direction extending over some years, I succeeded in obtaining a plaster of the same constituents as diachylon-ointment, and this plaster fulfilled the essential conditions of being suffi-