affected, and did not become implicated afterwards, excepting by direct structural continuity with the focus of the disease.

Secondly, the absence of any tendency towards *suppuration*, sloughing, or gangrene was remarkable. There was no loss of tissue up to the time of death; vesication had occurred on the surface of the growth, but the part was still covered with epithelium. There was venous turgescence in all the infiltrated tissues, as indicated by the dark colour which they assumed; but microscopic examination did not indicate any marked vascularity, such as would have been the case if the development of new vessels had progressed *pari passu* with the growth of the disease. The original tissues became more and more swollen by a process of infiltration; but the new product did not manifest any tendency towards permanent organisation, nor did it manifest any retrograde tendencies.

Thirdly, the combination of a very rapidly growing lympho-sarcomatous growth with a condition of *leukemia* is interesting. What is the relation of the two? That the one is not indispensable to the existence of the other, is shown by the numerous reported cases where lymphoid growths have been found, and the blood has presented no abnormality. The history of this case can leave no room for doubt as to which was the primary condition: the spleen was enlarged and leukemia existed when the patient first came under observation, and at a time when the morbid growth was small. Growths of this kind must result from either—

a. Proliferation of the connective tissue-corpuscles.

b. Hyperplasia of adenoid tissue.

c. Development from the leucocytes of the blood which have escaped from the vessels, have retained their vitality, and have continued to reproduce similar cells in the part where they are found. It seems probable that, just as pus results from the escape of the colourless blood-corpuscles from the vessels, so a lymphoid growth may result from a similar cause by a slight modification in the nutritive changes which the cells undergo. Presuming, then, that it is possible for the tissues to become infiltrated with leucocytes from the blood, a morbid growth of lymphoid origin may be a direct, though not a necessary, result of leucocythæmia. The commencement of the process may be accidental; but, having once commenced, the leukæmic condition is likely to cause a very rapid growth in any part once affected. A lymphoid growth is likely, therefore, to be of short duration when associated with leucocythæmia, whereas, in other cases, the process may continue for several years. In the case of Pope, death took place in seven months from the first appearance of the morbid growth; but, in the clinically and histologically very similar case reported by Dr. Beigel as "*lepra tuberosa*", and in which the blood was in a normal condition, the patient lived for three years after the morbid growth first commenced.

The cause of death was asthenia, in consequence of frequently recurring hæmorrhage. The blood was thin, pale, and had little tendency to coagulate. If death had not resulted from this cause, it would have been impossible for him to survive the rapid spread of the morbid infiltration of his tissues which was going on; breathing and swallowing were becoming impaired from the infiltration of the tissues in the neck.

It would be interesting, in future cases of this kind, to note the appearance of the colourless corpuscles, particularly with regard to the activity or otherwise of their amœboid movements, also to compare the size of the leucocytes within the vessels with the cells which are found to infiltrate the diseased tissues.

RIGOR MORTIS IN AN INFANT AT BIRTH.

By J. ASHBURTON THOMPSON,

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DR. GRIGG has added two important observations to his first description of the above case; that the jaw again became fixed after extension had been made and the stiffness thereby removed, and that the legs were not so firmly set as the upper part of the body. These two facts, considered in relation to each other and the well known phenomena of *rigor mortis*, are all important, and may be admitted to establish the presence of that condition in the case described. It is much to be regretted that they were not included in the original report of it, which even now lacks the scarcely less important information as to time of examination, and of the return of stiffness to the forcibly extended muscles. With these additional facts at command, I am not surprised that Dr. Grigg declines to discuss his case with me.

Still, I must confess myself unable to share the conviction with which Dr. Grigg asserts his view; and it is possible that others of the Associates may feel sufficiently interested in the subject to make it worth

while to point out the chief difficulties to be met with in maintaining it. In the first place, bearing in mind that rigidity invariably occurs in the bodies of individuals, whether they die within a few hours of birth or at the most advanced age, the extreme rarity with which it has been observed in the fœtus at birth is enough to raise a suspicion that the latter is of another kind. Those who wish to maintain its identity with the former, must account for its rareness; they must show that the one or two cases in which it has been detected differ in some essential particular from all the other cases in which it has not occurred; for, since the *rigor mortis* is the result of a physical property of a constituent of the muscular mass, it must always occur under similar circumstances. This difference cannot exist in the time of delivery as compared with the time of death; because all those obstetricians of experience who have failed to note rigidity of the fœtus have had the opportunity of inspecting bodies at every period, from a few minutes to a few weeks after death. Secondly, granting the liability of the still-born to cadaveric rigidity, on what ground is it to be anticipated that it may show itself earlier in those than in other bodies? Still, bearing in mind that it is the result of a physical change, must not the same conditions always modify it in the same way? Dr. Grigg himself supposes this fœtus to have lived within an hour and a half of birth, and there is every reason to regard this as the extreme period at which death may have occurred; for the hæmorrhage—or, in other words, the partial separation of the placenta—only began at that time. It died, in fact, of asphyxia; and that is a tedious mode of death *in utero*. What conditions are here to favour the speedy occurrence of cadaveric rigidity? Not the mode of death—that is one cause of its delay in the individual; nor the temperature to which the body was exposed, for a high temperature has no influence over the time at which the rigidity occurs.* So the precipitate occurrence of rigidity under circumstances unfavourable to the development of true *rigor mortis* affords a further ground for suspicion that it is rigidity of another kind.

I say "suspicion," because I ventured an opinion on grounds which I still think not unreasonable; and to better reason such an opinion will always yield. While, therefore, I now admit willingly that Dr. Grigg did observe *rigor mortis* in his case, I do not yet see clearly that it occurred *in utero*. Cadaveric spasm is fairly described by many writers—by Taylor among others; and it is a peculiarity of it worth noting in this connection that, whether the result of a final act of volition or of convulsive action, it persists until the supervention of cadaveric rigidity. Not, therefore, because the latter state was observed by Dr. Grigg under these apparently so adverse circumstances, was it the only state which had affected this fœtus.

ON PLEURITIC EFFUSION.

By JOHN RICHARD WARDELL, M.D., F.R.C.P.,
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[Continued from p. 615 of last number.]

THE first means to be had recourse to in order to ascertain the presence or otherwise of thoracic effusion is *inspection*. When the condition now considered obtains, careful observation notices less mobility in the affected side. There is generally, at the lower third, a greater or less degree of bulging, and the surface looks smooth and rounded. The intercostal spaces are less defined, and the intercostal muscles are often absolutely motionless. When the liquid has for a long time been effused, the intercostal muscles, from having been continuously stretched, lose their contractile power, become paralysed, flat, and then even tend to protrude. Albertini supposed, and Stokes believed, that such state denoted pus. A decomposing and debased fluid in contact with the wall of the chest, it has been thought, is sufficient to produce such flabby and flattened condition of these muscles. When the effusion is considerable, there is, as the result of left pleurisy, visible displacement of the heart; the apex-beat may be seen far away from its normal position, at or beyond the right edge of the sternum, or below the ensiform cartilage in the epigastric region. I saw one marked example of such dislocation of the heart not long ago, where the apex-beat was near the right nipple. Murchison says there is greater danger of fatal syncope when the effusion is in the left side. My own experience is certainly that there is more frequently irregular and feeble heart's action in left-sided effusion. Increase of motion in the sound side is often apparent. The patient, when asked, will tell you he feels the air go down deeper into the one side of his chest than the other. The side of effusion not

* See Taylor's *Medical Jurisprudence*, 1865, p. 14; see also, at p. 13, the account of experiments by Sommer, who placed two children, born asphyxiated and still, in a tepid bath of from 90 deg. to 99 deg. *Rigor mortis* did not set in until between three and four hours.