occurred externally. Therefore in this case, if in any, one would expect to find the placental tissue as vital and unaltered as it could be five months after its function and foetal circulation ceased. The drawing shows a villus from this placenta which can hardly be described, so complete is its disintegration. Compressed by organised blood clot and separated from its fellows, it is, indeed, but a wreck of a villus.

I believe that after the foetal circulation has ceased no constructive process continues in the foetal elements of the placenta. I find no trace of division in either epiblastic or mesoblastic foetal cells of a placenta whose embryo has been dead any length of time; while karyomitotic figures are common in growing villi hardened immediately after the death of the ovum. The foetal tissues may increase in size, but only through the swelling of degenerative processes. The maternal blood occupying the intervillous spaces clots and becomes organised, rendering firm and tough the originally spongy placenta. Indeed, the villi in a placental relic play a part comparable to that of a sponge graft, and afford a structure round which new connective tissue is formed, just like the fibres of a dead sponge would do. Young villi degenerate more quickly than mature villi, but their epithelium is more distinct and perfect than that of old villi. Therefore in determining the length of time a placenta has been retained, one should judge by the degree of organisation that has occurred in the blood clot between the villi, rather than by the condition of the villi themselves. Now the time occupied by the transformation of blood clot into fibrous tissue is fairly well known, so that there should be no difficulty in deciding whether any placenta has been retained for days, weeks, or months; though after fibrous tissue is once formed it doubtless undergoes very little further alteration.

The placenta has been supposed to grow after the death of the foetus in ectopic gestation. But recent work has shown that all progressive changes that occur are in the blood clot, and that none but degenerative processes occur in the villi.¹

In the present discussion on deciduoma malignum it is urged by some that foetal epiblast lives and grows in the maternal tissues, while others hold that the origin of the new growths in question is decidual cells, which are simply connective tissue cells of the uterine mucosa. It is, however, far from proved that foetal cells play any part in the matter, so that deciduoma malignum at present affords no argument against the above conclusions.

A CASE OF FATAL PERNICIOUS ANÆMIA IN A GIRL AGED 20.

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THE following case of this interesting disease appears to be worthy of record, owing to the early age, the sudden onset after mental strain and worry, and the fatal course:

M. A., aged 20, single, a school teacher, had always been strong and in robust health, and gave no history of any recent illness or blood loss.

At the end of December, 1895, she was noticed to be getting pale. This was attributed to mental strain and worry she had recently had. As she got worse, I saw her on January 29th, 1896. She looked very ill, complained of vomiting, dyspnoea, anorexia, dimness of vision, severe headache, extreme debility, and fainting. There was intense pallor, the skin being of a transparent waxen hue, with a lemon yellow tint. The mucous membranes of the lips and eyelids were quite colourless. There were no submucous or retinal hæmorrhages, but there was pallor of retinae and optic discs. There was no emaciation; no cedema of lower extremities; no osseous pains; no hepatic or splenic enlargement; no bleeding from any passage. The area of cardiac dulness was normal. A loud systolic murmur was heard at the apex and base, and a continuous venous hum in the vessels of the neck. No evidence whatever of any thoracic or abdominal disease, or of intestinal parasites was afforded. The patient was slightly constipated; menstruation was normal; the

tongue clean, but pale; the urine clear, of a dark colour, specific gravity 1011; it contained no albumen nor sugar, but gave a deposit of bright urates. The blood was pale and watery; the discs greatly reduced, and did not form rouleaux, but varied in size and shape, some large, others distorted, a few small and deeply coloured; granular masses were present. I regret I had not the appliances for estimating the hæmoglobin or hæmocytes.

The usual line of treatment was adopted—arsenic, iron, salol, etc.—with marked improvement for a few days. Then ensued an aggravation of all the symptoms, incessant vomiting of greenish fluid without blood. There was no epigastric pain. Suffice it to say that in spite of every care she went from bad to worse; the temperature, normal at first, now varied from 100° to 101° F., with slight delirium and cadaveric odour of the breath. The heart's action was more feeble, so that she became comatose and died on February 14th. No *post-mortem* examination was allowed. The home surroundings were good, and the other members of the family were strong and healthy.

MEMORANDA: MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, Etc.

CHRONIC HYDROCEPHALUS, WITH DEATH IN THE TWENTY-THIRD YEAR.

A CASE of hydrocephalus, lasting from infancy well into manhood, is sufficiently rare to deserve recording. The following is such a case:

B., a male, died on April 13th, 1896, aged 22 years and 1 week. He is said to have had "water in the head" when a year old, but was supposed to have recovered from this, though the head remained large. He grew up a bright, intelligent child till his 7th year, when the head symptoms recurred and rapidly developed. Very soon he became blind, and a few years afterwards his speech also was lost, though hearing remained unaffected till death. During the course of the disease he was subject to infrequent and very slight convulsions.

At death the measurements of the head were: Circumference at the level of the occipital protuberance 26 inches (this had been the measurement for some years); length over the surface from root of nose to occipital protuberance, 16 inches; from mastoid process to mastoid over the vertex, 19 inches. The skull was completely ossified, the face infantile in size and appearance, the teeth decayed.

The spine was sharply curved, the convexity to the left; the left thorax flattened, the right prominent and reaching down over the pelvis. All the joints were strongly flexed, the feet extended; the head of the left femur lay upon the ilium. The body and limbs were shrunken to the skeleton, and of boardlike rigidity. Only the shoulder and fingers of the right side were movable, voluntary movement being accompanied with tremors. A straight line from vertex to great toe measured 40 inches. The genitals were well developed, and covered with hair. For fifteen years he had been nursed in the arms like an infant, constantly whining and crying out. Death was preceded by coma of about four hours' duration.

Both parents are alive and healthy; two sisters are healthy young women. The mother had one stillbirth. This was all her family.

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JOHN LINDSAY, M.A., M.B.

CEREBRAL MENINGITIS FOLLOWING INFLUENZA. WHEN I first saw the patient, a boy aged 14½, he presented the following symptoms: Severe frontal headache, excessive lachrymation, together with pain on pressing over the eyeballs, aching pain in the lumbar region and legs, furred tongue, anorexia, somewhat scanty and high-coloured urine, and constipation. The temperature was 104.2° F. and the pulse 117.

From what the boy's mother told me, I came to the conclusion that the illness had commenced with a rigor and some

¹ See D. Berry Hart, *American Journ. Obstet.*, vol. xxv, 1892; and Webster's *Ectopic Pregnancy*, Edin., 1895, p. 101; also Alban Doran on Placental Polypus, vol. xxxvii, *Trans. Obs. Soc.*