slightly enlarged. With a ring forceps, fragments of apparently typical hydatidiform mole were removed, and then a curettage was performed. Microscopy revealed considerably more trophoblastic activity than in the original specimen, with variations in nuclear size and density, and occasional mitoses. No villi were seen in the curettings. Some bleeding persisted, and one week later the Aschheim–Zondek reaction was still positive, though remaining negative in dilution. *X*-ray examination of the chest showed no abnormal shadows.

In view of the recurrence of the mole with atypical trophoblastic activity, total hysterectomy with bilateral salpingo-oophorectomy was performed on July 21—that is, 91 days after evacuation of the mole. Both ovaries contained small luteinized and atretic follicles; in the right ovary there was also a recently ruptured small lutein cyst. The uterus was bulky and contained a haemorrhagic tumour approximately ½ in. (1.3 cm.) in diameter near the right cornu. The growth extended from the endometrium through approximately half the thickness of the uterine wall. Microscopically the growth was a typical chorionepithelioma with malignant trophoblastic elements invading the myometrium. Sections of the ovaries and uterine tubes revealed no secondary deposits.

The patient's progress was uneventful; at examinations 7 and 16 weeks after operation there was no clinical evidence of recurrence, and on each occasion the Hogben test was negative. Subsequent direct follow-up has not been possible, as the patient went abroad, but a letter reports that she was well in January, 1952.

COMMENT

In 1939 Brews observed that as a method of absolute diagnosis of active chorionepithelioma quantitative hormone assays had failed to realize initial expectations. By that time cases had been reported in which the biological tests for urinary gonadotrophin had remained negative in the confirmed presence of a tumour. Cuscaden and Bettinger (1940), in describing a case in which certain features closely resembled that now reported, refer to the possible fallacies of the biological tests and maintain an attitude of great caution in accepting reports of cases of chorionepithelioma without a positive Aschheim–Zondek reaction. The possibility of a negative pregnancy test in the presence of chorionepithelioma is mentioned by Baird (1950) and Browne (1950), while Sutherland (1951) has collected 19 reports of such cases from the literature, and Richli (1950) describes another in detail.

In trying to explain the negative and only weakly positive reactions the factors to be considered include haemorrhage, necrosis, thrombosis, and fibrous reaction; all of these may separate the tumour from healthy maternal tissue and so limit the absorption of gonadotrophic hormone. Browne (1950) suggests that the degree of anaplasia present in the cytotrophoblast may determine the strength of the Aschheim–Zondek reaction. Sutherland (1951) attempts to relate the very weakly positive reaction to an almost complete absence of syncytium, although he admits that this is not in accordance with the experimental evidence of Jones, Gey, and Gey (1943), who found that the Langhans cells were apparently responsible for the production of chorionic gonadotrophin.

The cases described by Browne (1930), Ehrhardt and Bureck (1939), Scott (1939), and Brews (1950) illustrate the dangers of waiting to establish a firm histological diagnosis in the presence of a persistently positive pregnancy reaction after evacuation of a hydatidiform mole. When the reaction has become negative soon after expulsion of the mole, and later in the absence of a new pregnancy it again becomes positive, radical treatment is indicated. This view, however, is not shared by Siegler, Ravid, and Tobin (1950), who, in presenting a review of 12 cases of chorionepithelioma and acknowledging the fallibility of the biological tests, advise that, "so long as the titre is decreasing or at least remaining constant, watchful conservatism may still be applied." Such a recommendation appears highly dangerous, bearing in mind the fatal cases of Brews and of Ehrhardt and Bureck, referred to above.

Shively (1950) assesses the reliance to be placed upon quantitative urinary gonadotrophin determinations by saying that, because of the variations encountered, they should be used only as confirmatory evidence in the diagnosis of these neoplasms and should not be regarded as necessary diagnostic criteria.

Publication of this case report has been approved by the Director-General, Army Medical Services.

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Traumatic Encephalopathy from Boxing

The risk of serious intracranial damage occurring in boxing is well known, and the following account may be of interest to those concerned with the welfare and medical examination of boxers.

CASE REPORT

A miner aged 26 had boxed on numerous occasions since the age of 15 and as a professional from the age of 20. An ample history, obtained from various sources, failed to reveal evidence of previous mental disorder or any other abnormality, except a minor degree of mental dullness indicated by his school work and Army records. His I.Q. was reported to have been 90 on the Stanford–Binet scale (Form L) when he was tested at some unspecified time before leaving school at the age of 14.

Two years ago, after becoming cruiser-weight champion of his area, slight changes in character became apparent, shown in the form of egocentricity and of monopolizing the conversation with accounts of the various opponents whom he had knocked out. Soon he was knocked out himself after only 18 seconds in the first round, and three months later, in an encounter with a boxer thought by all to be inferior, he took so much punishment that the fight was stopped in the fourth round. His friends were now finding him more quarrelsome and garrulous; he often complained of headaches, and he expressed mild paranoid ideas about his wife which eventually resulted in his admission to a mental hospital a year ago, after he had made threats to kill her with the carving-knife.

He was then found to be slightly confused, was alternately excited or unduly reticent, and had also a slight spatial and temporal disorientation, misidentification of persons around him, confabulation with an associated patchy memory defect for events of the preceding four months, and fleeting olfactory hallucinations. Abnormal physical signs were slurred speech, absent abdominal reflexes on the left side, and a slightly ataxic gait of cerebellar type. The cerebrospinal fluid and an *x*-ray film of the skull were normal.

Gradual improvement took place over a period of six months with sedation and rehabilitation, and he was then

discharged from hospital and obtained work as an unskilled labourer. Residual signs then were amnesia for events over a period of one to two weeks at the time of his last two fights, and what appeared to be a reduction in intellectual capacity. His I.Q. on the Stanford–Binet scale (Form L) was 65. On Raven's progressive matrices and on Kohs's blocks test he was at the 5 percentile and on the Mill Hill vocabulary scale at the 10 percentile level. There were now no abnormal signs neurologically. The E.E.G. showed reduced activity in all leads.

COMMENT

The difficulties in the diagnosis of this type of case have been summarized by Bremner (1950) and one has to admit that the difference in the I.Q. may be wholly or partly due to other factors, and also that further improvement might have been found had examination at a later date been possible.

One hesitates to go as far as Carroll (1936), who believes that every knockout is likely to cause irreparable damage, although the boxer intellectually below average is more likely to show persistent clinical abnormality. A further difficulty is the fact, as observed by Jokl (1941) and others, that severe cerebral injury may occur without anything unusual being observed by spectators at the time, and medical elimination of a contestant in such circumstances may be strongly opposed. Romino (1949) puts forward various suggestions to make boxing safer, such as equality of matching, medical records of each fight, and no fights for three months after being knocked out, to which might be added the use of psychometric tests at intervals and the search for minimal neurological abnormality.

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Comparison of Treatment of Haematemesis by Drip Transfusion of Stored Blood and by Direct Transfusion

The aim of treatment of haematemesis is to produce haemostasis; where a large artery is eroded this can be achieved only by surgery, but haemorrhage from small arteries and veins is stopped by the natural process of vasoconstriction and thrombosis. That repeated transfusions of stored citrated blood may inhibit this natural process, whereas small direct transfusions aid vasoconstriction and thrombosis, is suggested by the histories of the following three cases.

CASE 1

A married woman aged 52 was admitted to hospital on June 24, 1952. She was well until 12 hours previously, when there was a sudden onset of diarrhoea followed by melaena.

On admission her general condition was good, the haemoglobin being 73%. Nine hours later she passed a tarry stool and became pale. Systolic blood pressure was 70 mm. Hg and haemoglobin 44%. A drip transfusion of stored blood was begun, and 3 bottles (each 500 ml.) were given over a period of four hours, followed by 1 litre of normal saline, then 2 bottles of blood at a slower rate. Three hours after the transfusion had finished, haematemesis recurred and the patient became shocked. A surgeon advised medical treatment. A drip transfusion was begun, being continued for two days, during which period further repeated attacks of haematemesis and melaena occurred and 7 bottles of blood and 2 litres of saline were given.

Three days after admission the patient's condition was so low that surgical operation was hazardous, and it was decided to try direct transfusions. Three were given at intervals of approximately four hours, and her condition improved. Twenty-four hours later haematemesis recurred and a drip transfusion of 6 bottles of blood and 1 litre of saline were administered. This transfusion continued over 35 hours, during which period melaena and haematemesis occurred repeatedly.

Her condition was again desperate, and two further direct transfusions were given at intervals of four hours. Although several melaena stools were passed there was no further evidence of severe haemorrhage.

On July 1 the haemoglobin was 48%; two direct transfusions were given to raise the haemoglobin, which on July 15 reached 70%. At this stage a generalized erythematous rash developed, labelled the Stevens-Johnson syndrome, which delayed her discharge from hospital until August 9. *X*-ray examination of the stomach and duodenum was reported as N.A.D. All told, the patient was given 19 bottles of stored blood and 6 direct transfusions.

CASE 2

A man aged 36 was admitted to hospital on December 27, 1951, suffering from haematemesis and melaena.

On admission he was in a shocked state, and a drip transfusion was begun; 4 bottles of bank blood were given, and his condition improved. Within nine hours further haematemesis occurred, and the drip transfusion was recommenced. During the next five days the transfusion was continued and 29 bottles of blood and 9 litres of glucose-saline were administered. During this period the patient repeatedly vomited blood and passed melaena stools.

On January 3, 1952, he was seen by me. His general condition was very low; systolic blood pressure was 90 mm. Hg and haemoglobin 38%. During a period of two days he was given five direct blood transfusions. His condition improved, and no further bleeding occurred. *X*-ray examination showed no abnormality of the stomach or duodenum. He had been given 34 bottles of blood, 9 litres of glucose-saline, and five direct transfusions.

CASE 3

A man aged 28 was admitted to hospital on March 10, 1951. He was suffering from haematemesis and melaena, which occurred on the day of admission.

On examination his skin was pale and he was in a shocked condition. He was immediately given a transfusion of 4 bottles of stored blood by the drip method. The next day he vomited blood and the haemoglobin was 52%. Another drip transfusion was started, and during a period of 17 hours 6 bottles of stored blood were given; but during the transfusion further haemorrhages occurred and on March 13 the haemoglobin was 42%. Another 3 bottles of stored blood were transfused. On March 14 there was severe melaena, and the haemoglobin was 40%. Two bottles of bank blood were given, but again there was haematemesis.

The patient was seen by me at this stage. Three direct transfusions, each of 500 ml., were given on the 14th, 15th, and 16th. Subsequently there was no further bleeding. On March 24 the haemoglobin was 83%. *X*-ray examination on March 27 showed evidence of duodenal ulceration. He had been given 15 bottles of bank blood and three direct transfusions.

COMMENT

In these three cases severe haemorrhage occurred during the prolonged transfusion of large quantities of stored blood and ceased when fresh blood was transfused at short intervals by the direct method. Further investigation, such as a planned research project, would be necessary to establish the validity of this observation as indicating an untoward effect of stored blood transfusions when given to excess, and the purpose of recording these cases is to suggest that such an investigation would be worth while.

Recent work on blood coagulation and thrombosis emphasizes the importance of blood platelets in the natural