intermission from the onset of the attack, she did not think it had differed in character after the end of the first hour. At no time had she any feeling of air hunger.

She lay in a dazed, apathetic condition, and was markedly pale, but of a fawny white rather than the ashy grey colour of secondary anaemia. The pulse was 108 and the temperature 98° F. The abdomen was greatly distended and gave a sensation of hard tenseness. This distension was chiefly localized in the upper segment and passed under the costal margins. No sounds could be heard on auscultation, nor could any movements be recognized on inspection or palpation. The tenderness she complained of could not be described as severe. No blood was found in the vagina, nor was there any staining of her clothing. The os was dilated (2 cm.), with distended membranes bulging through. Concealed accidental haemorrhage was diagnosed and Caesarean section recommended. This her husband agreed to, but requested that the uterus should be preserved if found possible. She was removed to Longton Hospital.

At 7.30 p.m. on January 1st, 1924, under a general anaesthetic, the abdomen was opened through a mid-line incision extending from 1 inch above the umbilicus to 1 inch above the pubes. A little free sanguineous fluid escaped from the peritoneal cavity. On attempting to centralize the uterus it was found impossible to insinuate the hand between it and the costal margins. The distension of the uterus was definitely limited to the body, which was of a slaty grey colour with purplish mottling, while the lower uterine segment, below the anterior peritoneal reflection which was raised to the level of the umbilicus, was elongated and showed neither grey coloration nor mottling. A longitudinal incision through the loose peritoneum over this segment exposed sufficient of it to permit a 5-inch incision into it without reflecting the bladder downwards. This incision was made in the long axis. No difficulty was experienced in extracting a well developed dead child of about 7 lb. by the presenting breech. The skin of the scrotum had desquamated, otherwise the child was in a good state of preservation. The placenta was free in the uterine cavity and was removed with three or four large dark blood clots. These clots collectively would have filled a half-pint measure. Simultaneous with the extraction of the child 1 c.cm. of pituitary extract was given subcutaneously and the uterus lifted out of the abdominal cavity. It remained flabby and toneless, and except while directly compressed there was a steady oozing from its cavity. After fifteen minutes' compression without satisfactory result gr. 1/100 ergotinine citrate was injected directly into the uterine muscle. This had the desired effect, and the uterus was preserved. The placenta was of normal shape and size, but four-fifths of the villi showed that they had been compressed. The remainder occupied an arc embracing a third of the periphery of the placenta. When the uterus was removed from the abdominal cavity, within a minute of giving pituitary extract, the intestines were seen collapsed and ivory white in colour; this persisted during the time the abdomen was open. The incisions in the lower uterine segment and in the peritoneum over it were closed separately with continuous catgut and the abdomen closed in layers.

Except for severe epigastric pain which lasted for two days convalescence was uneventful, and she went home on January 20th; the uterus had then contracted down to the size of a two to three months' pregnancy.

While concealed accidental haemorrhage is one of the rarest complications of pregnancy (1 in 15,000 or more cases), it is one of the most serious. In all but those in which the haemorrhage has been trivial the child must die *in utero*, and in those severer cases the uterine muscle is so damaged and its power of contraction and retraction so destroyed that the mother's life is endangered by the probability of post-partum haemorrhage, if not by conditions arising antecedent to the birth of the child.

To draw conclusions from one case can only be justified by the rarity of the condition, and by the hope that others will compare such conclusions with their own personal experiences. The condition has been attributed to toxæmia, trauma, emotional disturbances, and endometritis, and is commoner in multiparae. This case throws no light on the subject, but the date and hour of its onset are significant—10 p.m., December 31st.

Diagnosis offers no difficulty. The sudden onset of abdominal pain in a woman advanced in pregnancy, with marked collapse and with no "show," should suggest the condition. The tense hard swelling of the uterus, particularly in its upper segment, with inability to hear the foetal heart sounds or uterine souffle, are sufficient to confirm the diagnosis.

The sequence of events following haemorrhage into the decidua basalis must depend on the amount of blood poured out of the ruptured vessels and on the increased intra-uterine pressure resulting. As long as the haemorrhage remains concealed between the placenta and membrane on the one hand and the uterine wall on the other the increased pressure acting through the liquor amnii tends to dilate the os to some extent, at the same time ballooning the uterus, and disseminating the fluid blood between

the bundles of uterine muscle. This sequence of events would contribute to a lowering of the intrauterine pressure if the haemorrhage ceased, but in reality it encourages further bleeding, with increased distension of the uterus and greater dissociation of muscle bundles until the intra-abdominal pressure is raised to or above that in the uterus; then the blood sinuses at the placenta site are subjected to compression which closes them and permits thrombosis to occur. It might be argued that such a state will occur earlier in a primipara or in a multipara with well developed and not overstretched abdominal muscles. Conversely, lax abdominal muscles will allow so great uterine distension that the peritoneum cracks and the dissociated muscle bundles are converted into actual ruptures permitting blood to escape into the abdominal cavity. Such cases have been described by Shannon and others. If this case can be taken as a criterion of what occurs in others, then the distension is limited to the uterine body, while the lower segment is merely stretched longitudinally and is otherwise not traumatized by the disseminating blood.

If the diagnosis could be made early, the ideal treatment would be to convert the concealed into an external haemorrhage, but as the first symptoms of concealed accidental haemorrhage arise from shock due to the rapid distension of the uterus, most cases are seen too late for this to be done without considerable risk. The firm application of an abdominal binder may assist in raising the intra-abdominal pressure and minimizing the haemorrhage.

To empty the uterus *per vias naturales* endangers the patient's life from post-partum haemorrhage, whereas if it is emptied through an abdominal hysterotomy opening the post-partum haemorrhage can be controlled by direct compression or by hysterectomy if other measures fail. If Caesarean section is performed the lower uterine segment operation appears to be more desirable than the classical method. It is the part of the uterus underlying a mid-line incision below the umbilicus; it is stretched longitudinally and there is ample room without the necessity of reflecting the bladder downwards; it is not traumatized, at least to the same extent, as is the body; and if the uterus should become infected it is shut off from the peritoneal cavity.

The collapsed and ivory-white condition of the intestines is difficult to explain, especially as no reaction appeared during the twenty to twenty-five minutes the abdomen was open. The greatly increased intra-abdominal pressure, with irritation of the splanchnic nerves, may have had some influence in producing this state, or it may be a phenomenon of shock as described by some writers on that subject.

A PROTEIN POISON THEORY:

ITS APPLICATION TO THE TREATMENT OF HEADACHE AND
ESPECIALLY MIGRAINE.

BY

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ALL nitrogenous food, animal or vegetable, contains a potential poison. Whether untoward effects are produced or not depends entirely upon the metabolic efficiency of the individual in respect of the particular protein concerned. If the metabolic capacity of an individual is insufficient to deal with this poison, without undue reaction, manifestations are produced. These manifestations may occur in any tissue of the body, and are the symptoms of disease.

In chronic disease the reactions are so slight that they are not evident until the gradual replacement of the destroyed tissue cells by connective tissue interferes with the function of an organ. This explains the slow development of arteriosclerosis, nephritis, and other diseases of like nature. On the other hand, the manifestations may be sudden and severe, as in cyclic vomiting and migraine. Most acute manifestations show a certain periodicity, indicating a cumulative action of the poison. Other acute affections appear immediately after the ingestion of the poison, as

in the case of urticaria, angio-neurotic oedema, and the various food idiosyncrasies. Convulsions may be caused by protein ingestion, and, indeed, this is a very usual cause of convulsions in children.

My theory differs from that of anaphylaxis, which has been generally accepted to explain various food idiosyncrasies. In anaphylaxis a preliminary sensitization is required, while my theory maintains that the metabolism of the individual is inadequate. Such an inadequacy for the metabolism of nitrogenous food is most usually inherited, but further damage can be prevented by appropriate diet. The protein poison may act directly on the tissue cells of the mucous membrane or the skin, causing, for example, in the one case bronchitis or colitis, in the other such a disease as eczema. Or again, it may act indirectly, as, for example, through the endocrine or nervous system.

It is impossible in this short paper, which I wish to devote to migraine, to discuss fully the pathogenesis of protein poison manifestations, but since heredity plays such an important part in the etiology of migraine, this at any rate must be considered. The old French school represented by Trousseau, Bouchard, and Dieulafoy laid great stress upon what they called the lithaemic diathesis, and insisted on the close relationship and inheritability of gout, asthma, eczema, migraine, epilepsy, and lithiasis. They also noted the frequent occurrence of cancer in certain families which had this lithaemic diathesis. They failed to see, however, that the real basis of the heredity is a damaged protein metabolism in which protein poison manifestations can take place in any tissue or organ of the body, so producing the diseases which they attributed to lithaemic diathesis. The disease and its symptoms depend entirely upon the site of the manifestations. Each cell of the body is an individual chemical laboratory and plays a part in the general metabolism.

I consider it is quite justifiable to assume that the molecular arrangement of the cell structure, and the characteristics of the cell, upon which depends its ability to function properly, are inherited in the same manner as the general physical characteristics of the individual are inherited.

Migraine occurs in individuals who have inherited a damaged metabolism for nitrogenous foods, and I have chosen it as the subject of this paper because the remarkable results that I have obtained in its relief by the practical application of the protein poison theory have been so conclusive as to leave no doubt in my mind as to its truth and value.

Headache is one of the commonest forms of protein poison manifestation. The headaches caused by protein poison vary in severity from the mildest headache to the severest forms of typical migraine. The etiology of all functional headaches is the same. In migraine we have, however, the typical inherited insufficient protein metabolism. It is true that other causal factors, such as eye-strain, may act in producing headache, but they are secondary causes and are incapable of producing migraine if the essential cause is removed. It is quite easy to prove that protein poison is the essential cause of headache by letting a patient resume the articles of diet the omission of which caused the headache to cease. It is true, however, that some patients on a low protein diet will acquire a certain degree of tolerance for the very foods that formerly caused headache. Other patients, however, will never be able to take certain protein foods. It is most important to know that extracts from protein foods such as soups and gravies, and fruit extracts also, though they contain no protein, contain the protein poison and are very potent in causing headache; indeed, one reason why dieting has been unsuccessful in the past is because meat extracts and fruit and fruit juices have not been suspected.

I have found that skin tests are unreliable, and the quickest and best way to ascertain what foods are deleterious in each individual case is to commence with a diet free from protein, especially animal protein, and then to build up gradually a suitable diet therefrom.

The foods which I have found potent in causing headache are meat, fish, poultry, game, and extracts such as soups and gravies made from them; eggs, milk, and cheese; fruit and fruit juices; coffee, tea, chocolate, and alcohol;

and, among the vegetables, tomatoes and mushrooms. This at the first glance seems to leave nothing to eat but cereals, butter, and vegetables; and, indeed, this is the basic diet upon which to put a patient for a few weeks until his headaches are controlled. I use a diet of polished rice, butter, toast, a few green vegetables, and water.

Many patients show immediate improvement, but some are worse the first week—those who formerly had occasional headache having it continuously. This is especially true of immoderate coffee drinkers. The second week there is considerable improvement, and in the third week the majority of patients are distinctly better. In women there is usually a recurrence of headache at the first menstrual period, but after a month or two they have no more headache, not even at the menstrual period.

So soon as the headache is distinctly improved the diet should be amplified, at first with other vegetables, then by a small amount of meat once a day. Milk may be used in the cooking (many patients cannot take raw milk in any quantity) and different articles of nitrogenous food added from time to time until the tolerance for different foods is ascertained. Very few subjects of migraine can take meat soups, fruits, or eggs in any quantity. Sweetbreads, mushrooms, tomatoes, and chocolate usually must be banned entirely. With some it is necessary to omit a single article only, such as eggs. Every article of nitrogenous nature must be suspected until proved to be harmless in the particular case. The protein of cereals and vegetables is usually well borne.

It must be remembered that every food of nitrogenous nature has its specific protein. Some patients who cannot take beef can take lamb or chicken. I have had patients who were very susceptible to such proteins as unpolished rice, almonds, and peppermint. Some can take tea with impunity, but cannot take coffee; as a rule, however, most can take a fairly generous diet if certain particular proteins are omitted.

It is best that all sufferers from migraine should be on a fairly low protein diet. I believe also that the less carbohydrate and fat that is consumed the greater is the ability of the patient to metabolize protein. This may explain why some cases of migraine have been relieved by adopting a low carbohydrate or low fat diet; but I am convinced from my experience that the protein is the real causal agent of migraine.

I am well aware that the statements I make will seem somewhat dogmatic, but I am convinced of the truth of them. A single trial of this method in the most obstinate case of migraine will convince the most sceptical, and the success of his treatment will soon tempt him to apply the theory in other cases of functional disease, where it is chiefly applicable. Diseases in which organic change has already begun cannot be cured by diet, but the protein poison theory throws light upon their etiology and therefore suggests the possibility of preventing all the diseases of metabolism. It gives a new viewpoint in such serious diseases as nephritis, arterio-sclerosis, and even tumour growth.

CANCER OF THE BREAST:

RECURRENCE 31 YEARS AFTER OPERATION.

BY

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I MAKE no apology for publishing a note of this case owing to its outstanding interest in its bearing on the age-long question, *When is cancer cured?*

I saw the patient a short time ago with Dr. James Bromley of Sible Hedingham. Her history was as follows:

Mrs. R., aged 81 years, was operated upon for cancer of the left breast thirty-one years ago by the late Sir Henry Butlin. She had no further trouble until a few months before I saw her, when Dr. Bromley discovered three small hard nodules beneath the skin of the left chest in the immediate neighbourhood of the scar of the operation wound. These had slowly increased in size. During the