2. If this be true, then it follows that the Fallopian tubes have no special physiological function in menstruation; they are neither "the cause" (if any meaning can be put into the term) nor "the starting point" of menstruation.

3. A practical deduction from the whole matter is that in operating on the broad ligament for the purpose of bringing on the menopause or involution of the uterus, the operator should endeavour to tie the chief branches of the ovarian arteries. Especially is this the case when the object sought is to bring about retrograde changes owing to the presence of small fibroid tumours of the uterus. There is little or no danger of starving the uterus by cutting off the blood supply to a dangerous extent; the danger is almost always that too little will be done, and the object sought for only imperfectly attained.

A LOCALISED EPIDEMIC OF INFLUENZA FATAL FROM HYPERPYREXIA.

By HUGH JONES, M.B.,

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At the spring meeting of the North Wales Branch of the British Medical Association (1892) I read a brief record of a series of cases of fatal influenza which had come under my care. These cases, so violent in character and so disastrous in result, occurred in a remote, comparatively inaccessible, mountainous country district. Twelve deaths occurred within a few miles of one another in a small sparsely populated district situated about seven to ten miles from the town of Dolgelly. The district only covers an area of about three or four miles, and is not traversed by any highway of communication between large centres of population. The industry is entirely agricultural, with the exception of a gold mine situated on the edge of the district, which gives employment to about a hundred men. The age of the patients ranged from 8 to 53 years. The twelve fatal cases occurred among males, although I also met with several mild and ordinary cases of influenza among females in that district. I therefore concluded that sex was an important element in these fatal cases. This is more striking in view of the strong case that has been made out for the infectious character of the disease by Dr. Parsons and others; for all these cases were largely and mainly nursed and attended by females. My observations, at the same time, tended to confirm the view that "influenza is mainly, if not entirely, spread by contagion," although, from the free mingling of neighbours with one another in a rural district, it was impossible to make any definite statement on the matter.

Signs of catarrh of the respiratory tract were practically absent. The onset of the attack was sharp, sudden, and well defined. The first symptoms were those common to ordinary influenza—namely, chilliness, headache, and pains about the limbs and trunk, along with all the signs of extreme prostration and muscular weakness. There was unusually great acceleration of the pulse rate from the beginning, as well as a marked elevation of temperature, but the hyperpyrexia, which was the great and leading factor in these cases, did not show itself on the first day of the illness. I entirely failed to find any physical signs of inflammatory mischief which would in any way account for the terribly fatal character of the disease. The skin acted freely throughout almost without exception. The tongue had invariably a thick white coating. The urine was high-coloured and loaded with urates, but was not deficient in quantity beyond what might have been expected from the free diaphoresis. There was nothing in the chest or abdomen which I could detect by physical examination which calls for special mention.

Indeed, all these cases at the outset came quite within the category of mild and ordinary influenza, and no prominent danger signals showed themselves till the end was comparatively near. The illness lasted from two to five days, and the patients continued in much the same state as described above until the temperature began to indicate the extent to which the fever poison was playing havoc with the system. The pyrexia of the first stage did not exceed 104°F ., but the ultimate hyperpyrexia ranged from 106.5° to 108.5° . With

the approach of this hyperpyrexia the great prostration began to express itself in extreme restlessness, which gradually merged into delirium, which became latterly of a most violent character. This violent delirium gave place to coma and collapse just before death.

I describe these cases as instances of fatal uncomplicated influenza owing to my inability to detect any physical signs which pointed to any organic and tangible change in the structure of any of the tissues of the body. Examination of chest and abdomen, as previously stated, was quite negative, and, although there were such conspicuous indications of serious interference with the functions of the different parts of the nervous system, there was nothing to show definite change of structure as such. The pupils showed nothing unusual, and there was no localised paralysis of motion or sensation in any part of the body. Instead of pyrexia we had hyperpyrexia, and instead of restlessness and stupor we had delirium and coma.

As regards treatment, I may say that I tried the ordinary febrifuge remedies, with antipyrin, quinine, salicylate of soda, and large doses of bicarbonate of potash; but I am not aware that any of them were of special use. It was quite impossible to try the cold pack, even if I had been partial to it. We did all that was in our power in the way of nursing and supporting the patients, but I have to confess that the resources and conveniences of the neighbourhood were unfortunately most limited.

It would serve no good purpose to describe the cases individually, but I may perhaps be allowed to refer to one case in particular to show how terrible a disease we were dealing with.

On June 26th, 1891, I first saw R. H., aged 34, at about 12 noon. The patient was a strong, muscular, healthy, well-developed farmer. He had been taken ill during the night with the usual initial symptoms—pains all over, tendency to chills, headache, etc. When I saw him he was perspiring freely, pulse very quick, and temperature 103°F . He had no cough, and I could find nothing in the chest. His face was flushed, but not livid, and breathing was free, although accelerated. The urine was loaded with urates. I saw him again the next morning about 11. The relatives reported a satisfactory night. The patient had perspired freely all along, had taken nourishment well, and there had been no tendency to delirium. The temperature was now 101°F ., the skin was acting well, breathing quieter, and pulse not so quick as on the previous day, and showed good volume. The patient expressed himself as feeling much better. In the afternoon, however, the friends noticed that he was not so well. There was something strange in his countenance, and they observed that the skin became intensely hot, and not so moist. With this the patient became so violently delirious that he could hardly be kept in bed, and died about 9 P.M., and just about an hour before I managed to reach the house after being sent for. This disease then killed this strong man in less than forty-eight hours.

The other cases, which I had more opportunity of following, did not terminate so rapidly, but eventually showed exactly the same features. The delirium was violent, and the highest temperature recorded on my thermometer was 108.5°F . The last death in this series of cases occurred on July 19th, 1891, and my remarks at the North Wales Branch meeting only referred to these, but on August 5th, 1892, I again met with a sporadic case, which was so similar in character that I felt compelled to give it the same name.

On August 5th, 1892, at about 10 A.M., J. W., aged 22, called at our surgery. The patient was a healthy, muscular, young man, and took a prominent and successful part in athletics, especially cricket. He did not feel well on rising in the morning. He felt chilly, general aches and pains, and some slight sore throat. I was struck with the quickness of pulse, and therefore took the temperature under the tongue, which was 102°F . Examination of the throat revealed nothing typical. There was some slight faucial inflammation, but no membrane or marked enlargement of the tonsils. I called at the patient's house to see him about 6 of the same evening. His temperature was 103°F , pulse still very quick. Bowels had acted satisfactorily after an aperient given in the morning, and the skin was acting freely. He felt the throat much better, and suffered now no inconvenience from it either in swallowing or otherwise. I saw him again at 11 P.M. The temperature had risen to 104°F ., but in spite of this the patient expressed himself as feeling very comfortable. I was somewhat alarmed at the continuous rise in temperature, but I hoped that under treatment and with free sweating the pyrexia would be less by the morning. However, when I was again called to see him at 7 A.M. I found the temperature was 106.5°F . The skin was moist, but there was no free perspiration. He was conscious, but his countenance bore a somewhat wild and strange expression. Pulse extraordinarily quick, breathing free, and face more pale than otherwise. At 7.45 A.M. the temperature was 108°F , and patient rambling. At 8.30 A.M. 109°F was recorded. Patient delirious and somewhat violent. Pulse running. At 9.30 A.M. the temperature was 110°F . Patient unconscious and somewhat rigid, breathing heavily and noisily; power of swallowing gone. He died a few minutes later, in spite of all that could be done for him—in less than twenty-four hours after he had visited the surgery.

I have called this case malignant influenza, although this may not necessarily be correct. The throat symptoms may

at first have pointed to a malignant form of suppressed scarlet fever, which was, however, not prevalent in the district at the time; but there was such free diaphoresis, and the throat trouble was so slight and transient, that I felt, judging also from the other points of resemblance to the first series of cases, that I was certainly dealing with the same disease, although this patient lived at a distance of some seven or eight miles from the district in which the other cases occurred. This suggests the question of the duration of the incubation period in this disease. This last patient had spent a whole day in the previously-affected district in the month of May, 1892, and had then visited some of the houses in which fatal cases had occurred in 1891. In connection with this, I would mention that the first death in 1891 from this special form of influenza occurred on March 29th, and that the next death occurred on May 12th, and this patient was the son of the one who died on March 29th. If this son received the contagion by personal infection from his father, then there was an incubation period lasting from about March 29th till May 9th, when the son began to sicken. Again, if the patient who died on August 6th, 1892, received the contagion on his visit in May, 1892, to the infected district, then two clear months—June and July and some days—must have indicated the incubation period. I merely mention these facts, and leave the profession to draw any inference from them.

MEMORANDA

MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, ETC.

ACUTE INVERSION OF UTERUS: SPONTANEOUS REDUCTION: RECOVERY.

ON April 1st, 1892, I was called to attend Mrs. C., *primipara*, aged 33, married ten months. She had been in labour some time. Pains were slight, contractions irregular, and the second stage very slow. A male child of average size finally born twelve hours after the onset of labour; the cord was very short and thick.

About five minutes after the birth of the child the patient complained of severe bearing down, felt sick and faint, gave a shrill scream, and immediately the inverted uterus appeared below the vulva, with the placenta attached somewhat to the left of the fundus. The placenta was peeled off, and hæmorrhage, which was free, stopped by hot water affusion. There was now marked collapse. An attempt was made at reposition, but handling the uterus caused intense pain and deeper symptoms of collapse. The condition of the patient becoming alarming, I sent for my friend Dr. McElwaine, and we again tried replacement, but so severe was the resulting collapse that we thought death had supervened. After about half a tumblerful of brandy had been got down the patient's throat she revived. The uterus was placed as far as possible in the vagina, further attempt at reposition being deemed dangerous.

April 2nd. The patient became stronger, and free from pain; there was oozing from the uterus, which was again protruding, a result of having been permitted to strain to pass urine. The womb was bathed and dusted with iodoform, replaced in the vagina, and antiseptic pads placed over the vulva. On consultation it was deemed advisable not to make any attempt at reduction till the patient was stronger and able to bear an anæsthetic. From this time till April 10th urine was drawn off daily, the vagina syringed with corrosive sublimate solution and iodoform, and salicylic wool pads applied. On April 11th an attack of pneumonia set in, the temperature rising to 104° F. On May 4th the patient was so far recovered that another attempt at reduction was made, but the organ was found to be so soft that the procedure appeared to be dangerous; further action was deferred until the uterus was completely involuted.

On July 1st the patient was seized with most acute pain in the vagina, sickness, and faintness. These symptoms stopped suddenly, and from this time oozing, which had been continuous, ceased; she lost the sense of weight in the vagina, and felt much better.

On October 29th the patient came to ask when I proposed doing the contemplated operation. I was astonished to find the uterus replaced, but slightly retroverted. The os was patulous, and the sound passed to a depth of $4\frac{1}{2}$ inches. The patient felt strong and looked in good health; the catamenia had not appeared.

The occurrence of this, "one of the most formidable accidents of parturition" (Playfair), is very rare. In Rotunda Hospital, where I studied, it had not been seen once in 190,800 cases; C. von Borun had not seen one in 250,000. Byford writes that no more serious complication can occur. Cross states that, of 109 cases, all died, 73 deaths taking place in a few hours. Thomas regards spontaneous reduction as a curiosity, Cross denies that it could occur, while Barnes does not see why it should not happen in first and second degrees. Spiegelberg and Shaw had each one case, but I do not know the particulars.

Stonehouse, Devon. W. H. WATERFIELD, L.R.C.S. & P.I.

CASE OF ACONITE POISONING.

AT 7.40 P.M. an urgent request was made to me to visit a married woman, aged about 40. I attended within five minutes, and found her retching violently in her garden. She was being attended to by two neighbours, who had given mustard and water to produce emesis. I was shown a 2-oz. stoppered bottle, about half full of Flemming's tincture (six times as strong as tinct. aconit. *P.B.*), from which the husband had seen the patient drink. Attempts to introduce a tube into the stomach increased the retching; tumblerfuls of warm water were therefore given repeatedly for twenty minutes, each draught being vomited. Sp. am. co. 35 *iss ex aqua* was given and retained for a short time; the patient was then put to bed, and another dose of sal volatile given.

The symptoms were intense burning sensation in the præcordial and epigastric regions, a feeling of suffocation, and later a numb condition of face and hands. The physical signs were pallor of the face, staring appearance, and undeglintening of the eyes: the pupils reacted to light, and were of normal size; the pulse was full, 80.

About half an hour after I first saw the patient, the surface of the body and extremities were cold. An hour after poison had been taken, the pulse had become irregular, and had fallen to 60. At 8.30 P.M., tinct. bellad. $\frac{1}{2}$ x was given and repeated hourly, and cognac $\frac{1}{2}$ oz. in milk was given at intervals of two hours through the night. At 9.30 P.M., the pulse was 60, the burning sensation was diminishing, the surface was warmer, but a "drawn" feeling in the face and tingling in the hands was felt. At 11 P.M., the tingling and numbness were passing off. The tinct. bellad. given every two hours.

The next morning at 8.30, patient (having slept for two hours) was tranquil, and all symptoms had gone. Pulse 72, regular.

REMARKS.—The drug, a teaspoonful of which the patient believes she swallowed, was taken on an empty stomach. As far as I could learn, emesis was brought on by the prompt action of the neighbours within five minutes of taking the poison.

Farningham, Kent.

T. F. HUGH SMITH, L.S.A.

MEMBRANOUS ENTERITIS: OBSTRUCTIVE JAUNDICE.

I. H., a strongly-built man, 48 years of age, had been subject to occasional "bilious attacks," lasting a few days, during which his stools were clay coloured. This condition invariably terminated in vomiting, when he was entirely relieved. There was no history of biliary colic.

On February 26th he complained of pain and distension in the epigastrium, and nausea. On the next day jaundice was marked, and the stools were clay-coloured. Saline aperients and cupping over right lobe were prescribed, and the patient gradually improved up to March 2nd, when he insisted on leaving for the country.

On March 7th he returned. Jaundice was more intense, and there was pain in the region of the gall bladder, the liver more particularly the left lobe, was swollen. The temperature rose gradually, until on March 16th it reached 103°. He had several rigors, and the stools were white, and highly offensive. The bismuth prescribed previously was replaced by salicylate