resistance retards the action of elasticity, just as any ordinary force is retarded by friction—in fact, it is spoken of as molecular friction.

Now, in our conception of retraction it is essential to realise that the shrinking of the uterine walls is probably always, and certainly at times, a gradual process, and, compared with contraction, is slow. Retraction does not necessarily follow close on the heels of contraction. It is a persistent force, and acts when unrestrained in the intervals between the contractions. The action is gradual, and is retarded in proportion to the amount of viscous resistance in the tissues. It is with and because of the diminution in the bulk of the contents that retraction occurs, but, even when the uterus has been emptied, some time elapses before retraction is complete. This is well illustrated in the condition of the uterus immediately after the expulsion of the placenta. The contraction which has completed the third stage passes off, permitting of partial relaxation of the walls until retraction is complete—and every practitioner knows that it is some time before that is the case and before the uterus can with safety be left to itself.

At present I have dealt with retraction as relating to actual labour only, and reserve for future consideration the question what part it may take in the production of the changes which occur in the preliminary stage of parturition.

There are, then, three properties to be recognised in the uterus, and when we realise the distinctive nature of each, and keep them in our minds separate one from another, we can understand the part that each one plays, and we obtain a conception of the uterus which satisfactorily explains many of the phenomena met with in pregnancy and labour.

First there is Muscular Contraction, the result of the energy rendered kinetic by a stimulus. It is powerful but intermittent, lasting only for a comparatively short time. It is resident in the muscular tissue alone, and causes only a temporary shortening of the fibres; so soon as the contraction has ceased the fibres are capable of returning to their original length. The force so derived is the only one that can reduce the bulk of or expel the solid contents of the uterus.

Secondly, the Molecular Tension of the tissues, whereby, when free from disturbing influences, they constantly tend to assume the smallest superficies consistent with the conditions. This property belongs to the serous as well as muscular coat. The energy differs from that of contraction in being feeble but persistent, and from that of ordinary elasticity in being always the same in amount however much or little the tissues are expanded. It gives rise to the continuous internal uterine pressure. It alone is the cause of the permanent diminution in the superficies of the uterine walls. It is not an expulsive force except in so far as fluids free to move are concerned; it may express some liquor amnii but cannot expel a clot of blood. Under its influence, contraction being absent, it gathers the walls around the uterine contents.

And thirdly there is the Viscosity of the uterus. It offers resistance to a rapidly dilating force, and explains the unlimited yielding without rupture to a small but persistent force. It retards and for a time renders the action of retraction imperfect. Through it the internal surface under retraction presents a true mould of the contents.

Further, to complete our conception of the uterus, we must remember that, whatever be the condition of the muscular fibres, they are surrounded by, and are closely attached to a serous membrane, which possesses the same property of molecular tension, and which evidently acts as an elastic capsule, offering resistance to any dilating force, and aiding in the establishment of perfect retraction.

This conception of the uterus enables us to understand various phenomena that the older views failed to explain. In Cæsarean section, whenever the child is lifted out, the uterus retracts at once, often grasping a part of the fœtus. Here it may be objected that the manipulation supplies the necessary stimulus for contraction in a uterus ready to act. In ordinary labour, however, we at times meet with cases where the uterus has become exhausted, and all our endeavours have failed to excite contraction. Nevertheless, on artificial delivery, by means of turning or the forceps, the uterus, though previously inert, follows down the diminishing bulk of the contents, and

retraction, though slow, is soon complete. This immediate response of a fatigued uterus always was a puzzle to me so long as I thought of contraction only, or that retraction is dependent upon contraction.

When the hand is passed into the uterus to remove the placenta, we find it may remain for some time in the cavity without exciting a contraction, but, the moment the hand is being withdrawn, the walls can be felt to retract, and the cavity to become obliterated. This is retraction simple in an empty uterus, not contraction.

Further confirmation of the truth and value of these views is obtained by observing the uterus immediately after the expulsion of the placenta. The contraction which has completed the labour soon passes off. By the hand externally one becomes conscious that the organ is less hard, and enlarges somewhat. What now is the condition of the organ? Contraction has ceased, the muscular fibres are relaxing and are capable of yielding to an expanding force. The only such force present is blood pressure, which, if no counteracting force be present, will refill the sinuses, the blood will escape into the uterine cavity, and further distend the organ. The counteracting force is the molecular tension of the tissues, not of the muscular fibres alone, but of the serous coat as well, producing retraction. Normally the shrinking is sufficiently complete to prevent any marked distension; but, under special conditions, the action is retarded by viscous resistance, and, if a fresh contraction does not come to the rescue, the muscular walls and cavity become filled with blood. As Matthews Duncan has remarked, "it is retraction, and retraction alone that is a safeguard against hæmorrhage."

The whole subject of the management of the third stage of labour and the treatment of *post-partum* hæmorrhage will fall to be re-examined under the view of the distinct and independent action of contraction and retraction. Fresh lines of inquiry are opened up as to the causes which affect the one and the other. Does frequent or long-continued contraction increase the viscosity, and so retard the action of the molecular tension? How far is the latter force increased or diminished by heat or cold? What is the true action of ergot? What is the influence of chloroform and chloral on the tension as distinguished from the irritability of muscular fibre? These and other problems demand fresh inquiry that will require time for careful investigation. Meanwhile it should be always borne in mind that the science of midwifery can be advanced, not only by the discovery of new truths, but also by clearing our knowledge of the encumbrance of unscientific methods of thought and ambiguous modes of expression.

NEURALGIA OF THE HEART, AFTER INFLUENZA, WITH A FATAL CASE.

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AMONGST the various complications of influenza, some of the most alarming are those connected with the heart. I do not refer to lesions of the valves, but to affections which appear to depend on the bulb and the nervous regulating mechanism. The three following cases occurred in well-to-do patients who were able to take the greatest care of themselves from the first; there was no exposure to weather after the first manifestations of the influenza; there was no other complication beyond that of the heart, and in no case was there a cardiac organic lesion. All three were alarming in appearance, and one was fatal in its ending.

CASE I.—An American gentleman, aged 39, had lived in Italy for some years. Did not have the influenza during its visitation two years ago. He was stout, took little active exercise, and was an inveterate smoker, consuming at least ten strong cigars per day. He sent for me simply to know if he had had the influenza, as he felt fairly well, and complained of nothing but weakness. From the description of his fever and aches during the previous days I said he probably had had an attack, and must remain indoors for some days. There was no fever, but I noticed that the man's was slow, small, compressible, and with an occasional intermission. The heart's sounds were normal; there was no cardiac pain. A preparation of caffeine and quinine was given. That evening the patient had a return of fever, and next morning I found the temperature 100°. This second attack of fever quickly subsided under salicin. The pulse fell to 56, but under digitalis became regular. The patient was allowed up, but confined to the house, and advised to keep quiet

and be very moderate in smoking; nourishment was ordered in small bulk, but frequently. Eight days after first being called to see him he had a business meeting in his house, smoked, played cards, ate a good dinner, and went to bed feeling very well. Next morning he was awakened by a pain in his chest, flatulence, and vomiting. He had no marked pallor; constant wearing pain in left chest, midway between the nipple and the shoulder. Pulse 90, soft, regular, compressible; tension not increased or prolonged; no rigidity or thickening of arterial walls. Cardiac impulse not felt, area not increased, no cardiac tenderness, and the pain was not relieved by pressure; heart sounds normal but feeble. Tongue slightly furred, complete anorexia, continuous eructation with flatulent distension of abdomen. The patient did not remain still, and movements did not aggravate the pain. The pupils were equal and normal in reaction. The breast pain did not go up to the shoulder or down the left arm, and as some of the other characteristic symptoms of angina pectoris were wanting, and the pulse tension was already very slack, I did not give amyl nitrite, but administered at intervals two hypodermic injections of $\frac{1}{4}$ -gr. and $\frac{1}{4}$ -gr. of morphine. Digitalis, ammonia, and carminatives by the mouth were rejected; enemata failed to expel the flatulence, yet the patient expressed relief on eructation. The temperature was normal; there was no cough or lung symptoms. The stomach rejected all nourishment, and nothing beyond ice with whisky and soda was retained. During the day the breast pain diminished, but never entirely ceased. In the afternoon two hypodermic injections of caffeine were given. The patient felt well enough to get in and out of bed without faintness or increase of pain. Towards midnight, after a turpentine enema and the application of turpentine stupes, the patient expressed himself as quite free from pain, and insisted on everyone retiring to rest. The pulse was 88 and regular. He remained quiet for an hour, and then asked for a pill of morphine (gr. $\frac{1}{4}$) as he could not get to sleep. While having his head raised to swallow it, he suddenly fell back, and, without a word, instantly expired, twenty hours after the first onset of cardiac neuralgia.

There was no necropsy, but the patient had the appearance of a man in whom the heart might be fatty and fatty. His father died of angina pectoris, after repeated attacks, at the age of 61. His mother died of apoplexy. He had never complained of heart symptoms, or had previously any serious illness.

CASE II.—A lady aged 50, had a mild attack of influenza two years ago. Feeling tired and aching after a long walk, she did not sleep well, and was up from early morning, with an acute pain in the epigastrium and an aching in both shoulders. She was pale, and evidently suffering; tongue clean; anorexia; no vomiting. The pain was referred to immediately below the ensiform cartilage; relieved by pressure. Pulse under 50, regular, compressible; heart sounds normal, area not altered, impulse not marked; no rise of temperature. I suggested the possibility of influenza, and only discarded the idea as I knew the patient had had several attacks of *embarras gastrique*. As morphine gr. $\frac{1}{4}$ produced no effect hypodermically, a $\frac{1}{4}$ -gr. was given and procured relief. After eight hours the pain, which had never quite disappeared, returned more acutely, and was referred to underneath the left breast. It was relieved by pressure; no cardiac tenderness. The pulse had fallen to 40, regular, but of alarmingly low tension. A hypodermic injection of caffeine and ether was given, and champagne and hot bouillon were pressed on the patient. In a few hours the pulse had risen to 50, but another morphine injection (gr. $\frac{1}{4}$) was required. Next morning the temperature was 99.5°; the breast pain continued, and as the pulse was over 50 and of fair tension I tried 2 minims of amyl nitrite by inhalation. The face flushed at once and the angina was instantly relieved; it returned, however, in a few minutes, and continued through the day. The flushed face remained; but this may have been explained by the rise of temperature, for by the evening it had reached 101.5°, while the pulse had risen to 70. The feverish symptoms yielded to salicin; the breast pain was sufficiently diminished to permit sleep, but for more than a fortnight and in spite of rest and treatment the patient was not free from cardiac pain.

CASE III.—A very spare lady, aged 61, who had already had two attacks of influenza; the first was in England a year ago; and the second last Christmas in Florence. In neither of these cases was breast pain complained of. On February 2nd she complained of headache, pains all over, particularly under the left breast. Temperature 101°, pulse 106. No marked pallor; face flushed readily on the slightest exertion; no increased or prolonged tension of the pulse; no cough and no chest symptoms. A belladonna plaster was applied over the apex, and digitalis and strychnine given. On February 9th (that is, after a week) the pulse had fallen to 80, and the temperature to 99°; the patient was allowed to sit out of bed. Next day the breast pain returned with double severity. It was constant and clutching in character; it had its maximum intensity under the left breast, but was also referred to both shoulders, particularly the left. There was a constant tendency to sigh, yet fear of drawing a long breath. Cardiac area not increased; impulse marked; sounds normal; marked tenderness in second left intercostal space and over the apex. The temperature was 101.4° and the pulse 130, regular, soft, compressible. The same evening, under the influence of two small blisters over the heart and 5 minims of digitalis tincture every four hours, the temperature had fallen to 100.2°, and the pulse to 108. Morphine was given both hypodermically and by the mouth; the pain slowly diminished during the following days, but not until a week had elapsed was the patient ever quite free from it. Digitalis, arsenic, nitroglycerine, amyl nitrite, and *nux vomica* appeared to make little impression on it; it "wore itself out."

In these cases there was no thickening or rigidity of the arterial walls; and in none was the urine abnormal. In none of the three cases was there dyspnoea, unconsciousness, convulsion, or excitement.

There was no doubt as to influenza being the immediate cause in all cases; although in the absence of an epidemic it might have been difficult to determine this. Predisposing causes were absent, except in the fatal case where excessive smoking doubtless acted injuriously on a heart which was

hereditarily vulnerable. In Case I the neuralgia came on after eight days of interval from the influenza attack; in Case II it appeared to precede it; and in Case III the manifestation of the two was simultaneous.

These cases would indicate that the influenza poison attacks particularly the nervous system, and may limit itself in its severity to one restricted portion of it. The bulb was undoubtedly attacked in all cases; in the first case the acute vomiting, and in the second the pain commencing in the epigastrium, point to the gastric fibres of the vagus being affected; while the local tenderness of the last case might suggest involvement of the cardiac plexus.

The absence of several symptoms removes these cases from the category of true angina pectoris—namely, marked pallor, coldness of the limbs, clammy perspiration, feeling of impending death, sensation of the necessity of keeping still, pain in the left arm, and increased and prolonged tension of the pulse. In addition there was no valvular lesion or evidence of degeneration of the vessels, and many observers consider one or other a *sine quâ non* of true angina. Their rebelliousness to treatment, and the fact that in once case sudden syncope occurred after the cessation of pain, point to the same conclusion. Dr. Walshe says that angina pectoris is rare before the 50th year, and excessively so before the 40th; also, that a first attack is rarely fatal. The influenza form of cardiac neuralgia would, therefore, appear to be at times more severe than the genuine "breast pang."

The indications for treatment are evident—rest and avoidance of heart strain. Where any premonitory symptoms are detected, as in the first case, digitalis and caffeine; and for the pain, morphine.

TWO CASES OF EMPYEMA QUICKLY FOLLOWING SIMPLE PLEURAL EFFUSIONS.

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In the treatment of effusions into the pleural cavity there is an essential difference to be made when the fluid is simple (sero-fibrinous) and when it is purulent, for whereas a simple effusion is generally absorbed without trouble, this result is extremely rare, and in view of the condition which is left, hardly to be wished for in empyema. In any case of pleural effusion, therefore, we should endeavour to ascertain whether the fluid is purulent or not, and for this purpose there is nothing so certain or satisfactory (if proper precautions be taken), as the insertion of an exploring syringe, provided a definite result is obtained from the puncture.

Having however proved by this means that an effusion is not purulent, the question arises: Do simple effusions tend to become purulent? The late Dr. Wilson Fox expressed the opinion that a simple pleurisy has no tendency to become purulent; Ashby and Wright² say that "an empyema is as a rule an empyema from the first," although in children there is naturally an increased liability for inflammatory effusions to become purulent. Dr. Drummond, of Newcastle, in a paper recently published,³ says that in an inquiry extending over two years he had not met with a single case of simple pleural effusion that went on to pus; on the other hand, Dr. Eustace Smith states⁴ "that the fluid quickly becomes turbid, and in children purulent." The two following cases, which occurred close together in my practice, support this opinion, and I believe the early detection of the purulent character of the effusions, before the formation of coagulated particles, contributed largely to the recovery in each case after only one aspiration.

CASE I.—W. T., aged 2½ years, a fairly well-nourished but delicate boy, first seen July 26th, 1891, with broncho-pneumonia of both lungs, the left being more affected than the right, respirations 45, pulse 120, and temperature 102.8°. The illness, which was not very severe, progressed satisfactorily until July 28th, when the child appeared more distressed, and the pulse and respiration, which had been greatly reduced in frequency, increased again. There had been no change in the treatment, nor did physical examination reveal any adequate cause for this. The next day there was increased dullness at the left base, and as this increased an ex-

¹ BRITISH MEDICAL JOURNAL, vol. ii, 1877, p. 752.

² The Diseases of Children.

³ BRITISH MEDICAL JOURNAL, vol. ii, 1891, p. 113.

⁴ A Treatise on Disease in Children.