On October 1st, the ninth day of disease, he was listless and apathetic. He was a bad colour, and the pulse remained frequent in spite of the fall of temperature. The liver edge could be felt 1 to 1½ inches below the costal margin, and there was epigastric tenderness. The pericardium was therefore again aspirated, and 200 c.cm. of thin purulent fluid removed. This was again followed by definite pericardial friction sounds. Later in the same day it was decided to drain the pericardium. This was opened (V.T.) under local anaesthesia (alypin), and one inch of the fifth left costal cartilage excised. The internal mammary vessels were retracted inwards, and the pericardium was exposed and incised. A considerable quantity of thin purulent fluid escaped. A drainage tube was inserted, passing behind the heart. After this he improved in his general condition. The pulse came down considerably. He began to eat his food. The wound discharged freely, the pus becoming much thicker. He also developed a cough which caused some pain. In spite of this, however, the temperature, which had been sub-normal at the time of the operation, continued the rise which had begun two days after this.

As the pyrexia continued it was determined to explore the left base, where dullness and diminished breath sounds persisted, and, pus being found, the left pleura was opened and drained on October 31st. The empyema was a very small one. After that the temperature fell to normal and he made a steady recovery. He went out on December 11th apparently in excellent health. The pleura had healed up some time before the pericardium ceased discharging.

At the present time, nearly a year after his attack, he is in robust health. He is able to carry on his work just as before. He plays football without any discomfort whatever. The apex beat is slightly outside the nipple line. The scar, which is about an inch to the right of this, is slightly retracted with each systole, but there is no retraction at the apex. There is no murmur.

As a rule pneumococcal pericarditis is diagnosed, or overlooked, as the case may be, at some time after pulmonary symptoms have established themselves in characteristic fashion. In this case it was fully developed, and causing mechanical distress by the third day. Whether there was also an initial infection of the lungs or not must remain uncertain, but if so it could hardly have preceded the pericardial infection. Neither the temperature nor the general appearance of the patient suggested a pneumococcal infection, although the presence of herpes and the pulse-respiration ratio corresponded to what is seen in a pul-

monary infection. There was a striking absence of cough during the first week, and indeed a pneumococcal infection was not suspected until the bacteriological report on the fluid was received.

In a very incomplete reference to the literature no definite case of a primary pneumococcal pericarditis of this severity has been found, or one in which surgical measures have been necessary on the fourth day of infection. Attention has of recent years been called to the possible danger of injury to the left pleura at operation, and Connor¹ reports a case in which this occurred, with the result that an empyema followed, which was not recognized during life, and proved

fatal. This may be the explanation of the empyema in our case, but on the other hand the presence of pleuro-pericardial friction on the left side prior to the first paracentesis shows that this pleura had not altogether escaped early infection.

There is a tendency in the literature to depreciate

the value of paracentesis in cases of pericardial effusion, many surgeons preferring the operation of pericardiectomy on account of possible injury to the heart or coronary vessels caused by the needle, but if a diagnosis of pericarditis with effusion is correct there should be little risk. Paracentesis is easy and quick, and relief, when the fluid has been drawn off, is immediate; the operation of draining the pericardium can then be carried out in more favourable circumstances. This was the procedure adopted in our case. The oblique sinus was not drained, as recommended by Ballance, and yet there was a complete recovery. Possibly there is less necessity for drainage of this space in cases met with in civilian practice than in those occurring as a result of war wounds. In support of this view one may instance the comparative success of arthrotomy for septic infections in civil practice, when compared with the unfavourable result of this operation in joint infections met with in war wounds.

REFERENCE.

¹ Connor: *Medical Record*, 1900, 77.

Chart showing pulse rate and temperature.

EARLY PREGNANCY AND EPITHELIOMATA.

BY

C. W. A. EMERY, M.B., B.Ch., B.A.O.,

WESTCLIFF-ON-SEA.

Do the conditions prevailing in the first three months of pregnancy assist in a study of the etiology and treatment of epitheliomata? It is in the hope of eliciting an answer to this question, or at any rate of inducing others to consider the suggestion contained therein, that I have written the following notes.

It has always seemed to me that a clue to the cause and treatment of epitheliomata through the conditions to be found in early pregnancy has never been given the care and thought to which it is entitled. There is no other normal condition in which one tissue, if it does not definitely attack, at least invades another, so that unless every possible clue fails we ought not easily to be turned aside and pronounce it a "blind."

The fundamental point to be kept in view in a study of any disease is that the normal human body is fully equipped to withstand disease, and it should be our duty to find out, in the first instance, why its resistance fails—not the nature of the attacking force. Remember that the tissues are fully equipped at the start, therefore the weakness is on their side. Working from this standpoint, we would have to allow that the body tissues are prepared to, and normally can, resist the growth of an epithelioma, and that if the

tissues are prepared against this particular growth they will resist any invading growth, whether malignant or not. This leads me to suggest that, as a result of this preparedness, the body will resist the normal invasion of the chorionic epithelium. The chorionic villi are able to invade the uterine wall, and so in some way this resistance has been overcome—but how? That the body shows signs of a reaction taking place is evidenced by the general malaise, nutritional disturbances, and sickness of pregnancy. We also know that lutein cells have appeared in the ovary after the ovum left it and these cells seem to grow in proportion to the rate of growth of the placenta, and that when the placenta ceases to grow, at the third month, these lutein cells also cease to grow. That there is some connexion between these two points is admitted. It is also generally admitted to-day that the function of the corpus luteum is that of an internal secreting gland. When Fränkel found that destruction of the corpus luteum in pregnant rabbits resulted in abortion, he concluded that it produced a secretion which maintained the implantation and growth of the ovum in utero. I suggest that this might be modified by saying that the secretion produces a body reaction, the result of which is to provide an antibody to the normal "body resistance to any invasion," and so the chorionic epithelium is able to maintain its hold, and that Fränkel's experiment allowed the normal body resistance full play so that the chorionic epithelium was repelled.

Pregnancy sickness is evidence of this auto-inoculation. This also explains the abnormal lutein activity found in

chorionic epithelioma. Overproduction of secretion results in completely antidoting resistance, and so invasion continues wildly. Underaction or inaction is a cause of sterility. Further, the action of lutein secretion in lowering or countering body resistance explains the preponderance in women of cancer cases under the age of 40, as compared with men under that age. The body must be primarily regarded as fully equipped, yet there is a weakness here and a clue to general resistance may follow deduction. The epithelioid character of the lutein cells and the fact that they contain a lipid substance is important when we remember that the body is prepared to resist an attack by epithelial cells. My meaning, however, will be clearer if I diverge for a moment.

It seems a very important point to me that in the body generally we do not find an epithelioma growing where there is an unadulterated layer of fat below epithelium. I use the word "unadulterated" advisedly. The commonest sites for epitheliomata are to be found where the epithelium comes into direct contact with mixed muscular or fibromuscular tissue—for example, in lip, tongue, cervix uteri, and following dissolution of continuity of a fatty layer by wounds, ulcers, burns, etc. A case of cancer of the abdominal wall would be rare in this country, and yet among the women in India who carry burning incense next their abdominal skin, and so get large sores with a breaking down of the fat layer, it is common. It must be noted, too, that the only area which the body normally prepares for the reception of invading cells (chorionic epithelium) does not possess a fat layer. *Fat, therefore, is protective.* Muscular tissue, especially mixed muscular and fibrous tissue, has some property which makes it a very receptive medium for proliferating epithelium.

That epithelial and lipid substances come so much into evidence in early pregnancy and produce some form of reaction on the part of the body, and that the fat of the body ordinarily exercises a protective action, are very important points. When I refer to "unadulterated" fat I mean fat which is not interlaced with elements such as I have said form suitable receptive media—for example, in the breasts. The action of fat may be purely mechanical: it may act as a protective blanket—we have evidence of germs taking advantage of this in the fatty envelope of the tubercle bacillus; and, to digress again, a method of attack on this envelope, preparatory to antituberculous treatment, is here indicated—but the close relationship of fat and epithelium in the body generally and the epithelial and lipid nature of the lutein cells suggests that either fat alone, or some interaction between the two, provides the blood with its resisting powers; or, again, that the fat in itself provides both mechanical protection and the resisting powers to the blood normally, and that, in a finer and more intimate form, in the lutein cell epithelial and lipid substances evolve an antidote to that resistance.

I do not think that impregnation of the ovum by the spermatozoon and subsequent events are in any way analogous to a proliferating epithelial cell, as I have seen suggested. An analogy would require the production of innumerable ova, not the much more highly specialized and complex result we get. The "microbe" is the epithelial cell itself, and its proliferation is the result of one of known methods. If, while proliferating, this cell is in contact with mixed muscular tissue, it is in contact with a new food, and sometimes intensive cultivation results. The epithelial cell, like any other living entity, will go in search of nourishing food, and, finding it, will greedily absorb it. This food being a vital constituent of muscle food, starvation and exhaustion result, with subsequent death.

The preventive treatment of cancer is of extreme importance, and is along the lines of greater internal and external body hygiene. An impaired drainage system must result in a lessened vitality of the body cells, and consequently impaired functional and resisting activity. Further treatment would follow a closer study of the exact uses of fat and isolation of the agent capable of producing increased body resistance, in conjunction with early diagnosis and its use after operation.

In conclusion, then, my answer to the question at the beginning of this paper would be in the affirmative.

British Medical Association.

PROCEEDINGS OF SECTIONS AT THE ANNUAL MEETING, BRADFORD, 1924.

SECTION OF NEUROLOGY AND PSYCHOLOGICAL MEDICINE.

T. GRAINGER STEWART, M.D., F.R.C.P., President.

DISCUSSION ON THE DIAGNOSIS AND TREATMENT OF COMPRESSION PARAPLEGIA.

OPENING PAPER

BY

ANTHONY FEILING, M.D., F.R.C.P.,

Assistant Physician to St. George's Hospital; Physician to the Hospital for Epilepsy and Paralysis, Maida Vale, and to the Royal National Orthopaedic Hospital.

THE subject for our discussion to-day, the diagnosis and treatment of compression paraplegia, is one which must appeal to the general practitioner as well as to the neurologist and the surgeon. Any form of paraplegia which is amenable to surgical intervention demands careful attention, and an early and accurate diagnosis is an essential preliminary to successful treatment.

We must first define what we mean by the term "compression paraplegia." For the purpose of this discussion I will define it as follows: "A pathological condition where a localized lesion causes pressure either on the whole contents of the spinal theca or on the spinal cord alone, with or without involvement of the nerve roots, in such a way that the functions of the spinal cord become progressively damaged."

In order to keep this subject within reasonable limits I am deliberately excluding from consideration cases of sudden or acute lesions of the spinal cord, caused by trauma or acute myelitis of any kind.

It is not of academic interest only to pause for a moment and inquire exactly how the functions of the spinal cord suffer—that is, how the symptoms are produced. For symptoms will often, indeed generally, arise some time before the cord is actually mechanically compressed or squeezed. The conditions are very different from those obtaining in, say, cases of gunshot wounds of the cord, with suddenly produced lesions which may entirely destroy the cord over one limited area. Here the interruption of function is complete physiologically and often anatomically as well. Ascending and descending degeneration of nerve tracts follow, and subsequent recovery depends primarily on the extent to which such secondary degenerations have occurred. But the morbid anatomy, as well as the conditions found at operations during life, show very conclusively that no such complete anatomical lesions exist in the majority of cases of compression paraplegia. Witness also the recovery, often complete recovery, which may take place after a successful intervention.

Microscopic examinations again demonstrate the exceedingly small amount of secondary degeneration which may be found even in cases which have shown a complete paralysis during life. Obviously, therefore, some factor must be at work other than the mere mechanical destruction of nervous tissues. This factor is almost certainly interference with the circulation in the cord. Probably the veins suffer first in most cases, with the production of a state of venous congestion. Later the arteries suffer, and an ischaemia or anaemia of the cord occurs, which, though insufficient to cause permanent degenerative changes, is yet enough to produce very considerable, even complete, temporary loss of function.

There is yet another factor to be considered, and one which is sometimes of great importance. This is the fluid