that in cases of cerebral tumour it may be possible by cytological examinations of the cerebro-spinal fluid to distinguish between tumours of tuberculous or syphilitic origin on the one hand and those of different formation on the other. In a tuberculous or syphilitic tumour I should expect to find a lymphocytosis and no such change in a tumour of another source. I have not had an opportunity to verify this. Again, in an ascitic fluid a lymphocytosis points to the effusion being due to tuberculosis.

Predominancy of polymorphonuclear cells.—An excess of polymorphonuclear cells in any serous fluid points to it being of an acute inflammatory origin. These cells can only have been derived from the blood by diapedesis through the vessel walls. Dr. Ross has tabulated 12 cases of acute infective conditions in which he examined the effusions and in all of these he found a very high percentage of polymorphonuclear cells. In two cases of post-basic meningitis these were the principal cells present. It has been proved by many observers that a large number of polymorphonuclear cells in a serous effusion or cerebro-spinal fluid occurs in acute infective conditions. When examining such exudations containing numerous polymorphonuclear cells cytological counts are in many cases not necessary, for the micro-organisms producing the condition are very frequently found in the films. The following are typical counts of the cells in two cases of pleuritic effusion caused by the pneumococcus of Fraenkel :—

Small lymphocytes	...	...	22.4	...	...	15.0
Polymorphonuclear cells	...	...	64.4	...	...	81.2
Endothelial cells	...	...	13.2	...	...	3.8

Only once have I examined cytologically a pericardial effusion and this was one due to the rheumatic diplococcus. The count was as follows: polymorphonuclear cells, 93.2; and small lymphocytes, 6.8 per cent.

The following are examples of cytological counts in four cases of cerebro-spinal meningitis, showing the great predominance of polymorphonuclear cells.

Polymorphonuclear cells	...	...	84.0	...	79.1	...	85.7	...	74.6
Small lymphocytes	...	...	15.6	...	18.6	...	11.9	...	24.0
Endothelial cells	...	...	0.0	...	0.6	...	0.0	...	0.7
Degenerated or cells not classified	...	...	0.4	...	1.7	...	2.4	...	0.7

In two cases of cerebral abscess in which I examined the cerebro-spinal fluid cytologically the differential counts were :—

Polymorphonuclear cells	...	...	66.4	...	97.0
Small lymphocytes	...	...	30.5	...	2.4
Degenerated or cells not classified	...	...	3.1	...	0.4
Endothelial cells	...	...	0.0	...	0.2

The cytodiagnosis in these two cases was of acute meningitis of non-tuberculous origin. With the help of the clinical history of the cases a diagnosis of cerebral abscess was made. In the first case the patient was operated upon by Mr. George Heaton and a large abscess was found in the right temporosphenoidal lobe, secondary to ear disease. In the second case the presence of a cerebral abscess was not proved until a post-mortem examination was performed.

Predominancy of endothelial cells.—When there is a large percentage of endothelial cells in a cytological count it indicates that the effusion is mechanical in origin. Bymechnical effusion is meant such a one as occurs in serous cavities as a part of a general oedema or in the peritoneal cavity as a result of portal obstruction. Endothelial cells are nearly always present in small quantities in all serous effusions, but it is only when they are in large numbers relative to the other cells that they are of any diagnostic value. In a mechanical effusion the cells present are usually not nearly so numerous as are found in an exudation of acute inflammatory origin and are even much fewer than those found in an exudation due to tuberculosis or syphilis. In two cases, however, I have found them in very large numbers. Sometimes there are seen collections of endothelial cells as though they had been shredded off the serous membranes. These collections of cells or plaques have before now been mistaken for portions of malignant growth.

It has been definitely proved by the observations of Dr. Ross that numerous endothelial cells indicate a mechanical origin for the effusions, notwithstanding the presence of polymorphonuclear cells and small lymphocytes. In ascites due to cirrhosis of the liver numerous endothelial cells are found and the following are typical counts from two cases :—

Endothelial cells	...	...	96.4	...	...	98.8
Small lymphocytes	...	...	3.4	...	...	0.2
Polymorphonuclear cells	...	...	0.2	...	...	0.4
Unclassified cells	...	...	0.0	...	...	0.6

The difference between an ascites due to cirrhosis of the liver and that due to tuberculous peritonitis can usually be ascertained by a cytological examination. In the former case there would be an excess of endothelial cells and in the latter an excess of small lymphocytes. As tuberculosis of the peritoneum does sometimes occur as a complication of cirrhosis of the liver it may be important that the ascitic fluid be examined.

There is an excess in endothelial cells in all serous effusions occurring in general oedema, but whether this is the case in the cerebro-spinal fluid under such conditions I do not know. I have never examined the cerebro-spinal fluid cytologically in a case of general oedema during life but in one case examined after death I found that the endothelial cells were over 93 per cent. I do not, however, attach importance to this observation as many of the endothelial cells were probably shed into the fluid after death.

Effusions due to malignant disease.—A few observers have stated that it is possible to diagnose the presence of a malignant tumour by a cytological examination of the effusion. This they do by recognising a few cells in the fluid as having been detached from the malignant growth. The recognition of isolated cells in this way is, however, practically impossible and in those cases where a cytodiagnosis of malignant disease has been made it is very probable that the cells considered to be of malignant origin were really endothelial cells after all. Such cells usually predominate in effusions which are caused mechanically by the presence of a neoplasm and often appear in plaques. Unless there be a large collection of the malignant cells in the fluid it must be impossible to diagnose the condition by a cytological examination.

Without doubt a cytological examination of a serous effusion or a cerebro-spinal fluid may be of great help in diagnosis. A diagnosis should not, however, be made on the cytological finding alone, and the examination of the cells in the fluid must only be considered as an additional help in diagnosis. As a general rule it can be relied upon, and only in a very few cases does it mislead. In a very rare case of acute tuberculous cerebro-spinal meningitis published recently in THE LANCET by Dr. T. Stacey Wilson and Dr. James Miller,² the cytological examination pointed to the condition being of acute inflammatory origin and not tuberculous, on account of the large percentage of polymorphonuclear cells present. The appearances at the post-mortem examination were all those found in the cerebro-spinal meningitis due to the diplococcus of Weichselbaum, but on microscopical examination numerous tubercle bacilli were found to be present.

Conclusions.—1. Effusions of tuberculous origin contain a large percentage of small lymphocytes, ranging in my series from 59 to 100 per cent. 2. Effusions of acute inflammatory origin contain a large percentage of polymorphonuclear cells, ranging from 64 to 97 per cent. 3. Mechanical effusions contain chiefly endothelial cells, the highest count being 98.8 per cent. 4. Effusions due to malignant disease can rarely be diagnosed by cytological methods alone, but when such a condition is suspected to be present the predominance of endothelial cells would greatly support that view.

Birmingham.

ACID INTOXICATION FOLLOWING ETHYL-CHLORIDE ANÆSTHESIA.

BY H. H. B. CUNNINGHAM, M.D. BRUX.,
F.R.C.S. IREL., M.R.C.S. ENG.,

OPHTHALMIC AND AURAL SURGEON TO THE ULSTER HOSPITAL FOR
CHILDREN AND WOMEN, BELFAST; LATE SENIOR CLINICAL
ASSISTANT TO THE ROYAL EAR HOSPITAL, LONDON.

ACID intoxication following on general anæsthesia has only been described comparatively recently, but so far as I am aware it has only been recorded as an after-effect of chloroform anæsthesia. Thus in 1894 Dr. Leonard G. Guthrie first drew attention to this subject by his paper on Some Fatal After-effects of Chloroform on Children published in THE LANCET of Jan. 27th (p. 193) and Feb. 3rd (p. 257) of that year. In the Transactions of the Ulster Medical Society for 1906-07 Mr. Robert Campbell, after mentioning the papers published on this interesting subject,

describes in detail three cases with the post-mortem appearances. But in this communication every case described followed chloroform administration. My case shortly is as follows:—

A well-developed, bright-looking, and apparently healthy girl, aged six years, was admitted to hospital for deafness owing to the presence of adenoids. On the morning of Nov. 13th, 1907, after having been prepared in the usual manner, ethyl chloride was administered and the adenoids were removed, the operation, including administration of the anæsthetic, taking about a minute; the child recovered completely and was then placed in bed. Towards evening she vomited twice but slept well during the night and no vomiting occurred. On the next day she complained of severe headache and of nausea. She retched a good deal during the day and vomited a few minutes after taking any food, so she was given some bismuth mixture in the evening. During the night she slept fairly well but vomited once, whereupon the medicine was repeated. On the 15th she complained of violent frontal headache and vomited immediately after taking anything by the mouth. Towards evening the pulse became rapid and small and the child felt very cold, so hot bottles were placed in the bed. During the night she slept fairly well but vomited twice after drinking a little milk. On the 16th the aspect of the child had completely altered during the past two days. She now lay in bed with her knees drawn up, taking no notice of anything, but answering when spoken to and complaining of great frontal headache and of pain in the abdomen; her eyes were very sunken and her face was somewhat pale, worn-looking, and drawn; in fact, her aspect resembled that of a patient who had suffered from some severe illness, such as typhoid fever, for some weeks. The child was very cold, her tongue was coated and dry, her pulse was quite feeble, very rapid and irregular; in fact, she was very ill. She had been given an enema simplex and was receiving bismuth mixture every four hours; she retained no food, vomiting immediately anything that she swallowed. It had been thought up to now that probably she was developing some intercurrent disease. However, her aspect and the continual vomiting made me suspect acid intoxication, so a sample of urine was obtained, which Dr. T. Houston kindly tested and found it to contain diacetic acid, thus confirming the diagnosis. She was now given rectal injections of 1 drachm of bicarbonate of sodium in 2 ounces of warm water every four hours, and during the night, also bismuth mixture and a little peptonised milk and water, this and the mixture containing 10 grains of bicarbonate of sodium to the dose, but both were vomited immediately after being drunk and so were stopped. On the 17th the child was distinctly better, the worn aspect had vanished, and the eyes did not appear so sunken. She looked brighter and felt easier and had no headache or pain in the abdomen; the pulse was still rapid but regular and better in quality. During the early part of the night she had vomited twice, but not since she was not given any food by the mouth, receiving rectal feeds of 4 ounces of peptonised milk containing 20 grains of bicarbonate of sodium every four hours. After 6 P.M. she was given peptonised milk in half-ounce quantities by the mouth which she retained. This was therefore gradually increased in quantity as no vomiting occurred, the rectal feeds being continued during the night. On the 18th there was no vomiting, the child looked brighter and better, and did not complain of pain anywhere. The pulse was slower, steadier, and improving in quality; the tongue was becoming clean. The rectal feeding was now discontinued, the patient being given 5 ounces of peptonised milk every two hours by the mouth; in the evening this was replaced by ordinary milk. On the 19th the child was improving rapidly; there was no vomiting and the pulse was normal. The urine tested on this day was found to be free from diacetic acid. The subsequent history was uneventful. The temperature during the illness never rose above $99^{\circ} 8^{\circ} \text{F}$., nor fell below normal.

This, then, appears to be a case of acid intoxication following ethyl-chloride anæsthesia, which when recognised and treated appropriately had a fortunate termination, though on the third day after the operation the little patient was so ill that this, coupled with the fatal terminations in the recorded cases following chloroform administration, made one give a very gloomy prognosis. In view of the able manner in which Mr. Campbell has described the whole subject of chloroform poisoning I will refer the reader to his paper and content myself by quoting as an explanation why

acid intoxication should follow ethyl-chloride administration the suggestion made by Dr. V. G. L. Fielden, the anæsthetist to the hospital, that the halogen group occurs both in chloroform and in ethyl chloride. In conclusion, I have to thank Miss Tate, the matron, for many of the notes on this case.

Belfast.

A NOTE ON THE DEVELOPMENT OF SECONDARY NODULES IN SUTURE SCARS AFTER OPERATIONS FOR THE REMOVAL OF CANCER.

By C. W. MANSELL MOULLIN, F.R.C.S. ENG.,

SENIOR SURGEON AND LECTURER ON SURGERY AT THE LONDON HOSPITAL.

IN THE LANCET of Nov. 9th, 1907, Mr. Charles Ryall called the attention of the profession to the very grave risk of cancer infection taking place during the performance of operations for the removal of cancerous growths, and instanced many cases in which it seemed to be obvious that such infection had occurred. One of the examples brought forward was the well-known frequent recurrence of the disease in the suture cicatrices after operations upon the breast, and several striking instances were mentioned in which infection had taken place around a laparotomy wound even when the operation had been merely of an exploratory character. A case that has been recently under my care illustrates this point well, but at the same time suggests that the mode of transference of the cancer infection is not of quite such a simple character as is usually believed. The sutures must bear a part of the blame, perhaps the greater part; but there is something more than the mere conveyance of a cancer cell from one spot to another by the needle or suture employed.

The patient, a man 48 years of age, was sent to me with the history that he had suffered from chronic gastric ulcer for the last 18 years, and that lately, in spite of medical treatment, the attacks of pain and vomiting had become more frequent. Cancer had supervened upon the old ulcer, as it so often does, and at the operation a tight stricture of the pylorus was found with some disseminated nodules scattered over the serous surface of the stomach. Posterior transmesocolic gastro-enterostomy was performed and the patient made an uneventful recovery. Six months later I saw him again. He was much better so far as pain was concerned and was considerably stouter, but there was a double row of cancer nodules on either side of the laparotomy wound. Two sets of sutures had been inserted: one, of stout catgut, interrupted, passing through all the layers of the abdominal wall except the skin; the other, of finer catgut, continuous, through the skin only. Practically all the suture points of the former series were infected; none of the latter. It was evident that cancer cells from the peritoneal cavity had invaded the wall wherever the parietal peritoneum had been pierced by a suture. The sutures had not carried the cancer germs in, for they had never been near the growth; but either by perforating the serous layer or by acting as irritants they had helped the development of the invading germs to such an extent that each point had become a cancer nodule. The immediate source of infection was in all probability the nodules noted on the serous coat of the stomach, which were constantly rubbing against the anterior abdominal wall. The line of the laparotomy incision itself seemed to be intact between the two rows of nodules.

The same explanation, it seems to me, is the more rational one in those cases in which cancer recurs at the suture points after operations for mammary carcinoma. It is not that the cancer germs are carried in by the needle or suture mechanically at the time of the operation and left to grow in the puncture made, but that they are more widely distributed in the surrounding tissue interstices than is apparent at the time, and only develop, or perhaps develop first, at those points where the resistance of the tissues is lowered by the presence of an irritant such as a suture.

That healthy tissues do possess some power of resistance to the growth of invading cancer germs is obvious. The distribution of cancer germs from a primary focus must begin at a very early date, but it is a long time before secondary