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Paraganglioma of the Vermiform Appendix

F. W. Lunev, M.D.

St. Joseph's Hospital, London, Ontario

Paragangliomata are tumors of chromaffin tissue developing from cells that are closely associated with the sympathetic nervous system and having their origin in neural ectoderm.

Early in the development of the embryo certain cells of the neural ectoderm bud from the neural crest and differentiate to form ultimately the sympathetic nervous system. These cells that have budded from the neural ectoderm later again differentiate (16 mm. embryo) to form first the sympathoblast which ultimately forms adult sympathetic ganglion cells and, second, the chromophiloblast which is the fore-runner of the chromaffin cell. These two types of cells are found closely associated. In the wall of the intestine the chromaffin cells occur in small groups among the ganglion cells in Auerbach's plexus in the muscularis and in Meissner's plexus in the submucosa. Because of their association with ganglionic cells they are known as paraganglia. Similar single cells of chromophilic character, the cells of Schmidt and Kull, occur in the mucosa throughout the gastro-intestinal tract between the columnar cells of the crypts of Lieberkühn. The genesis of chromaffin tissue may be more clearly understood from the following diagram (modified from Delafield and Prudden):

DEVELOPMENT OF THE CHROMAFFIN CELL

Neural Crest.
(NEUROCYTE)

SYMPATHO-CHROMOPHIL BLASTEMA
(Primary Sympathetic Cell)
(16 mm. embryo)

SYMPATHOBLAST

SYMPATHETIC GANGLION CELL

CHROMOPHILOBLAST
(18 mm. embryo)

CHROMAFFIN CELL

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The affinity of these cells for chrome salts was first pointed out by Oberndorfer during his discussion of a paper by Saltykov before the German Pathological Society in 1912. In 1914 Gosset and Masson studied these cells after silver nitrate impregnation and observed that granules having an affinity for the silver salt always existed in the depth of the cell between the nucleus and the basement membrane. Because of their affinity for silver salts, he spoke of them as argentaffin cells.

In a recent article entitled "Argentaffin Tumors of the Appendix and Small Intestine" Forbus gives a complete résumé of the literature on these tumors to which the reader is referred for the early works on this subject. The true nature of tumors originating in the chromaffin cells of the intestinal tract has not been widely recognized and many different opinions at different times have been advanced. They have been variously looked upon as (a) endotheliomata (Glasebrook) (b) basal cell carcinomata (Burckhardt); (c) tumors originating in pancreatic rests (Saltykov); and (d) spheroidal cell carcinomata. Because they usually occur in young individuals and run a benign course yet show microscopic features of a malignant infiltrating growth, they were named carcinoids by Oberndorfer to distinguish them from true carcinomata. This term has received wide acceptance because it is descriptive, yet noncommittal.

Hübbschmann in 1910 first suggested that the carcinoids, because of their yellow color, might have origin in the chromaffin cells of the intestinal wall. In 1914 Gosset and Masson made an elaborate study of the chromaffin tissue of the gastro-intestinal tract including the carcinoids and concluded that the cells which form the tumor are practically identical with the cells of the chromaffin tissue of the paraganglia which are filled with argentaffin granules.

The most recent comprehensive report corroborating Gosset and Masson's work is that of Forbus above referred to. He reports six cases and concludes that argentaffin tumors of the vermiform appendix have their origin in the cells of Kultschitsky and Schmidt of the crypts of Lieberkühn.

These tumors, while most commonly occurring in the appendix, are found throughout the course of the small intestine as small, discrete, single or multiple tumors. Lubarsch was the first to make any important contribution regarding them. He described two cases in which, at autopsy, were found multiple small tumors of the ileum. Because of their microscopic appearance he called them primary carcinomata of the ileum although they were discovered accidentally and did not appear to contribute to the patient's death.

The tumors in themselves produce no symptoms unless they develop to an unusual size, which is very rare. In the small intestine they are incidentally found during an exploratory laparotomy. In the appendix, however, they act as a foreign body and often produce local symptoms of a chronic or an acute appendicitis. They occur at any
age but are usually found in young adults. Even though they are found in the fourth or fifth decade or later, there is considerable evidence to indicate that they develop early in life and persist for years, during which time they undergo little if any change. This is suggested by the fact that in the appendix where they set up secondary inflammatory changes they are in most cases found in the young; on the other hand, in the small intestine, where they produce no symptoms or in the appendix where they sometimes exist without producing inflammatory changes for a considerable time, they are found in later life.

They comprise a new growth of most unusual type. In the gross they are recognized by their deep yellow or golden color. In the appendix, often one's attention is attracted to a small, nodular, firm swelling at the tip or in the distal half of the organ. Sometimes no definite tumor mass is visible but on section the masses of golden colored cells can be seen extending along the mucous and submucous coats towards the proximal end of the organ or may occur as a solid mass of cells completely replacing mucosa and submucosa. Unless serial transverse sections are made, many early cases or others in which the growth is very small may be overlooked. In some instances where there is extensive involvement of the appendix groups of similar cells may be observed in the meso-appendix.

Microscopically the tumor may be found at any point in the appendix. In those cases where there is nothing in the gross examination to suggest tumor other than the peculiar golden color, there being no deformity of the organ, the cells are found in groups in the submucosa or may completely replace both mucosa and submucosa. On the other hand, those cases with a definite bulbous swelling show as a rule extensive infiltration of the muscularis and subserosa. In some instances the growth may be seen to compress and displace the mucosa to one side rather than infiltrate it and replace it. The tumor consists of small or large groups of cells, each group being surrounded by a finely reticular stroma, or a thicker, more fibrous and hyaline connective tissue giving the growth a scirrhous appearance. In some instances, particularly in those cases where the muscularis is involved, the tumor occurs as fine or coarse columns or strands of cells infiltrating and extending along between the muscle bundles.

The morphology of the tumor cells depends upon the surrounding connective tissue reaction. When the stroma is scant and extends in thin bands to surround large and small groups of cells, they appear comparatively large and all adjoining cells have a definitely uniform appearance. The cells are rounded or irregularly polyhedral in shape and usually the cytoplasm is pale and not clearly outlined. The nuclei, on the other hand, stand out prominently. They are comparatively large, round and of uniform size and appearance and show no evidence of mitoses. In comparison with the cytoplasm, the nuclei are clearly circumscribed and deeply basophilic.

In those cases in which there is a great proliferation of the stroma the cells occur in smaller groups which are not clearly outlined and the
individual cells are smaller and appear compressed and distorted. In the muscularis the cords of cells are exceedingly fine, the cells are flattened and often little more than the nucleus can be seen.

During routine examinations in the past few years six cases have been studied. The histories of three representative cases follow:

Case 1—Patient, Miss F. S., age 20. During the summer of 1925 had an attack of abdominal pain. No diagnosis was made and no treatment administered. A physical examination made at this time showed no evidence of tuberculosis. The blood pressure was: Systolic 128, Diastolic 74. Since this attack she had complained of a dull ache in the lower right quadrant which was relieved by supporting the side. During mid-February, 1926, following an attack of diffuse abdominal pain with vomiting, which lasted about three hours, she developed acute pain with tenderness in the right iliac region with gradual rise in temperature and pulse rate. The temperature at time of operation, 20 hours after onset, was 102° F. At operation the distal 2½ cm. of the appendix was adherent to neighboring structures and covered with omentum. The abdominal cavity was otherwise normal.

Macroscopic examination.—The appendix is 6 cm. in length. The peritoneum is thickened and towards the tip is quite opaque and dull and covered with a greyish yellow film of exudate. The peritoneal vessels are injected and appear quite prominent. Fatty omentum is adherent at the tip. The thickness varies slightly between 5 and 7 mm., the organ being slightly swollen in its distal portion. There is, however, nothing in the shape of the appendix to excite suspicion of new growth. The organ was incised transversely into thin section. The lumen is found small and slit-like and is obliterated in the distal portion. The mucosa is atrophied while the submucosa is much thickened and of uniform greyish color except for one minute area recognizable in only one of the thin sections, in which there is noted a group of bright orange-colored cells. This group of cells is exceedingly small, not greater than 1 mm. in any diameter, and produces no alteration in the shape of the appendix.

Microscopic examination.—Sections at different levels of the appendix were studied. In the proximal portion there are evidences of a mild chronic inflammatory reaction; in the distal portion there is a picture of an acute inflammatory process with involvement of the entire wall including the peritoneum. The lumen is obliterated and the mucosa atrophied, there being but a few isolated, distorted and compressed groups of gland acini at the centre of the tissue. The tumor is very small and situated in the submucosa just within the muscle layers. The whole growth can be nicely contained in one low power microscopic field and is demonstrated in only one of several blocks of tissue cut. There is no invasion of the muscularis. The appearance would suggest that the growth has developed from the paraganglion cells associated with Meissner's submucous plexus. These cells have the characteristic appearance previously described. Fine strands of connective tissue
stroma separate the cells into small and large groups giving to the whole an alveolar-like appearance. The outline of the cells is not distinct and the cytoplasm is pale and finely reticular in structure. The nuclei are large, centrally situated, clearly outlined and of uniform size and appearance. There are no evidences of mitoses.

Subsequent history.—The patient made an uneventful recovery, returned to work in one month’s time and has gained slightly in weight.

Case 2.—Patient, Mrs. M. B., age 45. This patient has had no definite attacks of appendicitis but has had slight pain and tenderness in the right lower quadrant for some time. She complained largely of epigastric symptoms with eructations of gas two or three hours after meals. Lassitude, weakness and constipation were also prominent symptoms. The patient has no signs or symptoms of tuberculosis. The blood pressure is 130. At operation in mid-January the appendix was found bound down with firm adhesions to the neighboring structures. Nothing unusual was noted in the appendix at the time of operation but it was submitted for the routine examination. Before the incision was closed, general examination was made and nothing abnormal noted in the abdomen.

Macroscopical examination.—The organ is 4 cm. in length. The peritoneum is thickened, roughened and covered with fine adult adhesions. It is greatly constricted at its centre, there being only a fine thread connecting proximal and distal portions. Except for the constriction, the organ is of uniform thickness, 6 mm. There is nothing to suggest the formation of a new growth of tissue. On section, the lumen is found obliterated in both portions and in the distal part the cut surface has a yellowish fatty appearance. This yellowish color suggested fat and did not excite suspicion of proliferating chromaffin cells.

Microscopical examination.—The lumen in the distal portion of the organ is completely obliterated, the only remnant to suggest mucosa being a small group of cent rally situated lymphoid cells. Replacing the mucosa and infiltrating the submucosa are small groups of cells of typical morphology embedded in an abundant fibro- reticular stroma. The cells occur in solid masses, although there is, in some areas, some suggestion of atypical acinus arrangement. There is no infiltration of the muscularis. The peritoneum is slightly thickened.

Subsequent history.—The patient had a rapid, uneventful convalescence. Since operation the gastric disturbance has subsided, the patient has put on weight and feels much improved.

Case 3.—Miss J., age 28. In early January, 1926, the patient developed symptoms of acute appendicitis. Vomiting was an early and persistent symptom. Leucocytic count, 14,000. The patient’s blood pressure was normal. There were no clinical evidences of tuberculosis. At operation 36 hours after onset, an appendix showing all the earmarks of acute inflammation was removed. The surgeon stated that there was nothing unusual noted at the time of operation although he
observed that the appendix at its base appeared to be unduly enlarged. The wall of the cecum was normal and there were no enlarged lymph glands noted.

Macroscopical examination.—The appendix is 6 cm. in length and varies in thickness from 10 mm. at the tip to 12 mm. at the base. The peritoneum is thickened, roughened and dull and at the base infiltrated with purulent exudate. The subperitoneal vessels are prominent, being particularly thickened and tortuous near the distal end. There is no bulbous enlargement at any point to attract one's attention to a possible new growth. The gross picture before incision is one of acute inflammation. Attached to the appendix is a thick, firm, fatty mesentery which extends from tip to base. On section, a large, rounded, bright golden yellow mass 8 mm. in width is found at the tip and appears to completely replace mucosa and submucosa. It extends towards the base for a distance of $2\frac{1}{2}$ cm., gradually becoming smaller and rather abruptly disappearing. The lumen is completely obliterated by the mass. The tumor is clearly circumscribed and the muscle coats easily outlined, except for one small area near the tip where the growth is seen to infiltrate the entire appendiceal wall and extend into the mesoappendix where an irregularly shaped mass of tumor tissue is found $\frac{3}{4}$ cm. in diameter. There are no evidences of any tumor in the portion of appendix and mesentery approximating the cecum.

Microscopic examination.—The appendix shows a moderate diffuse inflammatory reaction. Except at the tip where there are evidences of extension of the tumor to the mesentery, the tumor is confined to the mucosa, the submucosa appearing thin and compressed occurring as a thin fibrous band just within the muscularis. The growth is very cellular, the cells being supported by a thin delicate stroma carrying small blood vessels and branching to surround large and small groups of cells giving an appearance of atypical alveoli. The cells are comparatively large, pale and of uniform size with ill-defined outlines. The nuclei are clearly circumscribed, rounded and of uniform appearance. There are no evidences of mitoses.

Subsequent history.—The patient made a rapid, uneventful recovery and since operation has been in excellent health.

DISCUSSION

Fifteen years ago it was pointed out by Hübschmann that these peculiar new growths had their origin in chromaffin tissue. This origin, however, is apparently not widely known or has not been accepted, because few, if any, of the present-day authors refer to the chromaffin origin of the so-called carcinoids and within the last few years two articles at least have been published in which the writers described them as carcinomata.

Forbus in calling them argentaffin tumors adheres to the nomenclature of Gosset and Masson. While this terminology is certainly descriptive, emphasizing one of the important features of the cell, it
Paraganglioma of the Vermiform Appendix

Paraganglioma does not in any sense indicate the genesis of the tumor. The term "Paraganglioma" would appear to be more appropriate because it not only indicates relationship with the sympathetic nervous system but links up these tumors with others of similar nature that have been heretofore described as paragangliomata in the carotid gland.

Gosset and Masson believe that these tumors all arise from certain cells described by Kultschitsky and Schmidt which occur in the mucous membrane of the appendix. Since chromaffin cells occur not only in the mucosa but also in the submucosa and muscularis, it seems quite possible that paragangliomata might arise elsewhere than in the mucous membrane. Case 1 is an example in which there are no evidences of origin in the mucosa. In this case, which appears to be an early one, the minute mass of cells is entirely confined within the submucosa and although serial sections were made no relationship to mucosa could be ascertained. In case 3, the tumor definitely appears to have arisen in the mucous membrane. It completely replaces the mucosa and at all but one point appears confined within the muscularis mucosae. In case 2, the extreme atrophy and scarring of the mucosa and the extensive diffuse infiltration of the tumor cells make it impossible to suggest the probable origin of the new growth.

Whether these growths should be looked upon as true neoplasms requires some consideration. They do not bear all the ear-marks of either malignant or benign tumors. Being non-encapsulated and showing definite microscopic infiltrative features one would expect the tumor to run a malignant course but in all authentic cases the clinical course is benign and the age incidence is far below that in which malignant epithelial tumors usually occur. It has been pointed out that all chromaffin tissue functions similarly to the medulla of the adrenal. If one could offer any reason why there might be a local demand for an increased amount of adrenalin, these masses of proliferating cells might be considered as simple hyperplasias. There is no evidence that they develop in individuals with low blood pressure although Primrose reports two cases which were associated with tuberculosis.

In the three cases here described there was no clinical history of tuberculosis and the blood pressure was normal.

Paragangliomata of the appendix are not common. The incidence is variously computed to be between .4% and .6% of appendices removed, although this percentage, no doubt, will be increased as standardization of hospitals becomes more generalized and the requirement for routine examination of all tissues is practised. As before mentioned, an acute appendicitis may readily mask one of these tumors unless the appendix is carefully cut in thin transections. Appendices that are opened longitudinally may fail to reveal small growths that occur in the submucosa; the six cases here referred to were found in the routine examination of 720 appendices. This is equivalent to .83%. 
Paragangliomata are tumors of chromaffin tissue and thus develop from neuro-epithelium. Because of the close association of sympathetic ganglion cells and the paraganglia, they may occur at any point supplied with sympathetic fibres. These tumors have occurred in the carotid gland, in the medulla of the adrenal and, under the name carcinoids and argentaffin tumors, have been described in the gastro-intestinal tract.

In the intestine, they occur most frequently in the appendix where they produce symptoms of an acute or chronic appendicitis.

Paragangliomata of the appendix develop early in life, run a benign course and in some respects resemble a local hyperplasia of chromaffin tissue rather than true tumors.

They are often very small and not recognized on casual examination. All appendices removed should be cut in thin transections, preferably after formalin fixation, rather than opened longitudinally at the time of operation.

A MONG the prevalent diseases of childhood we find a class which may be grouped under the one general heading of "Gastro-Intestinal Malfunctions." There is one malady included in this group, namely celiac disease, which we wish to present in this paper.

The disease is interesting although comparatively rare. It presents such an impressive, typical and pathetic clinical picture that when once seen is not so soon forgotten and when met with in practice becomes a problem requiring the utmost patience and perseverance in those concerned with the treatment.

Celiac disease was first referred to by Celius Aureleanus and Areteaus, but it was not until 1888, when Dr. Gee of St. Bartholemew's Hospital, described and differentiated it from other similar conditions of childhood, that the disease was known as a clinical entity. Subsequently such men as Gibbons, Bramwell, Cheadle and Herter have carried on individual investigations, each worker ascribing a specific name to the condition, which in most cases is an endeavor to indicate the particular views of the diligent, as to the cause of the disease. Up to a few years ago celiac disease was known in certain localities by such names as recurrent diarrhoea of children, intestinal or pancreatic infantalism, chronic digestive insufficiency and fat intolerance. Through the investigation and efforts of Abt, Still, and others, the above conditions have all been placed under the one heading and are at present considered as one and the same disease.

The etiology of celiac disease is at present unknown though numerous theories have been advanced in an attempt to determine the causative factor, none of these can be applied to every case, though they do fit into the scene in a limited number of cases. To show how conflicting the ideas of various workers are, we will present a few of the more interesting theories.

Gibbons attributes the disease to a disturbance in the nerve supply of the abdominal viscera, such that there is created a disfunction in the digestive process, permitting the absorption of toxic products. Bramwell claims it is due to a pancreatic deficiency, and Cheadle that there was a lack of formation of bile. Porter and Carter described a condition of ascending duodenitis affecting the mucosa of the pancreatic ducts, thus influencing metabolism. Herter claimed that he found an abnormal growth of Gram negative bacterial flora, which caused an inflammatory condition blocking effective absorption.

Overfeeding of carbohydrates and fats has developed into celiac disease, but in other cases it has developed on a well-balanced diet. It has followed in the wake of the more common general infections, although conclusively Abt states that tuberculosis and syphilis are definitely not causative factors, which fact is particularly interesting,
because in the case report which is to follow both of the above diseases have been found present in one or both parents, furthermore, none of the above etiological factors are sufficient to explain the origin in this particular case.

It is interesting to note that not only etiologically but pathologically as well the disease shows no definite findings. The changes that have been found at autopsy have been insignificant and generally found in only individual cases. Occasionally a small or large liver, a fibrotic pancreas, and a slightly enlarged colon with thickened walls, have been found. These changes have been ascribed, by most authorities, to the results of a mild concurrent infection during the course of celiac disease.

As previously mentioned, the condition is not very common, being found more frequently in girls than boys, appearing usually from 9 to 18 months of age, although cases have been recorded where illness commenced at 4 and 5 years. The malady generally extends over a period of three to four years, with an occasional case lasting as long as sixteen years. It is found more frequently among the well-to-do classes for the reason that infants of such families are more often artificially fed than those of the poorer people.

In considering the symptomology of celiac disease, we wish to present a typical case, the records of which were procured through the kind efforts of Dr. R. E. Struthers and by the gracious permission of Dr. Chandler, members of the staff of Montreal General Hospital, where the case was being treated.

The patient was a girl of 6 years, weight at time of admission was 18 lbs. 5 ozs. Her complaints on admission were failure to gain weight, intermittent diarrhoea, and distinct pallor. The history reveals a normal full term spontaneous birth, weight 8½ lbs., showing no abnormalities. For 15 months she was breast fed, receiving no complementary foods, then she was given cod liver oil and orange juice. At two years she was switched to bottle feedings, which were continued till age three, having refused other forms of food. From three years on, she was fed on a general diet including fish, eggs, potatoes, soups, biscuits, cakes and butter, taking her food well. About six months later she began to alternately lose and gain weight, never exceeding 28 lbs. and at the same time she developed intermittent attacks of diarrhoea, producing a sour-smelling stool of a porridge-like consistency and pale color, sometimes as many as six or seven stools per day. Six months ago a small amount of blood was passed, but this has not recurred. This condition was soon followed by a gradually increasing pallor. Two months ago she became nervous, restless, impatient and irritable. She has always been mentally bright, in fact appearing to be somewhat precocious. Since two years of age she has been able to walk and talk, and has been able to help herself. Her past history shows measles as the only previous illness. Her family history shows her father as a luetic and having a persistent cough, sputum negative for T.B. Her
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mother is also luetic. There are five children in the family, one of which is a positive case of tuberculosis. The physical findings as reported, with the exception of general appearance, development, and abdominal distension are negative. The patient is small, athreptic, maldeveloped, and infantile for her age. There is practically no panniculus. She is suffering no pain, though she complains of slight discomfort in her abdomen, which is distended, symmetrical, non-rigid, with no palpable spleen, liver or other masses.

Celiac disease generally develops insidiously, often with attacks of diarrhoea, varying greatly in severity. Authorities on the subject agree that such signs as arrested development, fluctuation in weight, pallor, abdominal distension, and the passage of stools containing a high percentage of undigested fats are typical of celiac disease. The above history demonstrates these points with remarkable clarity, giving what is so commonly termed, but not so often seen, the typical clinical picture.

Arrested development is strikingly demonstrated in the above case. At age six, the patient’s height was 35 inches, and weight was 20 lbs. 15 oz., the normal for a child of her age being 43.8 inches and 49 lbs. 3 ozs. respectively, her condition being that of a child of approximately three years of age. Her muscular development is subnormal though the ordinary movements such as walking and dressing are quite normal; x-ray plates of the long bones show normal development of the centres of ossification, a finding which is not always present. Her mental state seems abnormal due to her retarded physical development. Children suffering from this condition are as a rule bright intellectually, especially when considering the fact that they tend to be hypochondriacal and are difficult to teach.

The remarkable fluctuations in weight are shown clearly in the hospital records of the patient; at age two she weighed 28 lbs., which is the highest she has ever been, while at the time of admission she weighed only 20 lbs. 5 ozs., and has since gained and lost weight quite regularly. Her best weight was 24 lbs., about two months after treatment had been begun, but later she again dropped until at the time of receiving her record she weighed only 20 lbs. 15 ozs. Later reports have shown her to be steadily losing. According to her daily record sheets her loss in weight usually followed attacks of diarrhoea, stools being six to seven per day, sour-smelling, light colored, and of consistency of porridge, showing an acid reaction. Between attacks the stools became more formed and were not so offensive. The color of the stools is thought to be due to the fact that the bile pigments which are present are covered by the undigested fat, which amount to 38.2 per cent. of the ingested fat, which is well above the amount found in the normal stool. Of this total fat content, 41 per cent. was neutral fat and 59 per cent fatty acids. Cases have been reported where as much as 90 per cent of the ingested fat has been recovered in the stools.

Examination of the blood revealed a low red cell count of 2,700,000
as well as a hemoglobin per cent of 45, which results in a typically low color index. Upon transfusion the patient showed definite signs of improvement for about two months. Estimation of blood calcium and phosphorus showed a slight subnormal percentage of the latter, the report showing 2.6 per cent and the calcium 9 per cent. Herter claims this loss is brought about through the stools, having demonstrated the presence of these elements in excess in most cases of the disease, and furthermore he attributes the occasional appearance of tetany to the lack of calcium in normal proportions in the blood.

Such organs as heart, lungs, kidneys, liver and spleen are not affected by the disease, however, due to the general debility of the patients we have to be on the lookout for complications. These, in short, are generalized edema and ascites (due to nutritional disturbances), scurvy and rickets which can be controlled by including sufficient vitamin-containing foods in the diet, purpura and general infections, and occasionally tetany.

Celiac disease has to be differentiated from abdominal tuberculosis, megacolon, chronic pancreatic insufficiency, chronic cholitis and unsuitable dietary disturbances. These are ruled out by clinical and laboratory tests.

In treating celiac disease, the general consideration of diet is paramount. Fats are not digested. Carbohydrates in excess may bring on a severe form of diarrhoea, this leaves proteins as the main source of food. Accordingly the diet has to be built around them as a basis, for even these when fed alone set up an unpleasant putrefactive process which, though not distressing, is undesirable. These facts complicate the treatment and require careful regulation. The food, of course, must be sufficient to maintain the needs of a daily existence both as regards caloric values and vitamin content. This is accomplished by feeding breast milk, sour milk, dried milk or, better still in this particular disease, one to two pints of skimmed lactic acid milk plus up to 3 ozs. casein, a day. Protein milk often acts well, though it contains more butter fat than the former and may not be digested as well. Sweet cow’s milk is not tolerated by patients suffering from celiac disease. Cod liver oil and orange or lemon juice supply the necessary vitamins to prevent rickets and scurvy. When the patient shows improvement this diet must be augmented by gradually adding such things as scraped beef, chicken, lamb, fish, gelatin, eggs, boiled cereals, rice, ripe bananas, dextrin or starch. Green vegetables upset digestion and can be given only when cooked; these supply iron, mineral salts and vitamins. Butter should not be given. General considerations such as good nursing, pleasant, quiet surroundings, supplemented by massage, helio and hydrotherapy, passive exercise can be used with beneficial results.

Drugs are of little use. Iron may be given in severe anemia, while bile salts and pancreatic extracts or purgatives have no beneficial effects. Statistics are limited regarding the prognosis, but it is generally conceded that if concurrent infection or exhaustion can be inhibited,
recovery is complete, though in most cases protracted. The patient above referred to has shown no signs of infection to date and accordingly there is a fair possibility of a recovery.

**SUMMARY**

1. Celiac disease is a definite clinical entity.
2. Etiology is unknown.
3. Pathology is negative.
4. Signs and symptoms are clear cut and typical.
5. Treatment is simple dietetic.
6. Prognosis is good where complications can be controlled.

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**Problems of Cardiac Disease Associated with Urinary Retention**


The importance of cardiac problems in cases of benign prostatic hypertrophy causing urinary obstruction has been recognized in recent years by urologists, but the importance of always recognizing even minor grades of bladder retention associated with myocardial insufficiency is not always appreciated. In a case, presenting a predominantly cardiac picture, bladder obstruction is usually not sought for, especially when the signs and symptoms of the latter are not outstanding.

From a group of 40 cases studied and treated there appears to be a definite relationship between the chronic bladder obstruction and cardiac disease.

Two cases were reported of advanced myocardial failure, with ineffectual response to digitalis and rest, which gave amazing results when bladder retention, accidentally discovered, was cleared up.

In 22 cases of the group, cardiac arrhythmias were noted, which, in all but three cases disappeared after drainage had been established.

Auricular fibrillation, partial heart block, also occurred in a number of cases, and in most instances these irregularities cleared up after prostatectomy or bladder drainage, without the use of digitalis.

From these investigations it appears that a thorough search of the urinary tract should be made in all patients presenting cardiac problems.—*Mildred Wagner, '32.*
A CUTE rheumatic fever in childhood is a subject which must always have great interest for the general practitioner. Its etiology is still obscure, while the part it plays in the production of permanent, and often extensive, damage to the heart makes an attack one of anxiety and importance. The profession will therefore welcome the small volume just published on “Recent Advances in the Study of Rheumatism,”* by Doctor Poynton and Doctor Schlesinger, both of whom are connected with the Hospital for Sick Children, in Great Ormond street, London, England; and both of whom have given to the subject of rheumatism in childhood much study.

Unfortunately, under the term “rheumatism” are included several disease entities which appear to be distinct from one another; the volume has therefore two parts; the first deals with the acute forms of rheumatism, the second with chronic arthritic affections. It is with the first type of rheumatism that we desire to deal in the present paper.

Even when we limit our attention to the acute form, we note that the authors in the opening pages call attention to the difference between the acute form of the disease as met with in the adult, characterized by high fever, acute polyarthritis, sour perspiration, and not infrequently with cardiac damage—a type which they regard as on the wane at present—and the disease as met with in childhood manifesting itself by a commencement which may be insidious, with little or no trouble in the joints, but too frequently with an early involvement of the heart structures. Poynton states that in at least fifty per cent of the cases damage to the heart develops in the first attack, and is liable to become greatly increased with every recurring attack.

The high mortality rate of heart disease, as revealed comparatively recently by statistics, directed the attention of the profession to rheumatic fever in childhood as its most important factor. As a consequence, the literature on the subject became extensive, and the authors of this volume have done well to present us with this critical summary.

Although much research has been undertaken, Poynton and Schlesinger do not think we are yet able to assess the relative importance of heredity, diet, damp rooms, lack of sanitation, poverty, nervous strain, and city life as predisposing or exciting causes of the disease. Attacks are liable to occur in every phase of society. It is generally recognized that disease is more prevalent among the poor, especially among those who live in their own more or less defective homes. Children who live in the Poor-Law Residential Homes are seldom victims. Coates and Thomas¹ have emphasized the deleterious influence

of basement rooms in low-lying localities, and Thomson\(^2\) of homes built
in the vicinity of old water-courses.

At the Rheumatic Conference held in Bath in 1928, Dingall
Fordyce\(^3\) called attention to three predisposing factors, which he
regarded as important: instability of the nervous system, impaired
nutrition owing to digestive disorders, and an unhealthy state of the
lymphoid tissues; factors, he stated, which could be avoided by the
common-sense rules of general hygiene, proper hours of rest, and
avoidance of rush and excitement. Fresh air, sunlight, and the early
treatment of catarrhs he regarded as the best means of strengthening
vitality. Nutritional disturbances due to a deficiency of vitamin B,
lack of protein, and an excess of carbohydrate material he considered
to be predisposing to the development of an attack.

Although acute rheumatism is regarded as a sporadic disease, it
appears sometimes to assume an epidemic type. Cheadle, in his
lectures, spoke of the familial incidence of rheumatism. On the other
hand, the experience of the London hospitals is definitely against the
view that the disease is contagious. The authors suggest that while
it is not contagious in itself, the streptococcic infection often preceding
an attack may undoubtedly be contagious, and they refer the instances
of apparent contact infection in families and institutions to this
possibility.

The definite bacteriology of rheumatism, as stated above, still
remains an undetermined problem, but the close relationship of
streptococcal infection and acute rheumatism has during the past years
received much attention. Investigations appear to indicate that no
single type of streptococcus appears to be exclusively responsible for
the development of an attack. Swift and Kinsella\(^4\) report that they
have come to the conclusion that if the disease be streptococcal in
origin, no special strain can be singled out as the cause. Many investi-
gators are quoted who regard the streptococci as secondary invaders,
and think it probable that some entirely different organism may yet
prove to be the activating cause of the disease. In explanation of the
relationship between streptococcal infections followed after an interval
by symptoms of rheumatic fever, the authors suggest the probability
that we are dealing with an allergic reaction. They report two epidemics
of tonsillitis occurring at a Convalescent Home in the south of England,
for children with rheumatic heart disease. Previous to these epidemics
of tonsillitis, all the patients in the Home had been convalescent for
several months, and under daily supervision showed no sign of pyrexia
or quickened pulse rate. The symptoms of the tonsillar infection were
in no instance severe. Indeed, in some the attack in the throat might
have been overlooked had it not been for the pyrexia. In the majority
no sequelae followed the attack, and after a short pyrexia the
temperature dropped to normal, and complete recovery occurred. In
a certain number, however, after an interval of from 10 to 21 days
the temperature unexpectedly rose again with symptoms of a fresh
attack of rheumatism, which ran a more or less severe and quite
typical course. A similar sequence of events, viz., fresh attacks of
rheumatic fever unexpectedly following a mild throat infection after
a similar interval of 10 to 21 days has been noted in other children's
hospitals. Whatever the explanation may be, it would appear evident
from this series of cases that rheumatic relapses in children can be
provoked by acute streptococcal affections. A similar occurrence
sometimes occurs after an attack of scarlatina, in which rheumatic
symptoms develop after a similar interval of 10 to 21 days from the
first development of the throat trouble.

Although when regarded from a purely clinical standpoint, the
course, symptoms and pathology of an attack of rheumatic fever appear
to point definitely to a single infecting agent nevertheless, sufficient
evidence, in the opinion of the authors, has been brought forward to
show that while there is undoubtedly some connection between an
infection by various types of streptococci and an attack of rheumatic
fever, the original view of a direct infective action requires some
modification; and the explanation would appear to lie in regarding
the rheumatic attack as an allergic reaction provoked by the contact
of certain bacterial products with tissues previously sensitized to this
micro-organism. One of the common manifestations of bacterial allergy
in man is a localized inflammatory reaction. The fleeting pains so
common in rheumatic children may be evidence of a general rather
than of a special allergic reaction, and these are met with in other
diseases in which an allergic action has been more definitely estab­
lished. Scarlet fever is a known example, but an attack of rheumatism
has been traced to other infections, in which the streptococcus is in
evidence, such as measles and influenza. Simple streptococcal throat
infections may be the cause in many instances. From this allergic
standpoint an infection by one of the many types of streptococci may
serve as a rheumatic antigen. The authors add, however, that it must
be clearly understood that the theory of an allergic element in an
attack of rheumatic fever by no means excludes a bacterial origin for
the disease, since a bacterial antigen is an essential precursor of the
allergic state.

As regards the immediate treatment of an attack, the authors
call attention to the fact that the attitude of the medical profession
towards the salicylates has undergone a change. There can be no
question that for the relief of acute rheumatic arthritis this group of
drugs is still unsurpassed. So dramatic is the effect that it might
almost be called a cure, were it not for the fact that during its exhibition,
and in spite of the lowered temperature and increased comfort of the
patient the rheumatic and cardiac symptoms proceed unchecked. Few
physicians therefore now regard salicylates as a specific in the treatment
of this disease. Hanzlik's^ monograph is perhaps the most exhaustive
review of this subject. He doubts whether the addition of sodium
bicarbonate is of value in counteracting the toxic effects of sodium
salicylate. To be effectual, these drugs must be pushed in full doses, even to the appearance of toxic symptoms; but there is always danger in pushing salicylates too far in a frail rheumatic child. Still, without regarding them as actually specific, it must be recognized that this group of drugs has a marked influence on many of the patient's symptoms, and it would be a short-sighted policy to abandon the use of them in the treatment of the more acute manifestations.

It is interesting to note that the drugs which have a beneficial effect in rheumatic arthritis also have an influence on the joint manifestations of serum disease. It may be that a large part of the mystery which surrounds the variable effect of salicylates on the different rheumatic manifestations is reflected in its selective action on certain symptoms of serum disease. Strong evidence has been adduced that aspirin has the power of mitigating the more severe consequences of horse serum injection. Recently, a successful attempt has been made to prevent relapses occurring in rheumatic children after an attack of tonsillitis, by giving aspirin in fairly large doses during the quiescent interval following a throat infection.

In the late treatment of acute rheumatism in young children, after the acute symptoms have subsided, the establishment of supervisory centres and the employment of skilled home visitors must eventually prove of great assistance and lead to the prevention and amelioration of many cases of heart disease. In 1912, Poynton urged the necessity of a period of prolonged rest for rheumatic children under special supervision. This movement for the erection of convalescent homes and rheumatic clinics has now become one of national importance in Great Britain. It is essential, however, that all supervision and nursing should be based upon the conception of the disease as a constitutional affection; that is, not merely a form of heart disease or arthritis, or nervous disorder, but a disease which may attack at one or another time any of these systems. Undoubtedly heart disease is the greatest danger, but the study of that organ must go hand in hand with that of other rheumatic manifestations, and the same principle applies to chorea and arthritis. The good effect obtained by a prolonged convalescence in quiet country surroundings, combined with moderate discipline and graded education, certainly favors the view that an element of nervous strain exists as a prominent predisposing cause, not only in chorea but in all the other forms of acute rheumatism.

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A Case of Anemia in Pregnancy

FRANK D. POOLE, M.D.

Meek Fellow in Obstetrics and Gynecology, Victoria Hospital
London, Ontario

IT has been fairly definitely established that throughout a normal pregnancy there is a marked drop in the percentage of hemoglobin. This may be termed a physiological anemia. In contradistinction to this type there appear from time to time anemias which are associated with definite pathological conditions existing before or at the time of pregnancy. The association of toxemia and the alteration in the blood picture in pregnancy at the present time does not appear to be clearly understood. The case reported below suggests that there is a definite connection between the alteration in the blood picture and pregnancy toxemia and also the probability that anemia is one of the manifestations of a toxic condition without other clinical signs. It is believed that in this case there was a definite toxemia existing.

Case history of a female, age 39. The history of her past illnesses was negative, except for scarlet fever in early childhood. The first pregnancy was in 1920. Up to ten days before delivery there was nothing unusual about her condition. Suddenly she developed noticeable edema, saw specks in front of her eyes and suffered from severe headaches. These disappeared after delivery. In 1923 she apparently had a repetition of these symptoms and miscarried at about five months. The fetus had been dead several days. In 1925 she carried another child to six and one-half months when she began to show generalized edema and labor was induced. During her present pregnancy she was asked to report to the hospital from time to time to have a thorough examination, as her pregnancies up to the present one had not been carefully supervised. The results of the examination show:

<table>
<thead>
<tr>
<th>Urinalysis</th>
<th>Jan. 23</th>
<th>March 12</th>
<th>April 25</th>
<th>May 27</th>
<th>June 26</th>
<th>July 12</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albumen</td>
<td>1 plus</td>
<td>Negative</td>
<td>Negative</td>
<td>1 plus</td>
<td>1 plus</td>
<td>trace</td>
</tr>
<tr>
<td>Specific Gravity</td>
<td>1025</td>
<td>1020</td>
<td>1015</td>
<td>1014</td>
<td>1020</td>
<td>1015</td>
</tr>
<tr>
<td>Cast</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>Erythrocytes</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
</tr>
</tbody>
</table>

Examination of Blood—

<table>
<thead>
<tr>
<th>Non-Protein</th>
<th>Nitrogen</th>
<th>Creatinine</th>
<th>Sugar</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>27.3 mg.%</td>
<td>1.2 mg.%</td>
<td>.92 mg.%</td>
</tr>
<tr>
<td>1930</td>
<td>20.4</td>
<td>1</td>
<td>.75</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Blood Count</th>
<th>Jan. 23</th>
<th>March 12</th>
<th>April 25</th>
<th>May 27</th>
<th>June 26</th>
<th>July 13</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin</td>
<td>60%</td>
<td>36%</td>
<td>40%</td>
<td>48%</td>
<td>46%</td>
<td>67%</td>
</tr>
<tr>
<td>Erythrocytes</td>
<td>4,010,000</td>
<td>2,820,000</td>
<td>2,960,000</td>
<td>3,650,000</td>
<td>3,390,000</td>
<td>5,010,000</td>
</tr>
<tr>
<td>Color Index</td>
<td>0.67</td>
<td>0.57</td>
<td>0.60</td>
<td>0.59</td>
<td>0.61</td>
<td>.61</td>
</tr>
<tr>
<td>Leucocytes</td>
<td>8,250</td>
<td>12,000</td>
<td>9,900</td>
<td>8,350</td>
<td>10,500</td>
<td>5,850</td>
</tr>
</tbody>
</table>

per cu. mm.
A CASE OF ANEMIA IN PREGNANCY

Neutrophiles .......... 59% 74% 60% 38% 37%
Mature Forms....... 50% 70% 46% 32% 35%
Young Forms........ 9% 4% 14% 6% 2%
Lymphocytes ........... 37% 21% 34% 58% 63%
Eosiophilies .......... 0% 0% 4% 1%
Monocytes .......... 2% 3% 2% 3%
Basophiles ............ 1% 2%
Myelocytes ........... 2% 0%

Appearance of red blood cells—Jan. 23, pale; March 12, pale, variations in size; April 25, pale, variations in size; May 27, pale, variations in size and shape, some polychromasia; July 13, pale.

Urea Concentration Test—

<table>
<thead>
<tr>
<th>Time</th>
<th>Volume cc.</th>
<th>Urea %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st hr.</td>
<td>155</td>
<td>162</td>
</tr>
<tr>
<td>2nd hr.</td>
<td>100 Normal</td>
<td>168</td>
</tr>
<tr>
<td>3rd hr.</td>
<td>70</td>
<td>125</td>
</tr>
<tr>
<td>1st hr.</td>
<td>1.64</td>
<td>1.3</td>
</tr>
<tr>
<td>2nd hr.</td>
<td>2.80 Normal</td>
<td>1.2 (Note)</td>
</tr>
<tr>
<td>3rd hr.</td>
<td>3.32</td>
<td>1.8</td>
</tr>
</tbody>
</table>

Blood Pressure—

<table>
<thead>
<tr>
<th>Time</th>
<th>Volume cc.</th>
</tr>
</thead>
<tbody>
<tr>
<td>92/50</td>
<td>Same</td>
</tr>
<tr>
<td>102/70</td>
<td>118/75</td>
</tr>
<tr>
<td>100/70</td>
<td></td>
</tr>
</tbody>
</table>

Blood Wassermann—Negative
Blood Cholesterol—

<table>
<thead>
<tr>
<th>Dilution Test of Kidney Function—Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specific Gravity Volume Test—Normal</td>
</tr>
<tr>
<td>Basal Metabolic Rate—1%</td>
</tr>
</tbody>
</table>

On June 29, 1930, it was felt that there was danger in prolonging her pregnancy on account of the development of edema and change in the urea concentration test. Accordingly labor was induced medicinally and she was delivered of a living child. Her recovery has been apparently complete and the child’s condition at the present time is excellent.

As has been shown by the blood picture of July 13th, 1930, there appeared to be a fairly rapid return to normal.

It is to be noted that along with rest in bed at frequent intervals that careful attention was paid to her diet. The diet consisted of one rich in vitamins with special reference to vitamin “E.” It was also rich in mineral content and was of a high caloric (about 3000 cal.) value. It included abundance of green vegetables, wheat germ and liver. The medication consisted of preparations of iron, copper and calcium. Cod liver oil was given daily.

The continuance of the pregnancy under this treatment suggests that in this case there was some probable dietary deficiency which in former pregnancies was essential for its normal continuance and the success of this diet and treatment might indicate that it could be used to advantage in the handling of other forms of pregnancy toxemia.

The author wishes to acknowledge his indebtedness to Dr. W. P. Tew and Dr. E. M. Watson, under whose direction the report was made.

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3Skajaa, Kr.—Acta obstetrica et gynecologica Scandinavica 8; 371-430; 1929.
Huntington's Chorea
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Huntington's chorea is a disorder of the nervous system of chronic type, characterized by irregular coarse or fine, non-rhythmical, non-coördinated, jerky movements and ataxias. It is progressive and shows certain definite hereditary factors. It occurs in adults and is marked by mental deterioration.

George Huntington, an American physician, born in Long Island, New York, in 1850, had an opportunity of observing several of these cases in his practice and is accredited as having given the first comprehensive and distinctive description of this disease which bears his name.

Following is a synopsis of a typical case history which illustrates the above characteristics.

L., admitted to Westminster Hospital, June, 1920, aged 31; married, farmer. He served in the army in Canada, England and France.

Family History*

Father died at the age of 45. The patient had been unable to feed himself for two years previously owing to shaking of his arms and nervousness. Two of the father's sisters were described as being very nervous. Mother was living at the age of 50. Brothers, one was living and was described as very nervous; one sister living. The patient was married at the age of 18 and there were four living children, their ages ranging from 3 to 12 years. Two children were still-born.

Previous Illnesses

Mumps and measles in childhood. The fact was elicited that he had always been nervous and shaky. In July, 1920, this man was described as follows: Physical examination shows that he is of small stature, poorly nourished, present weight is 109 pounds. Heart and lungs are clear. No abnormalities were noted in the cardio-vascular, respiratory or abdominal systems.

Neurological System

The pupils react normally. The knee jerks are brisk, there is a generalized coarse tremor. His gait is unsteady and staggering.

Mental Status

On admission no delusions or hallucinations were elicited. His judgment and memory were only fair. At times when questioned he would stare in a confused manner for a couple of minutes before answering. His mental age by the Princeton Scale was ascertained

*From patient at time of admission.
to be about 13 years. In March, 1921, his physical and mental conditions were described as being unchanged. He would wander aimlessly about the ward with a staggering gait. There were times in which he would be quite confused and experience great difficulty in making his wants known. He did a small amount of work on the ward and attended entertainments.

This man's condition has retrogressed to such an extent throughout the eleven years that he has been a patient in this institution that, whereas, on admission, he was able to assist to some extent, although inefficiently, with farm work, he has for several years now been confined to bed, a helpless invalid, unable to do anything for himself. He has fallen several times. His movements are characteristically incoördinated, involving practically all the muscles of his body. On attempting to make any intentional gesture or movement his arms wave wildly in the air, his fingers are extended backwards, his head nods forward and he makes weird grimaces in an ineffectual attempt to speak. The outstanding features of this case are:

(1) Steadily progressive incoördinated movements occurring in an adult with progressive mental deterioration.

(2) A definite history of choreic movements in the father and mental and nervous abnormalities in the paternal aunts and in one brother.
Case Report Gastric Tetany Complicating Pyloric Obstruction

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Staff of Medicine, Victoria Hospital
London, Ontario

P. L., a male patient, aged 37, first complained, while in army service in 1917, of severe epigastric pain occurring postprandially. Treatment was instituted but as he did not improve the patient was discharged from the army. A short time after discharge he began to vomit; the vomiting was of a projectile nature, and did not improve during D. S. C. R. treatment extending over a period of one year. Since this time the patient has had various forms of treatment with no relief. He was first seen in the out-patient department of Victoria Hospital on November 3rd, 1930, and complained of projectile vomiting, occurring seven or eight times each day, epigastric pain of a more or less continuous character, anorexia, and loss of weight and strength.

X-ray report.—The stomach is large and dilated. Great waves pass across it from the cardiac end to the pylorus, but these quickly subside leaving the stomach in a condition of atony. The barium is seen leaving the stomach but without the formation of a duodenal cap. What should be the cap is an irregular passage which is apparently constricted. After five hours about five per cent of the barium has escaped from the stomach. After twenty-four hours, notwithstanding the fact that between the second and third examinations the patient has vomited copiously, there still remains approximately 70 per cent retention of the barium, and about 15 per cent has passed through. After forty-eight hours there is still 35 per cent retention of barium in the stomach. The rest of the barium is in the colon from the caecum onwards.

X-ray diagnosis.—Cicatrical duodenal ulcer causing marked pyloric obstruction.

Laboratory findings.—Urine: alkaline, specific gravity 1030, negative for albumen and sugar, amorphous phosphates four plus, blood Wassermann negative.

The patient was strongly advised to accept surgical treatment, but would not consider operation. He was subsequently seen in the O. P. D. and had not improved under alkaline treatment.

Hospital record.—On December 2nd, 1930, at 4 p.m., the patient was admitted to Victoria Hospital complaining of severe pain throughout his body, most marked and continuous in both arms, and intermittent but severe in the abdomen. The condition had begun at 3 a.m. on the same day with numbness and tingling in arms and hands. Later
the arms, wrists and fingers became spastically flexed with severe pain. He was very thirsty and consumed large quantities of fluid which he vomited forcibly in a short time. He also complained of spasms of rigidity in both legs and in the jaw, the latter preventing him from speaking until the spasm had subsided.

Physical examination.—Showed a thin, emaciated white male of middle age, lying in bed in considerable pain. His arms were rigidly flexed over the chest and at the elbows and wrists. The fingers were flexed at the proximal metacarpal-phalangeal joints but extended at the more distal joints. The thumbs were adducted. The sensorium was quite clear. The pupils were pin-point and there was evident nystagmus; the tongue was coated and the teeth were in poor hygienic condition. The heart was negative except that the sounds were of poor muscular quality. The lungs were normal. The abdomen showed no masses, rigidity or tenderness. The deep reflexes were absent throughout the body; the abdominal reflexes were absent although both cremasteric reflexes could be obtained. Babinski, Oppenheim and Gordon were equivocal.

Laboratory findings.—Spinal puncture showed clear fluid under a pressure of 12 mm.; cell count 3 per c.mm.; globulin slightly increased; Wassermann anticomplementary; colloidol gold curve negative.

Progress notes.—December 2nd, 1930, 10 p.m.: symptoms have subsided; arms relaxed and complains of no pain; sensorium quite clear.

December 3rd, 1930, 5.30 a.m.: patient had another attack of rigidity of both arms with extreme pain; trismus of the facial muscles was quite marked; sensorium clear; marked thirst; patient has vomited several times during night. Codeine grains one hypodermically gave little relief.

7.30 a.m.: pulse became weak and irregular; breathing shallow and rapid; patient ceased to breathe.

Post-mortem.—Showed chronic duodenal ulcer with marked pyloric obstruction and marked dilatation of the stomach; tonic contraction of both forearms and hands; emaciation; chronic passive congestion of lungs and liver; adenoma of the upper part of left adrenal.

Summary.—Although no blood chemical studies were carried out on this case, it is quite apparent that the diagnosis was that of gastric tetany complicating pyloric obstruction.

Tetany is a condition characterized by hyperirritability of the nervous system, both sensory and motor, manifesting itself in certain cases by spontaneous spasms and contractions of various groups of muscles, or of the musculature of the whole body. Tetany is seen in patients of all ages and under various circumstances, and can be readily classified into two groups by changes in the serum calcium level:

(a) With lowering of serum calcium,

1. occurring in infants, commonly associated with rickets and frequently termed spasmophilia;

2. following injury to or removal of the parathyroid glands;
3. following severe and long continued diarrhoea.

(b) With no demonstrable decrease in serum calcium,
1. occurring idiopathically in healthy workmen;
2. cases associated with maternity, severe intoxication or acute infection;
3. following obstruction in the upper intestinal tract.

The symptoms of tetany vary from a mild degree of muscular stiffness or tightness, with or without pain, to severe tonic muscular spasms involving commonly the hands and forearms, less frequently the head, neck and legs. These spasms may be associated with intense, agonizing pain. Sensations of numbness, prickling, and tingling in the hands and feet and around the mouth are complained of at times. The hyperirritability of the motor nerves is made evident by their increased reaction to faradic stimulation (Erb’s sign), and mechanical stimulation (Chvostek’s and Trousseau’s signs). The importance of these signs cannot be overestimated because many cases of tetany show no evidences of muscular spasm or disturbance of sensation.

Gastric tetany is occasionally seen following marked obstruction in the upper intestinal tract, generally by a benign lesion, but by no means occurs in all cases. Frequently the symptoms in such obstructions are referable to alkalosis, such as headache, nausea and dizziness, or in the acute cases drowsiness and absolute prostration.

McVicar claims that tetany may be anticipated if the alkali reserve exceeds 100 per cent.

The actual cause of gastric tetany is not known; some authorities believe that all forms of tetany are an expression of parathyroid deficiency and the result of some disturbance in the calcium metabolism of the body. The serum calcium level in gastric tetany is normal. However, the marked dehydration, alkalosis and chloride depletion observed in these cases may conceivably affect the calcium metabolism by altering the rate of diffusion of electrolytes and fluids in the tissues. Recent work by Snell indicates that the concentration of diffusible calcium may be considerably reduced in tetany, although the level of serum calcium be unchanged.

Until quite recently the occurrence of gastric tetany was looked upon as an ominous, if not fatal, sign. However, by frequent gastric lavage and a determined attempt to combat the underlying dehydration and chloride depletion, remarkable recoveries have been observed. Intravenous administration of 2000 c.c. or more of physiologic solution of sodium chloride each day together with 2000 c.c. of liquid food by mouth and by rectum are given, and often in two or three days the severely toxic patient is in relatively good condition. Operative correction of the obstruction is, of course, necessary, otherwise the patient would die of starvation. In some cases, however, the patient goes down-hill despite every effort, and recent work has shown that in these cases a severe grade of nephritis is often present, which terminates in fatal uraemia.
A Case of Hodgkin's Disease

E. H. KINSMAN, '31

MRS. M., aged 27, was admitted to the Queen Alexandra Sanatorium on October 20th, 1929, complaining of weakness, persistent cough, intermittent pain in the lumbar region and right hip and shortness of breath. She had noticed swellings in the right side of the neck for two years previous to admission.

PAST HISTORY

The patient had influenza in 1918, lasting two weeks, and from which she made a good recovery. In August, 1928, she became pregnant and the following month noticed that she was tiring readily on slight exertion. In November she contracted a severe cold and has seldom been free from cough since. In January of the following year she suffered from pain in the lumbar region, which was at times quite severe. This pain was not relieved when her child was born in May, 1929, and has been more or less constant, being present at the time that she was admitted. Her confinement was without complications and she was able to be up on the fourteenth day. Weakness, however, persisted and increased. Cough and dyspnoea became more marked, and she had several night sweats. In July, 1929, pulmonary tuberculosis was diagnosed and she was advised to adopt rest treatment. She accordingly remained at home and her temperature was taken regularly up to the time of her admission, and showed a constant elevation, frequently being as high as 101° F. During this period of rest at home pain developed in the shoulders, neck and right hip as well as in the lumbar region. Loss of weight was also a feature. Her weight four years previous to this time was 130 lbs. After confinement it was 113 lbs., and on admission it was 102 lbs.

PHYSICAL EXAMINATION

The patient appeared quite ill with marked emaciation and pallor. The temperature was 103° F., pulse 120 and respirations 28. The glands on the right side of the neck were quite noticeable and were found to be firm and discrete on palpation. A group of glands in the right inguinal region showed the same characteristics. The heart was rapid and there was a faint systolic murmur over the mitral area. The breath sounds were harsh and somewhat broncho-vesicular in character and whispered voice was increased over the right interscapular region and fine moist rales were heard. X-ray films of the chest showed a scattered and rather discrete parenchymatous process in the right apex and first interspace. A consolidation was also present in the right base medially, with a rather discrete lateral margin, the characters of which suggested it to be glandular in origin. No outstanding opacities were seen in the left lung.
LABORATORY FINDINGS

Repeated sputum examinations failed to show any tubercle bacilli and urinary findings were negative. Blood counts were done as follows:

<table>
<thead>
<tr>
<th></th>
<th>Oct. 26</th>
<th>Nov. 3</th>
<th>Nov. 21</th>
<th>Jan. 18</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red Blood Cells</td>
<td>3,540,000</td>
<td>2,500,000</td>
<td>1,650,000</td>
<td>2,200,000</td>
</tr>
<tr>
<td>White Blood Cells</td>
<td>22,400</td>
<td>34,875</td>
<td>36,500</td>
<td>76,000</td>
</tr>
<tr>
<td>Haemoglobin</td>
<td>60</td>
<td>55</td>
<td>55</td>
<td>Not taken</td>
</tr>
<tr>
<td>Polymorphs</td>
<td>86</td>
<td>91.5</td>
<td>84.5</td>
<td>92</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>9</td>
<td>3.5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Eosinophiles</td>
<td>1</td>
<td>0</td>
<td>.5</td>
<td>1</td>
</tr>
<tr>
<td>Basophiles</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Myelocytes</td>
<td>0</td>
<td>1.5</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>

Blood Wassermann test was negative.

PROGRESS

During the observation period the temperature varied between 101°F and 103°F. The pulse rate varied between 110 and 124. After ten days of careful study a tentative diagnosis of Hodgkin's disease was made on the presence of the enlarged glands in the cervical and inguinal regions, which had remained hard and discrete over a two year period; the finding of enlarged glands in the mediastinum on x-ray; the persistent elevation of temperature; and the blood picture. The patient was confined to bed and became progressively weaker. Emaciation and pallor increased and dyspnoea and cough became more distressing. The temperature remained about 103°F. The rapid pulse persisted with a gradual decrease in volume. Pulse rates as high as 150 were found. The glands in the cervical, mediastinal and inguinal regions increased in size, and glandular enlargement of the same character appeared in the left cervical region. As the disease progressed the glands in both axillary and both inguinal regions became involved. A slight icterus developed with itching of the skin and marked discoloration of the conjunctivae. Some weeks before death, myocardial decompensation became evident, manifested by the presence of fluid in the pleural and abdominal spaces and edema of the extremities and dependent portions of the body. Patient died on February 10th, 1930.

PATHOLOGICAL FINDINGS

A gland was removed from the left axillary region. On examination it presented a firm, slightly elastic character and smooth surface. Microscopically it was found to be very cellular, containing lymphocytes, eosinophiles, plasma cells, mononuclears, and giant cells of the Dorothy Reed type, which confirmed the diagnosis of Hodgkin's disease.

SUMMARY

The important features of the case would appear to be:
1: The appearance of firm, discrete, enlarged glands in the neck two years previous to admission, with gradual increase in size and extension of the process to other glands throughout the body.
2: The long febrile period and progressive weakness.
3: The pressure signs in the mediastinum, manifested by dyspnoea and distressing cough.
4: The progressive anaemia and polymorphonuclear leucocytosis.
5: The pathological picture seen in the gland post mortem.

I wish to thank Dr. W. C. Sharpe of the Queen Alexandra Sanatorium staff for his assistance, which has made this report possible.
Some Observations on Mastoid Complications

J. R. ARMSTRONG, M.D.

On looking up the literature, I find relatively little has been written on the important problem of mastoid complications and sequelae. Ballance, 30 years ago, emphasized the importance of some of the common complications met with, viz.: sinus thrombosis, meningitis, brain abscess and erysipelas.

An important fact to remember is that complications which were developing before operation may manifest themselves afterwards, such as sinus thrombosis, meningitis, labyrinthitis, general sepsis, remote septic conditions as arthritis, nephritis, etc. Of course, local infections may follow as in other operations, as erysipelas, stitch abscess, cervical adenitis, and abscess in adjacent tissues.

Many cases of sinus thrombosis, meningitis, and brain abscess are not sufficiently evident from the symptoms until it is too late for further surgical procedures to be of benefit. Indeed, in some of these cases, the pathology is so remotely situated that surgical intervention is practically impossible—as in the case of an Indian who died of meningitis in Victoria Hospital. At post-mortem a marked necrosis of the petrous portion of the temporal bone was found. This no doubt had existed for many weeks, practically without symptoms. The original mastoidectomy was performed in November, 1930. About two weeks afterward a sixth nerve paralysis occurred. This continued without any other untoward symptoms for several weeks, when symptoms of meningitis appeared from which he died in a few days.

The idea in the mind of the writer of this article is the recording of the incidence of six cases of sixth nerve (abducens) paralysis occurring in the course of convalescence following mastoidectomy during recent years—three of them this winter. In all six cases, convalescence was normal for a week or two following operation, when, suddenly, the inability of the eye on the operated side to turn out was noticed. In one case convalescence was normal and the patient (26 years old) was going about for twelve weeks when both sixth and seventh nerves showed evident paralysis. This case had gradually increasing headache for a week or so preceding the paralysis. This patient had a stormy time for two or three weeks, with temperatures of 103° to 105.2°, daily severe headaches, relieved for several hours by aspirating, daily, the spinal fluid, which at times was under great pressure, with markedly increased cell count and the presence of streptococcus, haemolyticus. This case was diagnosed as having definite meningeal irritation or perhaps meningitis. However, she recovered and has remained well up to the present time, i.e., about two and a half months.
Two cases of abducens paralysis, both in women about 35 years of age, in St. Joseph’s Hospital, three or four years ago—two to three weeks following mastoidectomy. In each of these the patient had a slight chill followed by temperature of 103°. In each case the operation wound was reopened, granulations removed, and the regions thoroughly inspected for necrosed areas, pus pockets, or localized pachymeningitis. In each case nothing was found about the area to account for the symptoms. Both patients recovered without further trouble.

Another case, age 22, seen in consultation, with sixth nerve paralysis appearing a week after mastoidectomy. After another week patient showed other symptoms of intracranial involvement, viz., edema of disc, occasional headache, and nausea, slow pulse, with occasional exacerbation of temperature, spinal fluid under slight or no pressure. These mild symptoms continued for about ten days or two weeks, when patient developed typical meningitis and died in 48 hours.

These cases bring one to a serious consideration of the problem as to the relationship of the sixth nerve to mastoiditis. Are these cases of abducens paralysis due to intracranial pressure? Many of them show no other evidences of such pressure. Are they due to the fact that the sixth nerve is quite exposed in the cranial vault before it disappears over the tip of petrous portion of temporal bone on its way into the orbit and hence exposed by contact to a dehiscence or perhaps necrosis of the petrous position? Is the petrous position involved in many of these cases and to what extent? Is it a developmental affair—as we know that both petrous and mastoid are developed from the same cartilage and are parts, finally, of the temporal bone.

Is the petrous involved more often than we have cognizance of—and subsides—and remains quiescent until perhaps some future trauma or debilitating illness sets a spark to the tinder again?

The foregoing brief discussion demonstrates the fact that although abducens paralysis is comparatively rare, it is nevertheless a serious sequel to mastoidectomy when it does occur. Until its etiological factors come to light, very little if any, and no rational, treatment can be instituted. We, therefore, view with considerably anxiety the advent of the sixth nerve paralysis following mastoidectomy.

On the evening of November 4, 1847, a date worth remembering, Simpson (1811-1870) and his assistants inhaled several substances without any marked effect. Ether still remained the unrivalled anesthetic. As this moment Simpson happened to remember that a Liverpool chemist named David Waldie had spoken to him about a certain heavy colorless liquid. Simpson looked for the bottle, but could not find it. Probably when he was on the point of remarking that it wasn’t of much importance anyhow, the amber-colored bottle was pulled out from the bottom of a heap of waste paper. Simpson scrutinized it again and shook his head dubiously. It seemed to him too ponderous to be of much value.
I have deliberately chosen eclampsia because of its importance to the general practitioner and also since the preventive treatment and especially the curative treatment of eclampsia has changed very radically in a scant ten years. At that time, while the eliminative treatment was considered very necessary, the crux of the treatment was the rapidity with which labor and delivery could be induced and accomplished, irrespective of the condition of the patient. Vaginal hysterectomy, high forceps, forcible and rapid dilation of the cervix, and caesarean section were all very much in vogue, but slowly the trend of the very best thought has been toward much more conservative treatment whereby the press of activity towards extreme operative treatment, with its attendant high mortality rate, has been practically discarded in all the large maternity clinics.

I quite realize that the average general practitioner will only see one or two cases of eclampsia a year, and that he cannot possibly evolve a line of treatment gained by experience from his own cases. One prominent obstetrician goes so far as to say that if the general practitioner is painstaking enough in his prenatal supervision there need be no cases of eclampsia developing convulsions. He says the pregnant mother should have an urinalysis and a blood pressure taken every two weeks during her carrying period and that with a systolic blood pressure of 160 m.m. and an albuminuria which persists on a milk-carbohydrate diet, with rest in bed, labor should be induced irrespective of the viability of the child.

In 1896, before the Obstetrical Section of the Royal Academy of Medicine in Ireland, Tweedy made the statement that food is the actual exciting cause of eclampsia: "Many observers have endeavored to prove the existence of an anaphylactic condition in eclampsia, and most of them have centered their attention on the placenta as the offending structure. So far as I know, it has never been suggested that substances taken by the stomach could act as an anaphylactic poison. In other words, it has been assumed that the epithelium of the intestinal tract is protective in this particular; and why it should be, is at least surprising. All of the structures of the body are liable to degeneration—the kidney epithelium when diseased permits the passage of albumin, similarly the liver and pancreas can cease to function. Why this universal rule should be withheld from the stomach epithelium would be wonderful if it were true, but that it is not true will be proved in the further study of eclampsia. Observation has shown us that many pregnant women do not drink enough fluid. Concentration of the toxins,

*Read before Toronto Alumni Association of the University of Western Ontario.
which arise from maternal and foetal waste may overtax the excretive organs and cause degeneration. The curious cravings which some pregnant women experience are due entirely to the accumulation of toxins causing much indigestible food to be taken; and this food is frequently incompletely masticated—thus potato skins, lumps of apparently raw cabbage, orange pulp, etc., have been removed from the stomach and bowels by lavage.” So much for Tweedy’s theory as to the causes for eclampsia as published in 1896. He may not have been far wrong. However, in 1926, thirty years later, Williams of Johns Hopkins advocated a great deal of raw vegetables, well masticated, of course, in the dietary of the pregnant woman, with three quarts of water extra intake each day, as follows: two glasses at bedtime when the stomach is empty, two glasses half an hour before breakfast, a glass of water every fifteen minutes for an hour between 10.30 and 11.30, at which time the stomach is supposed to be empty; and also a glass of water every fifteen minutes between 4 and 5 in the afternoon. He agreed with Tweedy that an extra intake of water is absolutely essential for the pregnant woman, whether toxic or not. In supporting the theory that food was the cause of eclampsia, Tweedy claimed that his patients always improved during the starvation period, and that even milk was harmful or even fatal.

Stephenson said: “It is not determined whether pre-eclamptic toxemia is the forerunner of eclampsia or whether it is a distinct entity. The after-effects of the pre-eclamptic condition are more pronounced and last longer than do those of the eclamptic condition. The occurrences in maternity cases is between 2 and 3 per cent. The etiology of the disease is unknown. Areas of degeneration and necrosis are found around the portal vein in the liver, which areas are the result of thrombosis in the small vessels, possibly due to agglutination of red blood cells. The kidneys show degeneration and necrosis of the renal epithelium, particularly in the convoluted tubules. The extent of the lesions in the kidney is variable according to most pathologists, and the tendency is to consider them secondary to the toxemia. The brain is usually the seat of edema and thrombosis. Many of the small vessels contain thrombi, and when the fatal termination is delayed, these vessels are surrounded by a necrotic and degenerated brain tissue. A definite hyperemia has been described but is not universal. The heart shows myocardial changes in the majority of cases. The diagnosis of eclampsia is not difficult, but it is important to endeavor to determine the disturbance of function after symptoms occur prior to the onset of convulsions. During the pre-eclamptic stage, tests to find characteristic changes in function should be applied. Dyes have been used in these tests, introduced intravenously. The most important ones used are Rosenthal’s and Delprot’s rose bengal tests. An abnormal liver will eliminate all the dye in a few minutes. In damaged livers, the dye remains much longer in the blood stream. There is no liver function test now available which is devoid of danger, easy of application, and at the same time
reliable. The injection of dyes intermuscularly or intravenously is often accompanied by some resulting damage at the site of injection, occasionally by thrombosis and embolism. The taking of blood samples in the Rosenthal tests is difficult and not always accurate—even in the hands of an expert. The duodenal tube method of demonstrating the initial appearance of dye in the bile is not practical in this group of toxemias, and at the present time the information derived from these procedures in liver function do not justify their employment. Kidney functions are better adapted for use in these toxemias. The readings, however, are not constant and the demonstration of lower function is more apt to come in cases where some damage has been done to the vascular system in the kidney, or in patients with pre-existing nephritis, except in definite nephritis, positive findings come very late in the disease, usually at a time when simple methods—such as the study of the urinary sediment and routine examination of the urine gives us an accurate picture. The tests we now employ may still be too crude to demonstrate finer changes in the involved organs. The value of these tests must not be overlooked, however. After the acute toxemia has subsided they give much valuable information as to the function of the organs, particularly with reference to permanent damage. This information is of great importance in the prognosis for future pregnancy.”

Blood chemistry is not especially important, except for blood urea. The findings in blood chemistry of eclampsia are as follows: urea, creatinine, uric acid, blood sugar, and lipases may be increased.

An examination of the eye-grounds should be made in all cases of acute toxemia. Positive findings such as retinitis, hemorrhage, and choked discs reveal the seriousness of the condition. In the presence of other positive findings with a severe toxemia, prompt termination of the pregnancy is essential. Blood pressure readings are important; a gradually increasing pressure, particularly diastolic, usually indicates toxemia.

One author suggests routine weighing of the patient to check up on the obese and the diet indicated. Routine tonsil and teeth examinations for focal infections; thyroid examination for endocrine disturbances of the unstable cardio-vascular type, all these should be carried out in addition to the tests mentioned above to help prevent eclampsia.

A great deal has been done during the last twelve or fifteen years to decrease the mortality due to eclampsia. This has been brought about by instituting efficient pre-natal care, and by the conservative treatment of eclampsia as laid down by Tweedy of Rotunda, and Stroganoff of Leningrad. Prevention should receive our first consideration. In the past too little attention has been given to pre-natal care. It is only in recent years that medical schools and obstetric clinics have given due attention to the care of the pregnant woman; and there are those who believe that eclampsia is wholly unnecessary, such as Davis and Edgar. Their claim may not be wholly correct, but it is nearly so.
DeLee states in a recent bulletin that since pre-natal care has been instituted in the Chicago Lying-In Dispensary it is very rare that a case of eclampsia comes in, and that in all his years of obstetrical experience only two of his pre-eclamptics have gone into convulsions. And the record of the Virginia Medical School Clinic is nearly as good. This, in my opinion, if true, should be an eye-opener to the average general practitioner, who, when confronted with a case of eclampsia in his own practice, has only two people to blame—either himself, for overlooking the pre-eclamptic condition, or the carelessness of the patient in not reporting regularly to the physician.

Since the pre-natal clinic at the University of Oregon Medical School was opened about three years ago, over five hundred cases have been registered, and not one of the eclampsia has developed in the clinic. These statistics put the onus of eclampsia squarely up to the attending obstetrician. I, myself, must take my share of the blame, inasmuch that I lost an eclamptic whom I failed to induce in time, in spite of the fact that confinement to bed, drastic purgation, extra water intake, milk diet with carbohydrate failed to clear up her marked albuminaria or reduce a diastolic blood pressure of 115 and a systolic of 180. In spite of these symptoms she felt very well. She had no headaches, no edema, no blurring of vision, and slept well. At the eighth month I told her husband and mother that I should take her to the hospital and induce labor, in view of these symptoms, which were not, of course, discernible to them. They persuaded me against my better judgment to allow her to go to full term, which I reluctantly did. She carried full term and had spontaneous delivery with the aid of a little chloroform. Two hours after delivery she had a severe convulsion. Eliminative treatment was instituted without avail, and she died in coma ten hours after delivery. I feel that the cause of her death was due to my inexperience, as I had ample warning of the severity of her pre-eclamptic condition.

Lowering of the present mortality due to eclampsia will be slow, because it depends first upon the education of medical students and physicians as to what good pre-natal care should be; and second, the education of the expectant mothers as to the importance of such care by the physician.

Briefly stated, pre-natal care for the prevention of eclampsia should consist of the following: The patient visits the physician's office early in pregnancy, at which time her history is obtained, special attention being paid to previous acute infections, especially scarlet fever and rheumatism. A complete physical examination is made, attention being given to infected teeth, tonsils, and ulcers on the body. The blood pressure is recorded, urine examined, instructions given as to diet and exercise and she is asked to report every three weeks up to six months. The patient is instructed to report if she suffers from headache, disturbance of vision, the swelling of feet, hands or face, dizziness, neuritis or constipation. During the last three months the patient is
seen every two weeks. If her blood pressures arise from normal to systolic 160 and there is a trace of albumin in the urine, with or without edema, she is put on a milk-carbohydrate diet and given an ounce of magnesium sulphate every morning. The amount of urine excreted is measured. If she does not improve, as indicated by a lowered blood pressure, lessening of the edema and albumin, she is put to bed and the treatment continued. Most cases will improve and go on to term without further difficulty; if not, and the patient’s condition becomes worse, as indicated by the blood pressure going higher, albumin increasing with casts, headaches becoming more severe, disturbance of vision, the important thing at this time is that her condition must be taken seriously. There has always been an attitude on the part of most physicians that the pregnant woman has some super-resistance to disease.

A non-pregnant woman with the above symptoms would be considered very ill and the attending physician would give her the closest attention; this is not so necessary when she is pregnant. If a patient’s condition remains more or less stationary and it is early in the viability of the child, then we temporize in the hope that the patient may be carried along, so that the possibility of getting a living child will be increased; but if she has reached the eighth and a half month of pregnancy, labor should be induced. However, if her condition becomes worse, as indicated by a high blood pressure with a systolic of 180 or 190 or a diastolic pressure of 100 and twitching of the muscles, disturbances of vision, vomiting, pain in the epigastrium, pregnancy should be terminated at once regardless of the viability of the child. The method of induction depends upon the case. If not too urgent, castor oil and quinine may be attempted; which in a small percentage of cases will be successful. Watson gives as high as twenty doses of 3 min. of pituitrin every hour. If this is not successful, the use of one of the various bags is quite effective in multipara and can easily be inserted through the cervix. In primipara, MacPherson advocates the use of a small rectal tube in the cervix and packing in the vagina. In early primipara near term, when it is very desirable to get a living child, I believe caesarean section is justified. I may just say in passing that last year I saw, with a colleague of mine, an elderly primipara who was very anxious to have a living child, but who presented all these pre-eclamptic symptoms in her latter months of pregnancy. My colleague temporized and carried her along to full term in the face of rising symptoms. She was sent to the hospital and her first pain at full term produced a convolution. Her blood pressure was 200 systolic, and 110 diastolic; casts in her urine and albumin, marked edema of her feet and some in her hands. On examination, the cervix showed no dilation and she was sent immediately to the operating room and caesarean section performed. We got a living baby and the mother made an uneventful recovery. I may say that during the post-partum period strenuous eliminating treatment was carried out.
In the treatment of eclamptic convulsions we have what is known as the radical or surgical and the conservative or medical methods. Until 1891 accouchment force was the only method in use. At that time Duresshen introduced abdominal hysterectomy and later vaginal hysterectomy. This method became very popular and spread rapidly all over the world. The surgeons were very much pleased with that method of delivery and that notion is still held by those who are unaware that the mortality has been reduced from 30 per cent when operative means are used to about 12 per cent by more conservative methods. In 1897 Stroganoff of Leningrad and about 1903 Tweedy of Dublin began the conservative treatment of eclampsia. Their methods are almost diametrically opposed and yet each has about the same percentage of mortality. In addition, there are the methods of MacPherson, of New York; Williams of Johns Hopkins, and quite recently the use of magnesium sulphate as reported by Lazard of Los Angeles; also the vetraum viride method of Gillespie of Cincinnati. This drug in the hands of most men has been very unsatisfactory and its use has been largely discontinued, therefore, we shall not consider it as a recognized method of treatment.

In February, 1925, Lazard, of Los Angeles, reported seventeen cases treated by the intravenous administration of magnesium sulphate with one maternal death, a mortality rate of 5 per cent. It is given in doses of 10 - 25 c.c. of a 10 per cent solution intravenously. One patient, who had had seventeen convulsions, received three 20 c.c. doses of the 10 per cent solution of magnesium sulphate within six hours; in addition, the patient had gastric lavage and 3 oz. of magnesium sulphate were left in the stomach together with colonic flushing of soda and glucose. The danger to be feared in its use is the paralyzing effect on respiration. This is overcome by the intravenous administration of 10 c.c. of a 2.5 per cent solution of calcium chloride, which should always be in readiness if respiratory embarrassment is exhibited; however, this rarely occurs. Future reports on the use of this salt will be watched with much interest, and it undoubtedly will be added to the list of helpful treatment in the care of eclampsia.

The conservative treatment by Williams has reduced the mortality to 12.8 per cent., whereas previously to 1912 the old radical treatment was 22 per cent. His regime is summarized as follows: first, the patients are placed in a quiet, darkened room and are disturbed as little as possible. I believe in the past this absolute quietness has not been emphasized enough by medical teachings; so essential is this that Stroganoff chloroforms his patients for all and every manipulation even if they have to be moved from one room to another or for gastric lavage or colonic irrigation or any intravenous medication. Secondly, a hypodermic injection of ½ gr. of morphine sulphate is given at once and may be repeated in the presence of undue restlessness or repeated convulsions, but not more than ½ gr. is given in the first twenty-four hours. The Rotunda treatment has discontinued the use of morphine
on account of its tendency to lessen bowel secretions. Third, the patient is kept turned on one side with the foot of the bed elevated as long as coma persists. Mucus is swabbed from the pharynx as it collects. Four, venesection after the second convulsion is performed under a nitrous oxide anesthesia if necessary, and 1,000 c.c. of blood is withdrawn unless the systolic blood pressure falls below 100 m.m. or the pulse rate shows alarming change under the process. This is, of course, contra-indicated in the presence of marked anemia. Five, water is given freely, if desired when conscious. Those who drink on account of coma are given 500 c.c. of 5 per cent glucose solution intravenously, which may be repeated in 12 hours. Six, no attempt is made at delivery until the cervix is fully dilated unless some definite maternal indication apart from the eclamptic condition is present; herein is the great difference between the conservative and radical treatment. The old radical treatment used accouchment force in many forms such as forcible dilatation, high forceps, vaginal hysterotomy, version, etc. It will be noted that no attempt is made at delivery until the cervix is fully dilated unless there is some other definite indication other than the eclamptic condition. The results of the Johns Hopkins Clinic have been quite satisfactory in spite of the danger of removing 1,000 c.c. of blood with the prospect of losing more at the time of delivery.

MacPherson, who follows the old Rotunda method of treatment, reports a mortality rate of 9.3 per cent. The method is outlined as follows: first, the patient is placed in a dark room. Second, the blood pressure is taken and a catheterized specimen of urine is taken. If the systolic blood pressure is over 178 mm. bleeding is done until pressure is 150 mm. Then lavage leaving two ounces of castor oil in the stomach, with colonic irrigation of 5 gallons 5 per cent solution. Third, morphine sulphate ½ gr. on admission followed by ¼ gr. every hour until the respirations drop to eight per minute. He states that at this time convulsions have usually ceased; the patient will be in labor as it happened in practically all of his cases, and will be delivered normally or by easy forceps in a short time. The routine is as follows: gastric lavage with a gallon of soda bicarbonate (1 dram. to the pint) and at the conclusion of the washing a pint of soda solution with 4 ozs. of mistura senna compound or 3 ozs. castor oil is left in the stomach. The bowel is first washed out with one or two quarts of soap enema, then a colonic irrigation is given with a No. 24 stomach tube passed into the colon about eighteen inches, using five gallons of the soda bicarbonate solution, one or two pints of soda solution with four ounces of purgative being left in the bowel. The bowel irrigation is repeated every five hours. During convulsions the patient is placed on her side, a gag between her teeth and mucus swabbed from the mouth. As soon as possible purgatives are given by mouth every five hours until the toxemia disappears. The patient receives no nourishment except water until there is a large increase of urine. In cases of profound coma with little or no edema, bloody urine, thirty to forty ounces soda
bicarbonate is given under the breasts, this being repeated every six hours. If delivery does not take place normally, nothing is done until the head is found to be in the vagina; it is then delivered by forceps. By using the above method, Tweedy has recently reported twenty-nine cases without a maternal death. This line of treatment seems admirably suited to the general practitioner and is probably as good as any.

Stroganoff, whose method of treatment has never gained favor in this country, largely on account of the use of chloroform and chloral, lays claim after treating 823 cases by his older and what he calls newer improved prophylactic method, to the possibility of obtaining the following results in non-neglected cases if his method is strictly adhered to: first, recovery of mothers from eclampsia, 100 per cent. Second, immediate cessation of convulsions in approximately 90 per cent as soon as treatment is commenced.

Stander, of the Johns Hopkins Clinic, visited Stroganoff early in 1924. He was so impressed that in October of the same year there was adopted at the Hopkins Clinic a similar regime except that chloroform is forbidden. This method may be best understood by giving the first twelve hours of a case reported by Stroganoff which is as follows:

The patient was immediately given chloroform, 2 drams, for a period of ten minutes, after which the systolic blood pressure was 138 mm. and 90 mm. diastolic. As soon as a hypodermic of morphine could be prepared 1/2 of a gr. was given while the patient was under chloroform. This was given mainly on account of the possibility of eliminating the noise of street traffic. The case proceeded as follows:

11.55 a.m. Blood pressure unchanged, pulse rate 70, temperature 98.6.
12.00. Four and one-half ounces of urine removed by catheter under chloroform, 2 1/2 drachms. Analysis of urine: acid, sp. gr. 1.024, albumin, no sugar, very many casts (granular); epithelial cells present, leucin and tyrosin absent.
12.15 p.m. Chloroform 45 m.
12.15 p.m. Per rectum chloral hydrate, gr. 30, milk 2 ozs. Normal saline, 3 1/2 oz. under chloroform 20 m. (5 drachms total). Part of this was returned per anus in fifteen minutes. Therefore, at 1.25 p.m. Per rectum chloral hydrate gr. 15, milk 1 oz., normal saline 13 drachms, under chloroform 20 m.
2.25 p.m. Morphia one-fourth gr. subcutaneously chloroform 30 m.
4.25 p.m. Per rectum under chloroform 45 m., chloral hydrate gr. 15, milk 1 oz., normal saline 1 1/2 oz.
6.25 p.m. Rectal injection repeated without chloroform. Blood pressure 154, temperature 98.4°, pulse rate 70, sleeping.
9.45 p.m. Per rectum under chloroform (40 m.) chloral hydrate gr. 15, milk 1 oz., normal saline 1 1/2 oz. Urine passed voluntarily, 6 oz., patient moved under chloroform to another ward.
Chloroform, total 7 drachms; chloral hydrate 1 1/2 drachms and morphia 7/12 gr.; urine 16 1/2 oz.
Following day patient received: 2.00 a.m., chloral hydrate 15 gr. by mouth; 5.35 a.m., chloral hydrate 15 gr. by rectum; 10.10 p.m., chloral hydrate 23 gr. by mouth, cascara 2 drachms.
The next day: 8.00 a.m., chloral hydrate, 23 gr., and during the following two days the patient received 68 gr. of chloral in doses of 15 to 23 gr.

It will be noted that Stroganoff uses chloroform to prevent convulsions during the handling of the patient. It is seen that he uses it
when he takes the blood pressure, catheterizes, gives rectal instillations, or moves the patient from one room to the other. My observation has been that in this country we are less careful in respect to preventing convulsions. Nearly all of us have seen patients have convulsions, when they are taken from their bed at home and put in the ambulance stretcher; again when first put in the hospital bed and again when placed in the hospital stretcher and another when put on the operating table. It would seem that a patient with eclampsia, who lives many miles from a hospital, would have a less number of convulsions if she were left at home to be treated by some conservative treatment and at the same time she would be less apt to have more convulsions, making her chance of recovery more likely.

In conclusion, I would state that the general practitioner must depend largely for guidance on the men who see large numbers of eclamptics in the large institutions. One's own practice does not allow him to form an intelligent opinion, on account of the scarcity of eclamptics. One should bear in mind that De Lee and Williams in a large number of cases at their respective clinics have had no eclamptics—that is no eclamptic that went to the convulsive stage. One must necessarily draw the conclusion that if the general practitioner concentrates his energies and intelligence on painstaking pre-natal care that he will have very few eclamptics go to convolution and require these various treatments. Nevertheless, they do occur from time to time, and one should have at his finger-tips the most modern and efficient treatment that has been devised in recent years. All of these strictly eclamptic treatments leave out the reckoning extreme accouchment force, which makes the operating room unnecessary. He can with the aid of skilled nursing carry out any of these lines of treatment, or variations of such, without the aid of a hospital; and that the idea that every eclamptic must be rushed to a hospital and probably placed in a private or semi-private ward in view of this treatment, would seem to be a lot of unnecessary handling. The patient must have skilled nursing, which I believe can be carried out to advantage in her own home. Most certainly, all these men are agreed that severe eclampsia should not be handled or moved any more than necessary. This phase of the treatment—the prevention of convulsions—has not been emphasized enough in recent teachings, as they are all agreed that the more convulsions the patient has the less liable the chances are for recovery. The modifications, such as Johns Hopkins, using no chloroform; Stroganoff using a lot of chloroform; the Rotunda using no morphine but a lot of high colonic irrigations, and Stroganoff using no colonic irrigations but much chloral hydrate may be followed as the general practitioner individually sees fit. They are certainly all agreed that there is no indication in eclampsia of radical accouchment force.
Abstract Anemia as a Problem in Diagnosis and Treatment*

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Toronto

Anemia develops from a disturbance of the blood-forming organs—bone marrow and spleen. It may be caused by loss of blood, increased destruction, defective or deficient formation of blood. The known etiological causes of anemia are hemorrhage, infections, malignant growths, chemical poisons, food deficiencies and pregnancy. In the majority of instances the blood-forming organs are affected secondarily to other systems of the body and the anemia is merely a symptom and a late manifestation of the primary condition. Less often disease affects the blood-forming organs primarily, and anemia is one of the chief and primary symptoms present. In the former, special treatment of the anemia is seldom indicated, the type of treatment depending on the nature of the primary trouble. With primary disease of the blood-forming organs special treatment for the anemia is necessary but must take into consideration the origin and causes of the disturbance in the blood-forming organs. In the anemic patient, therefore, the cause of the anemia must be found before an intelligent form of therapy can be prescribed.

The clinical diagnosis of the presence of anemia is usually not difficult and can be confirmed by the estimation of the hemoglobin. On the other hand, the search for the cause of the anemia present is often difficult, demanding a thorough and complete examination of the patient, including a hematological examination of the blood. For the purposes of investigation, cases of anemia may be roughly classed as follows: (1) Cases with symptoms of anemia plus symptoms characteristic of diseases of a system or systems of the body other than the blood-forming organs; e.g., duodenal ulcer with hemorrhage, carcinoma of the stomach, rheumatic fever, pulmonary tuberculosis, etc. (2) Cases with symptoms of anemia with chronic splenomegaly; e.g., Banti's syndrome, bacterial endocarditis, familial or acquired hemolytic jaundice, myeloid leukemia, etc. (3) Cases of anemia with special signs and symptoms; e.g., periodic sore tongue and numbness and tingling—pernicious anemia; recurring attacks of mild jaundice and splenomegaly, with or without a family history of jaundice—familial or acquired hemolytic jaundice. (4) Cases with symptoms of anemia plus purpura; e.g., essential thrombocytopenic purpura, subacute bacterial endocarditis, etc. (5) Symptoms of chronic anemia with general debility; e.g., anemia due to focal infection.


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If one recalls the known etiological causes of anemia while taking the clinical history and making a physical examination, suggestive clues as to the primary causes of the patient's anemia are often found, e.g., tarry stools—duodenal ulcer with hemorrhage; hematemesis in the young with splenomegaly—Banti's syndrome; fever—an infection; loss of weight in middle life—new growth; the type of occupation—poisoning by lead, benzol or arsenic. Many other examples might be given. Following this plan of investigation, different cases of anemia will tend to fall into the groups suggested above, and the possible causes of the existing anemia become more clearly defined. Having determined the group to which the case belongs, a consideration of the results of a hematological examination with the symptoms and physical findings present, usually makes clear the cause of the anemia and the type of treatment indicated.

It is impossible in the time available to discuss in any detail the treatment of the anemic patient. Effective treatment demands a correct and complete diagnosis and is determined by the cause or causes of the anemia found. Symptomatic treatment alone has no place in the treatment of the anemic patient. If the blood-forming organs are affected secondarily (Group 1) the treatment is that prescribed for the primary condition and, if effective, will usually cure the anemia. If the anemia is related to conditions causing splenomegaly (Group 2) treatment must be based on the cause of the splenomegaly: radiation therapy in leukemia and Hodgkin's disease, or splenectomy in Banti's syndrome, familial or acquired hemolytic jaundice, or essential thrombocytic purpura.

In the treatment of Addison's "pernicious" anemia special treatment is necessary: rest in bed until the hemoglobin is over 60 per cent is very important. If the patient is given daily 150 - 250 grams of liver or a potent liver extract the equivalent of 400 grams of liver, digestive disturbances are soon relieved and the anemia is cured in four months or earlier if the patient adheres to treatment and no infection is present. Minor neurological disturbances are relieved but the more severe ones yield very slowly to treatment and liver appears to be more effective than liver extract. It is specially important, therefore, that cases of pernicious anemia be treated early before severe neurological manifestations develop and that the hemoglobin be maintained at 90 per cent or over by continuous treatment to prevent the development of neurological changes or the aggravation of existing ones. Too many pernicious anemia patients are going about partially treated. The physician should insist on patient reporting monthly for re-examination.

One of the many disturbances caused by focal infection is anemia. It is important to search carefully for foci of infection and have them eradicated if possible at a suitable stage in treatment. This applies to the treatment of all cases with anemia. Iron in adequate dosage is of value in certain forms of anemia, particularly those due to infection or chronic hemorrhage. Transfusions of blood are indicated in acute
hemorrhagic anemia, to assist in controlling bleeding and in patients with severe anemia requiring operation. As a general measure in the treatment of anemia, transfusion of blood is not to be recommended.

If the physician will make it a rule not to be content with a symptomatic diagnosis of anemia, but consider the presence of anemia like fever, an indication for a complete examination of the patient to ascertain the cause, the treatment of the anemic patient will be much more satisfactory to both the physician and patient.

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New Subscriptions—
Acta orthopedica.
Bulletin of hygiene (British).
Pharmaceutical journal.
IN 1861, a student at the Medical College of Ohio, with a graduating dissertation on epilepsy, was wrestling with reams of medical literature for references on the subject. That the exercise was rather strenuous is shown in the aim he made, viz.: "to establish for the use of American physicians a fairly completed medical library, and in connection with this to prepare a comprehensive catalogue and index which should spare medical teachers and writers the drudgery of consulting ten thousand or more different indexes or of turning over the leaves of as many volumes to find the dozen or so references of which they might be in search." The result of this aim was the "Specimen Fasciculus" which appeared in 1876, after Billings—the erstwhile student—had experimented with all known methods of cataloguing and indexing from the ordinary type of author entry to the Dewey system.

Shortly after this event, Billings acquired an able and loyal assistant in the learned Dr. Fletcher, and the Surgeon General's Library became the birthplace of the Index Catalogue and the Index Medicus. As plans for the publication of the Index Catalogue approached fruition, it became apparent to the two editors that while such a publication might give the cream of the world's literature up to the date of the particular lettered volume (B. 1881, C. 1882) on each individual subject, there was need of a current periodical index of the newer medical literature. Thus the Index Catalogue became the parent of the Index Medicus which appeared in January, 1879, as "a monthly classified record of the current medical literature of the world."

Up to 1900, the attractive feature of the Index Medicus to university professors and research workers was its appearance in monthly numbers. While, as Garrison has said, they afforded to men like Welsh or Osler or Jacobi a purview of their specialties or of the whole field of medicine, as pleasant and arresting as were the morning Spectator or Tatler to the eighteenth century Englishman, the subscription list never exceeded five hundred. From the start it was evident that a journal of this type financed by private enterprise was foredoomed to
financial disaster. In 1899, with a history covering the bankruptcies of two publishers, it suddenly collapsed. And so ended the first series of the Index Medicus.

The interim 1900-1902 would represent a complete break in current medical bibliography were it not for the attempt made by three French doctors to carry on. To Drs. Marcel Baudouin, Potain and Richet our library owes the three volumes of the Bibliographia Medica.

Then, in 1903, thanks to the Carnegie Institute of Washington, the Index Medicus was revived and assured of handsome and loyal financial support. The second series ran from 1903 to 1920, the third from 1920 to 1926. From 1916 to 1926 there ran parallel with the Index Medicus the Quarterly Cumulative Index of the American Medical Association. In 1926 an auspicious event occurred in the medical world for it was decided that the Index Medicus and the Quarterly Cumulative Index were—

"This day to be conjoined
In the estate of honorable marriage."

The natural sequel was the appearance early in 1927 of the first issue of the Quarterly Cumulative Index Medicus. At the present time it is the only general medical index in English, and for English readers anything left out of it might just as well have never been written.

The chief points of merit of the Q. C. I. M. in regard to its use are that:

First, it indexes itself alphabetically both as to authors and subjects.
Second, subject entries are brief English extracts of the actual content of the article indexed without reference to titles unless accurately descriptive.
Third, for the benefit of foreign research workers, the author titles are presented in full in the original language, with the exception of Slavic, Scandinavian and Oriental, which are given in English translation.
Fourth, the quarterly material is accumulated semi-annually in two volumes. The new books and pamphlets of each trimester or semester are presented apart, by authors and subjects, with attached prices and the addresses of the publishers.

In these days where all the applied sciences are inter-related, the proper classification of many complex titles becomes a difficult matter. As only one entry of such titles can usually be made, it becomes necessary to find the centre of gravity of the title. Thus articles on the oculocardiac reflex, the effort syndrome (D. A. H.) and latent cerebral tumor in a case of epilepsy, find their places automatically under Reflex (oculocardiac), Heart (neurosis) and Epilepsy (Pathology). The growth of medical literature has been so intensive and the range of titles so extensive that Fielding Garrison of the Surgeon General’s Library has been driven to exclaim "The curse of recent medicine is the gigantic, senseless proliferation and prolixity of its periodical literature. It has been the despair of librarians, the enslaver
of indexers, the nightmare of classifiers, the bugbear of editors, and even in Dr. Fletcher's time was the occasion of his celebrated witticism: 'The whole world is in a conspiracy against medical bibliographers.' Some day there may be minor Nobel prizes for those who decline to write unless they have something really new to say and who can state their intention in titles of telegraphic brevity.'

Despite these drawbacks—perhaps because of them—the Q. C. I. M. is able to exert a decisive, preponderant effect upon the quality and reliability of our medical literature. When one considers that each volume brings in round numbers 56,000 titles and sources of literary contributions including monographs, or a total of 112,000 annually, its manifold usefulness is more than apparent. It is worthy of the sincere support of the medical profession the world over. The fact that there are as many "new" sciences as there are up and coming nations makes the Q. C. I. M. for all those seeking information the guiding star through a sea of books.

Special reference should be made to the urogenital work of Jacob Henle (1809-1885). He discovered cylindric casts in the urine; pointed out that varicocele is almost invariably left-sided; described the expanded outer half of the Fallopian tube, known as Henle's ampulla; the portion of the uriniferous tubule, known as the canal of Henle; the granular mononuclear cells in the seminiferous tubules, known as Henle's cells; the fibrin formed by precipitating semen with water, known as Henle's fibrin; the remains of the gubernaculum surrounding the vas deferens and vessels of the spermatic cord, known as Henle's internal cremaster; and the striated muscular fibres encircling the prostatic and membranous urethra, known as Henle's sphincter. But his most interesting find in this field was the U-shaped turn of the uriniferous tubule which is formed by a descending and an ascending loop-tube, known everywhere as Henle's loop. Concerning this discovery, the fortunate Henle wrote one of his characteristic notes to Pfeufer.
Physio-Therapy
In the War Memorial Children's Hospital of Western Ontario.

KATHARINE M. H. DICKER, C.S.M.M.G.

The aims of physio-therapy treatment fall into three classes, namely:
Prevention; Correction; and the Sustaining of the Correction. Under Prevention comes the removal of cause or counteraction of tendency before the inevitable deformities arise. The term Correction is self-explanatory; but sometimes the importance of Sustaining the Correction is overlooked. By whatever means the correction is produced, this cannot be sustained unless constant after-care or follow-up treatment is given, especially in growing children.

Prevention is a part by itself, and an increasing field of work, of which the ideal is that it should eventually supplant correction. But of Correction and the Sustaining of the Correction, perhaps 25 per cent. of physio-therapy work should apply to the former and 75 per cent. to the latter.

The physio-therapy service of the War Memorial Children's Hospital has grown, since 1925, from 7 patients, with a part-time masseuse who, without any special accommodation for her work, went from bed to bed giving treatments, until today there are seventy patients and a department equipped with apparatus to aid in corrective exercises and means for giving the following types of treatment: ultraviolet, infra-red rays, faradism, radiant heat, and whirlpool baths.

During the past two years there has been a great deal of voluntary help rendered by the Junior Club, both in assistant duties in the Hospital, and in taking children to and from it for treatment; however, since they bring in many patients who would otherwise be unable to attend, their efforts in this latter direction put the department in the peculiar position of requiring more help, the more help it receives.

The increase in the number of patients necessitated an increase in the personnel, and for some time past there have been one full-time and one part-time masseuse, while, during the past year, the work has grown so much that steps are now being taken to add still further help.

By far the best results are obtained if treatment is begun early. Congenital deformities, such as torticollis and club feet, if treated from the first two or three days of the baby's life, can nearly always be entirely corrected, without operation, within the first two or three years. If these conditions are not treated till the child is two or three years old, bony changes take place which can never be entirely rectified. Also if fractures are given physio-therapy in the early stages, there is much less likely to be stiffness of adjacent joints and wasting of muscle, due to disuse. Anterior poliomyelitis, if treated after the temperature has been normal for from ten to twelve days, shows much less wasting and contracture, and more function, than cases which are left for several weeks or months before commencing physio-therapy.
A large number of our cases are of anterior poliomyelitis. These children receive massage to prevent wasting of muscle; heat to improve the circulation; and re-education of muscles to help improve their function. There are several children with spastic paralysis at the present time. Exercises for co-ordination form a large part of their treatment. Quite a few cases of paralysis due to traumatic injury have been treated with success, of late. One such case of peroneal paralysis was treated recently with faradism, heat and massage, and responded remarkably well.

Diathermy is available for abdominal distention, pneumonia and many other conditions if prescribed.

The whirlpool bath recently installed gives another means of treating chorea, certain types of heart cases, and conditions where the circulation is impaired.

The mercury vapor lamp which we have had for three years has been responsible for completely or partially curing many skin diseases, such as eczema; curing or suspending rickets, tuberculous joints and wounds; and helping to unite non-union fractures of long standing. It also has been the means of increasing the calcium content of the blood in anaemic infants.

We have, in addition, a carbon-arc lamp which is used principally to give combined ultra-violet and infra-red treatments for rickets and general debility.

At the present time about twenty children are being treated in a class for postural defects. Most of these bad postures are due to rapid growth and malnutrition, while some are torticollis cases in the later stages of cure, and a few are due to uneven length of leg owing to congenital defect or tuberculous joints.

In cold weather the class is conducted in the gymnasium where there is a mirror occupying almost one entire wall, in which all the children can see themselves while performing their exercises. One is often hardly conscious of a poor posture unless confronted with it; and good posture is much more easily obtained in this manner. Many of the exercises are given to gramophone music, as it is found that the children respond in a more spirited manner with music than without.

As soon as it is warm enough, the class is taken on the roof in the fresh air and sunlight. At all times these exercises are taken in the minimum of clothing, so that there is freedom of movement and a more accurate supervision of posture. Children with exceptionally poor posture are given individual attention.

Fourteen children are now attending a remedial swimming class. Most of this number are polio cases, and a few are children suffering from general muscular weakness. It is surprising how much easier it is for weak muscles to function in water than in air; some of these children who have very unstable gait and weak arms can swim quite well.
The Hospital is fortunate in having a very fine splint department. This refers both to the quality of the splints, which are lightly and soundly made and of excellent fit; and also to the ready and helpful spirit of co-operation which that department gives at all times.

Such is the good work of the clinics, service clubs and public health nurses, that the number of staff cases is considerable; yet there is a great interest taken in private cases, and every possible individual attention given to them.

It is a very difficult problem for the Hospital to come in contact with all the children who require this form of treatment, in spite of the excellent work being done in that direction by medical agencies and other organizations; but the writer feels strongly that the first step toward our ideal of Prevention is the detection, in its earliest stages, of any case suitable for physio-therapy, and the commencement of treatment as soon as the condition is discovered.

Editorial

(FROM THE DIPLOMATE, JUNE, 1929)

The following is by L. Clark Hepp, Chairman of the Honor Committee, of the University of Colorado School of Medicine, in answer to the query “How does the Honor System work at the University of Colorado School of Medicine?”

“DURING the fall of 1916 some of the students noticed that there was cribbing going on in examinations even when the instructor was present. This caused considerable ill feeling among certain students because those who were known to be dishonest often received the best grades. When one of the instructors was approached with the problem he stated that the honor system might be instituted and possibly this trouble could be done away with. A group of students became interested in the question and in a short time had drawn upon a set of rules and a constitution which were presented to their class for adoption. Slight alterations were made and later the proposal was adopted unanimously by all the medical students. This Constitution of the Honor System, as it was called, was then presented to the faculty who heartily approved it and accepted it without any amendments or changes. It became effective the next school term. This same Constitution is the one used today at the University of Colorado School of Medicine with but one slight change.

“Before entering the School of Medicine the prospective student receives a letter explaining the Honor System. When he enters the school, he signs the following pledge: ‘On my honor, I pledge to abide by and support the Constitution of the Honor System of the University
of Colorado School of Medicine so long as I remain a student in this institution.'

"All violations of the Constitution of the Honor System are dealt with by an Honor System Committee, which is composed of five students, one from each of the first three classes, and two from the senior class. One of the seniors is elected as chairman. Any student or faculty member observing or suspecting cheating is honor bound to report such case to a representative of the committee within five days. The accused is given an opportunity to appear before the Committee to defend himself against the charges made. No witness appears before the accused. In case the committee finds the accused guilty it meets with a committee of the Executive Faculty to review the evidence and make recommendations.

"The Honor System applies to both written and oral examinations. Violations consist not only in cheating in any examination or class exercise, but also in falsifying attendance records regarding obstetric work or autopsies. Violations also include the committing of any dishonorable act in relation to the medical school.

"The students feel that the Honor System works exceptionally well. In the thirteen years that it has been in force, a number of cases have been investigated, three men have been expelled from school, and one man was suspended for one quarter. As students, we feel that proper conduct at the school is our individual responsibility. This not only applies to examinations but to general conduct about the school. It seems rather unusual to see forty students all in one room taking an examination without a single instructor in the room, and then suddenly four or five men get up, go out and have a smoke, and return to the examination and continue writing without the least suspicion connected with the act. A student is not permitted to talk about the examination while it is in progress, but he is free to go any place in the building providing at least one other student accompanies him. A list of 'suggestions' in regard to 'proper conduct' is printed and distributed to new students for their guidance.

"There is very little possibility for personal grudge or favoritism to enter into such a system. Each individual is a committee of one to keep things straight. The system originated with the students and is run by them. The faculty is happy over this arrangement and they have the utmost confidence in the students' ability. The modern school of medicine is practically a graduate school. Because the students are older and more serious minded they are interested in a common cause, Medicine. This common purpose unites the students more closely, and a spirit of co-operation is more easily attained than is possible in a general university with its varied interests. The Honor System creates a better feeling among the students, and the faculty does not have to waste its time with our troubles. The punishment administered to the offender is perhaps more severe than it would be if the case were to be settled by the faculty alone, yet the Honor Committee of the Executive Faculty is a check on the students in judging the severity of the act and in recommending the punishment. If a student is inclined to be dishonest, this system does one of two things to him: it either makes him honest or else he leaves the school.

"From the students' point of view, the Honor System stimulates scholarship, places all students on an equal footing, serves to strengthen the moral fibre of the weak, and incidentally keeps out of the medical profession those who would become dishonest practitioners."
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