amount of reserve in view of the unusual history and symptoms. Immediate operation was decided upon.

Operation.—Under ether anaesthesia a right pararectal incision was made, and on opening the peritoneal cavity some free, non-odorous, blood-stained fluid at once escaped. The right iliac fossa was isolated with gauze swabs and the caecal region exposed without difficulty. The appendix showed nothing more than a superficial congestion—evidently a concomitant feature and not the cause of the abdominal lesion. A rapid appendicectomy was performed. On removing the gauze swabs one had not far to seek for the true lesion. Adherent to the anterior abdominal wall of the hypogastrium by recent adhesions was a coil of small intestine, afterwards located to the distal part of the ileum. The adhesions were easily broken down and the affected coil of gut was delivered through the wound. The length of this coil was roughly estimated at about 18 inches, and the distal limit of the disease was apparently some 8 inches from the ileo-caecal valve. It was intensely congested, much thickened, almost black in colour, and there were associated with it enormously enlarged mesenteric glands. Excision of the coil was decided upon and carried out, care being taken to include some three or four inches of apparently healthy bowel on both proximal and distal sides. The mesentery was divided close to the bowel and an end-to-end anastomosis established. Drainage of the pelvis was effected by a small rubber tube through a stab wound in the suprapubic region, and the original wound was sutured in layers. The whole operation occupied rather less than one hour, and the boy's condition was, under the circumstances, quite satisfactory.

After Treatment.—Saline infusions per rectum were given for 24 hours and then discontinued. The temperature fell to normal after the first 24 hours, the pulse-rate decreasing *pari passu*. The drainage-tube, after draining an inconsiderable quantity of blood-stained fluid, was removed on the third day. The bowels acted well on the third day after calomel had been administered in one-grain doses hourly for four hours. The boy made satisfactory progress for two weeks, at the end of which time he had a recurrence of his original symptoms—blotchy rash on the forearms and legs, slight swelling of the knees, streaks of blood in the stools, and paroxysms of colicky pain. Salol, 5 gr., and a mixture containing sod. bicarb., 5 gr., and tinct. belladonna, 5 minims, were administered. The pains ceased during the next 24 hours, and the rash and the joint signs gradually disappeared. The patient subsequently made rapid progress, and returned home one month after the operation, apparently in normal health.

Pathology.—The specimen was forwarded to the Public Health Laboratory, York-place, Manchester, for examination, and I am indebted to Dr. G. D. Dawson for the following report:—

"The specimen consists of 26 inches of the ileum. The whole bowel, but especially the middle third, is dark red in colour. Near the mesentery the peritoneum, elsewhere smooth, has a limited but thick covering of fibrinous deposit; here the bowel wall is puckered. The mucosa is deeply congested, swollen, and covered with thick mucus. Enormous ulcers are present along the whole length of the specimen. Towards the end they are small, but in the middle third one oval ulcer measures $1\frac{1}{2}$ in. in length by $\frac{1}{2}$ in. wide, and another $2\frac{1}{2}$ in. long by $1\frac{1}{2}$ in. wide. In every case the margin is irregular, the base ragged in appearance.

Microscopical Examination.—(a) Transverse section through large ulcer. On either side of the ulcer the deeper parts only of the mucosa are present; the rest is more or less disintegrated, and the surface is covered with a layer, sometimes of considerable thickness, of degenerated epithelial, and other cells held together by mucus. The mucous membrane is absent from a large part of the surface, the ulcer extending to the submucosa. The submucosa, especially in the region of the ulcer, is much swollen and cedematous, and contains numerous extensive hæmorrhages (the pigment is partly diffused) and some large areas of small-celled infiltration; a large proportion of these small cells are of the polynuclear type. Beneath the ulcer the structure of the muscular coats is indistinct; there is much cedema and hæmorrhagic infiltration. At either side hæmorrhages are common in the muscular coats.

(b) Transverse section through less affected part. The changes are similar in type but less advanced. There is the same superficial necrosis of the mucosa, which, however, is nowhere completely absent. Interstitial hæmorrhages are numerous in the interglandular stroma and in the submucosa. The section passes through a group of much enlarged lymphoid follicles.

(c) Sections stained for bacteria. No Gram-positive organisms are present. In the mucosa, but not in the deeper layers, there are numerous clusters of medium-sized Gram-negative bacilli (probably *B. coli communis*).

Cultivation.—Cultures from different parts of the inner surface gave pure growths of *B. coli communis*.

Inoculation.—On inoculation into guinea-pigs cultures of the *B. coli communis* isolated did not appear to be unusually virulent.

Conclusion.—Acute enteritis of phlegmonous type with extensive hæmorrhagic extravasation."

(It is necessary to state that the above examination for bacteria is inconclusive, as I, unfortunately, placed the specimen in a weak solution of formalin before conveyance to the Public Health Laboratory.)

Comments.

The chief features of interest in this case are the differences which separate it from the usual type—notably, the age of the patient, the site of the lesion, and the apparent predisposing cause. The fact that the disease can occur in a child makes it probable that no age is exempt, and it would appear that in children, phlegmonous enteritis—either primary or secondary—occupies a place in the consideration of the differential diagnosis of acute intussusception. The unusual site of the lesion in the lower intestine is difficult of explanation, but there is no question that such a site is a distinct advantage, offering greater facility for operative measures and consequently a much more favourable prognosis.

It is interesting to note the apparent similarity between the initial subcutaneous cellulitis and the subsequent phlegmon of the intestine. Whether the latter had any causal relation with the former is mere conjecture, the difficulty being to discover a reason which will adequately explain the connexion.

References.—1. Taylor, G., and Lakin, C. E.: THE LANCET, 1911, ii., 224. 2. MacCallum, J. B.: Johns Hopkins Hosp. Bulletin, 1906, p. 254. 3. Adams, J. E., and Cassidy, M. A.: Acute Abdominal Diseases, p. 285. 4. Zoepffel, H.: Deutsche Zeitschr. f. Chir., 1920, p. 266.

LOSS OF WEIGHT IN CASES OF ARTIFICIAL PNEUMOTHORAX.

By L. S. T. BURRELL, M.A., M.D. CAMB.,
M.R.C.P. LOND.,

PHYSICIAN TO BROMPTON HOSPITAL FOR CONSUMPTION AND DISEASES OF THE CHEST; ASSISTANT PHYSICIAN TO THE WEST LONDON HOSPITAL;

AND

MURRAY Y. GARDEN, M.D. ABERD.,

ASSISTANT RESIDENT MEDICAL OFFICER, BROMPTON HOSPITAL.

IN artificial pneumothorax treatment the time-honoured background in a typical picture of progress—namely, the gain in weight—is frequently absent. As a rule when this treatment is started there is a drop in weight for the first few weeks. This loss, however, though disquieting at the time, is generally made up as the treatment continues; but usually when one lung is collapsed the normal weight is never reached, although the weight obtained is greater than it was at the beginning of the treatment. A certain small number of cases, on the contrary, gain weight from the beginning. Others after the initial loss of weight do not begin to gain—except possibly for a short time—but continue to lose steadily, and in one case in particular this loss assumed alarming proportions.

Impaired Metabolism.

We think this loss can be explained on the basis of an impaired metabolism; for it is not difficult to imagine that the collapse of one lung will cause a general metabolic upset to which the body has to accommodate itself. As we hope to prove in a subsequent paper, efficient aeration of the blood becomes more difficult, particularly if the movement of the uncollapsed lung is restricted by a displaced mediastinum. This opinion is supported by the work of Dr. S. W. F. Underhill.¹ The diminished oxygen concentration of the blood apparently causes an inefficient combustion of carbohydrates and fats. A consequent demand on the body fats is made by the

¹ Brit. Med. Jour., 1921, ii., 779.

liver. These respond with great readiness and there is a tendency to fatty infiltration of that organ. We have seen evidence of this in the post-mortem room, in cases where apparently no other explanation was forthcoming for the infiltration by the condition of the other organs. This reasoning explains the tendency to loss of weight and subcutaneous fat in these cases, especially under circumstances which call for an increase of the release of kinetic energy, such as is consequent upon dyspnoea or increased muscular exertion. As treatment continues, however, the uncollapsed lung seems to be able to accommodate itself to the altered conditions and to carry on oxygenation of the blood more fully; metabolism becomes more stable, and the patient begins to put on weight if his muscular activities are restricted. This has an important bearing on the later stages of pneumothorax treatment.

Displacement of the Mediastinum.

While this seems to us the most probable explanation of the initial loss of weight, we have to seek some other or some further cause in those cases where the loss continues. In these cases physical signs, supported by X ray findings, showed that there was a direct relationship between the displacement of the mediastinum and the loss of weight. In two cases conditions approaching pleural hernia were seen, and in one case the shifting of the mediastinum and the loss of weight were due to the stretching of an anchoring adhesion. In these cases, although the patients were placed under the most suitable conditions for gaining weight, the loss continued. When, however, the pressures were lowered and the mediastinum allowed to return towards its normal position, there was at once a gain in weight, and if the mediastinum was again displaced the loss in weight returned. In all these cases the first symptom was loss of weight, and if the displacement of the mediastinum was increased dyspnoea and cyanosis appeared. It seems quite clear to us that this is only a further stage of the initial loss which occurs at the beginning of treatment by artificial pneumothorax; that the uncollapsed lung, hampered by a displaced mediastinum, fails in its function of oxygenating the blood and thereby affects metabolism and causes loss of weight. If in addition there is dyspnoea the increased respiratory effort deprives the body, already over-taxed, of a large amount of energy. This is further supported by the fact that the small number of cases which gain weight from the first have shown either very slight or no displacement of the mediastinum.

From the above it might seem that under no circumstances in the course of artificial pneumothorax treatment must the mediastinum be displaced, but we have to use our discretion and treat each individual case on its merits. Different cases will stand different amounts of displacement of the mediastinum before symptoms appear. Again, where adhesions have to be stretched at the expense of this displacement we may, in cases showing little or no emaciation, decide to bring it about, disregarding the consequent loss of weight, in order to obtain a more efficient collapse of lung, and later when the adhesions have stretched to allow the mediastinum to return. Naturally the circumstances which affect our decision, such as the character of the adhesions, the amount of displacement which can be caused with impunity, and the activity of the disease, vary widely in different cases. As a general rule we do not approve of high intra-pleural pressures, and if the pressure is raised in the hopes of stretching adhesions, it should be done with the utmost caution; if, as so often happens, the adhesions show no signs of stretching, the pressures should be lowered.

Illustrative Cases.

The following cases illustrate the relationship of the displacement of the mediastinum to the loss of weight. They are all cases of pulmonary tuberculosis in which tubercle bacilli have been found.

CASE 1.—Female aged 17. Infiltration and fibrosis of left upper lobe and apex of left lower lobe of lung. Before artificial pneumothorax was induced, even whilst on absolute rest, evening temperature was 99.6°–100.5° F. Patient admitted to hospital July 12th, 1921; pneumothorax was started six days later.

Table of Weights.

		st. lb.			st. lb.
July 12th, 1921	..	8 8	Nov. 28th, 1921	..	7 12½
Aug. 9th	..	8 9½	Jan. 2nd, 1922	..	7 9
„ 24th	..	8 8½	„ 24th	..	7 9
Sept. 11th	..	8 4½	„ 31st	..	7 10½
„ 20th	..	8 3	Feb. 14th	..	7 12
Oct. 20th	..	8 0	„ 20th	..	7 12½
Nov. 7th	..	7 12½	June 6th	..	8 5½
„ 14th	..	7 12½			

The initial gain, we think, was due to hospital feeding and rest. Then began the loss as the lung was collapsing. On Oct. 20th X ray showed good collapse of the left lung with slight displacement of the mediastinum to the right. So far the loss of weight was not disturbing, and the patient was up for six hours daily with normal temperature, pulse, and respiration. However, as the loss continued the patient was put back to bed; this stayed loss of weight until after the Christmas festivities, when the weight again dropped. On Jan. 12th, X ray showed considerable displacement of the heart and mediastinum to the right side, with bulging of the upper part of the mediastinum simulating pleural hernia. No refill was given from Dec. 30th until Feb. 10th when the mediastinum had come back towards its normal position. With careful control by X ray refills were given to prevent the re-expansion of lung without causing displacement of the mediastinum, and the patient began to gain weight.

CASE 2.—Female aged 22. Infiltration of upper and middle lobes and apex of lower lobe on right side with evening temperatures of 100° F. Artificial pneumothorax induced Sept. 20th, 1921.

Table of Weights.

		st. lb.			st. lb.
Sept. 14th, 1921	..	7 8	Dec. 30th, 1921	..	7 9
Oct. 3rd	..	7 10	Jan. 9th, 1922	..	7 10½
„ 22nd	..	7 12	„ 24th	..	7 8½
„ 31st	..	7 11	„ 31st	..	7 8½
Nov. 11th	..	7 12	Feb. 13th	..	7 9½
„ 28th	..	7 12	„ 20th	..	7 10½
Dec. 17th	..	7 11	June 6th	..	8 8½

The loss on Dec. 17th was due to the stretching of pleural adhesions allowing much displacement of the mediastinum. The pressures were lowered and the mediastinum returned slightly—hence the gain on Jan. 9th. The pressures were again raised and the mediastinum further displaced, and the patient once more lost weight. On allowing the mediastinum to return towards its normal position the patient began to gain weight. The treatment is being continued; the patient is afebrile and has no cough or sputum.

CASE 3.—Female aged 21. Infiltration of whole of right lung and slight infiltration of left apex. A right-sided pneumothorax was induced Oct. 27th, 1921.

Table of Weights.

		st. lb.			st. lb.
Oct. 22nd, 1921	..	9 6½	Feb. 14th, 1922	..	8 5
Nov. 14th	..	9 7½	„ 27th	..	8 5
Dec. 12th	..	9 5½	March 13th	..	8 5
„ 30th	..	9 1½	„ 27th	..	8 7
Jan. 9th, 1922	..	9 1	April 24th	..	8 7½
„ 24th	..	8 12	May 8th	..	8 8
„ 31st	..	8 8	„ 22nd	..	8 8½

The initial gain which usually follows hospital feeding and rest occurred, but gave way to a steady loss. On Jan. 30th the mediastinum was much displaced and the upper part of the mediastinum bulging to the left, simulating pleural hernia. By lengthening the intervals between the refills and regulating the quantity of gas given (at the same time taking care that the lung did not re-expand and produce adherence of the two layers of pleura) the mediastinum was allowed to return to the normal position. The loss in weight was at once stopped and she is still under treatment, but is now free from cough and sputum and in good general condition.

CASE 4.—Female aged 24. Infiltration of right upper and lower lobes. Pneumothorax induced Jan. 19th, 1922.

Table of Weights.

		st. lb.			st. lb.
Dec. 9th, 1921	..	7 9	April 10th, 1922	..	7 10
„ 20th	..	7 12½	„ 24th	..	7 11½
Jan. 9th, 1922	..	8 1	May 8th	..	7 12
March 13th	..	7 9½	June 6th	..	7 13
„ 27th	..	7 8½			

The initial gain due to hospital treatment is followed by loss continued up to the end of March. At that time the mediastinum was considerably displaced to the left. It

was allowed to return to the normal position, and the patient at once began to gain weight. The treatment is being continued, and she is free from cough and sputum.

As may be supposed, our theories regarding loss of weight affect our after-treatment of artificial pneumothorax cases to a large extent. Of course, each case has to be considered individually, and great departures may have to be made even from our general principles. During the transition period—that is, until the body has accommodated itself to the collapse of one lung—our aim is to increase anabolism and to decrease katabolism, and to do this the patient should be kept on absolute rest. This period varies greatly in different individuals according to the extent and acuteness of the disease, the number and nature of adhesions, the condition of the uncollapsed lung, and numerous other factors. In cases where collapse is being successfully obtained, where the pulse-rate remains good, the temperature normal after refills, the initial loss of weight slight, and no displacement of the mediastinum occurs, the initial period of rest need not be long. On the contrary, when the lung is held up by adhesions, when the general condition of the patient is poor, and when the temperature remains unstable, it is better to keep the patient on absolute rest, it may be for months, if his psychological condition will stand it. As the body accommodates

itself to the altered conditions, as evidenced by a gain in weight and a general feeling of well-being, we gradually extend the patient's activities, at first allowing him an hour on a couch. For the first six months at least the patient should lead a guarded life, and sudden or strenuous physical exertion should be avoided. Indeed, we think that great care should be taken throughout the treatment to prevent the patient from over-exerting himself. We do not mean that no patient should be allowed to work whilst undergoing artificial pneumothorax treatment; on the contrary, we have had many patients doing hard manual labour during the later stages of the treatment. But it must be remembered that the strain on the metabolic processes can be reduced by restricting the patient's muscular activities and output of energy, and we have seen many a patient who was not doing well improve and become a most satisfactory case after being made to take more rest, just as we have seen the same happy result follow the re-adjustment of the intra-pleural pressure by altering the intervals between the refills or the amount of gas given until the optimum pressure for the time was found.

We have to thank Dr. J. J. Perkins for the facilities and help he has given us; also the sister of the ward for her helpful suggestions and the care she has bestowed on the after-treatment.

Clinical Notes :

MEDICAL, SURGICAL, OBSTETRICAL, AND THERAPEUTICAL.

A NOTE ON
PAROXYSMAL TACHYCARDIA
CONTROLLED BY PARATHYROID GLAND TISSUE.
BY B. GLANVILL CORNEY, I.S.O., M.R.C.S.

THE endocrine glands may be said to vie with each other in the singularity of their functions; the consequences of their removal, or their impairment through injury or disease, are no less remarkable in the case of the parathyroid bodies than in that of the thyroid or suprarenals. Physiologists allege, for instance, that the function of the parathyroids is of an antitoxic character, and that in bringing about the destruction of guanidini in the blood they exercise a controlling influence over muscular metabolism and maintain muscular tone.¹ If this be true, and guanidine is a poison, it seems natural that the heart should participate in the general steadying effect of these bodies on the muscular system; and the striking relief afforded by their administration in the case of a patient with paroxysmal tachycardia (whom I will call A) recorded by Dr. Clement Dukes² becomes explainable on that basis.

The case of another patient (whom I will call B) is now before me, in which violent paroxysms of tachycardia bade fair to terminate the patient's life within a very few weeks, or even at any moment. B is a male, with a history of 30 years' residence in a (non-malarial) tropical country, and, moreover, is the subject of non-syphilitic arterio-sclerosis of long standing, with a blood pressure of 215; A, a female, with no such complications, has a structurally sound heart, and her case has been quoted as one of "a veritable cardiac epilepsy."

History.—B, a retired surgeon, had his first attack of paroxysmal tachycardia seven years ago, followed by others at intervals of from one to two months, the paroxysms lasting from a few minutes to nearly an hour. Two and a half years ago they became longer and more frequent, with pulse-rate of 160 per minute, and marked polyuria during the first hour and a quarter of the attack. Urine had been of low specific gravity for at least 12 years, habitually

1008 to 1010, and free from albumin or glucose. After each paroxysm came a corresponding diminution and condensation of renal excretion for a day or two, even in cold or damp weather. Any unusual muscular effort, especially if sudden, precipitated a paroxysm; but paroxysms also occurred without being traceable to any directly inciting cause. In 1919–20–21 they often lasted from 8 to 14 hours. Apart from this liability B's general health prior to 1921 gave him nothing to complain of beyond some loss of energy, insomnia at times, and a decline in his walking powers commensurate with his age (now 70).

No drug or dietetic treatment seemed to exercise any restraining influence over the attacks. By the end of October, 1921, B was having a very bad time, which continued, with one free interval of nine days at Christmas, until the middle of February, 1922. In the 87 days immediately preceding the 12th of that month he had 34 paroxysms (an average of about three a week) of a few minutes' to 17 and once 20½ hours' duration; the pulse-rate during the attack was always either 152 or 160. The attacks were completely disabling for the time being and very distressing. Dorsal decubitus, with a slight inclination towards the right, was the only tolerable position. The pulse between attacks was irregular and intermittent, usually 84 in frequency, being the short, hard, sudden, sharp beat of arterio-sclerosis. Frequent heart flutters occurred, accompanied by intermission of the pulse, and lasting a few seconds at a time. About this period B noticed an increased liability to cramp in the muscles of the calf and abdomen when stretching in bed; and some fibrillary twitchings in the eyelids and superficial muscles of the thigh and trunk at times.

In January, 1922, the patient was not confined to bed, except during the paroxysms, but he could not rise from a chair, walk from one room to the next, or turn over in bed without an immediate and alarming acceleration of the pulse; any slight muscular effort (raising a window sash, or gently ascending a few stairs) induced a paroxysm of several hours' duration; the act of relieving the bowels provoked a paroxysm on three or more occasions. He was afraid to go out of the house alone, and had to cancel all his engagements.

Parathyroid Treatment.—In these circumstances B decided, with the assent of a medical friend, to give parathyroid gland a trial; and on Feb. 14th he began to take a tablet containing gr. $\frac{1}{16}$ of the dried substance (Parke, Davis and Co.) three times daily by the mouth when the stomach was empty (as far as practicable), and has continued this measure, sometimes taking only one or two tablets in the day, ever since. On the 15th and 16th the pulse was very irregular and intermittent, and a subdued paroxysm lasting four minutes occurred. From that time forward the result was—as Dr. Clement Dukes said of A's case—dramatic. B described it in his own case as "magical." The paroxysms ceased on the third day after beginning the treatment; in the five months which have since elapsed only three attacks have occurred, for each of which a determining cause was traceable. The general health has greatly improved in all respects; there have been no more cramps or muscular twitchings, and the renal excretion is more normal in volume and specific gravity. B no longer feels the subsidiary heart-

¹ Quart. Jour. Exp. Phys., 1917, x., 325. W. H. Howell: Physiology, 1918, p. 877.

² Brit. Med. Jour., 1921, ii., 987.