the vapour of *oleum sinapis volatile* from the neck of a bottle (say a six-ounce medicine bottle) at the bottom of which there is about half an ounce of the oil. This gives off the vapour all the more readily if the bottle is warmed over a lamp. Two or three sniffs generally result in a good cough and the expulsion (with a little encouragement) of the ejecta from the larynx and trachea into the basin. The sister in charge of the throat room at Brompton Hospital is of the opinion that if this does not excite a cough there is probably no laryngeal tuberculosis. I cannot go quite so far as this, but I am sure that the procedure described has often given us a "positive" result when all previous examinations had proved "negative." In a recent case a history of syphilis led to a diagnosis of that disease as the cause of the hoarseness which was complained of, no bacilli having been found, but a sniff of the oil led to a cough and the expulsion of a small quantity of sputum, in which the bacillus was found in fair abundance. This procedure should, of course, not be preceded by anaesthetization of the nose or larynx.

Another method of value when by any chance the former fails is the *intralaryngeal injection*, by means of a syringe, of a few drops of the lotion we use for cleaning up the larynx—namely, a weak solution of sodium bicarbonate to which is added a little hydrogen peroxide. This is done under the guidance of the laryngeal mirror, and it often works well, but in some cases we are balked by the obstinate closure of an irritable larynx.

In a case of this kind I recently tried another process. The patient either could not or would not cough, and

failed to respond to the methods above described. I therefore applied to his case the principle of *transnasal instillation* used for the introduction of oily solutions into the larynx. The patient was seated with his head thrown back and his mouth wide open. He was instructed to pant in and out through his mouth and not to swallow. While he was doing this I gently syringed about half a drachm of the soda and peroxide solution drop by drop through the nose. A small quantity entered the larynx and started a cough which resulted in the expulsion of a little sputum into the basin. Tubercle bacilli were found, and the diagnosis established. The patient was not upset in the least, and reported himself later as feeling much better.

I have occasionally caught on the *laryngeal mirror* a little patch of sputum sufficient for staining purposes, if the patient coughed spontaneously or did so by instruction during the examination. The examiner does well to wear a muslin veil when practising this last method.

The administration for a few days of *potassium iodide*, if not otherwise contraindicated, is known to facilitate the expulsion of sputum, and my colleague Mr. Ormerod and I have made use of it for this purpose.

These processes have helped in many cases of doubt or difficulty, and I describe them in the expectation that others will find them useful. It need hardly be said, however, that they should not be practised without discrimination. The most appropriate cases are obviously those in which there is doubt as to the diagnosis, and in which, therefore, the disease has not reached an advanced stage.

THE RESISTANCE FACTOR IN DISEASE:

WITH SPECIAL REFERENCE TO SEPTICAEMIA AND ALLIED CONDITIONS.

BY

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In January, 1927, I was called into consultation in a case of facial erysipelas. The patient, who was *in extremis* and who died some six hours later, appeared to have been an otherwise healthy and abstemious man, and his death drew attention to the unsolved problem: What is the true or chief factor in causing the death of a patient who is suffering from a septicæmic condition? The question was put to several medical colleagues, and the answers were interesting, if not very illuminating. One man replied that the cause of death was a profound toxæmia; another, that the centres in the brain were put out of action by bacterial toxins; a third gave it as his view that death was due to cloudy swelling of all the cells of the body; a fourth answered that he did not know, and a fifth suggested that death was due to a lack of resistance on the part of the patient.

Such diversity of opinion indicates the difficulty of the question at issue, and although we may theorize as to the cause of death, the majority of us must admit frankly that we can offer no certain explanation of it. But we are on safe ground when we say that death has resulted from the patient's lack of resistance to the effects of bacterial invasion. This is, of course, a self-evident fact. If his resistance had been all-sufficient, it is unlikely that he would have died.

"The patient seemed to have no resisting power" is a phrase that we have used, often wonderingly, when a previously healthy man has succumbed to some infection—when he has been poleaxed, so to speak, by a disease from which he might reasonably have been expected to recover. Six men may have lobar pneumonia, and one—often seemingly the healthiest prior to the illness—may die, while the remaining five recover. Or death may occur unexpectedly following an apparently successful operation for appendicitis, or from septicaemia following a normal childbirth in which no manual or instrumental interference was required.

What is this lack of resistance, this subtle x factor, which determines a dangerous downhill course or even a fatal issue?

I desire to put forward the hypothesis that our successful resistance to bacterial infection depends largely on the orderly response of certain of our endocrine glands—namely, the sympathicotrophic group comprising the thyroid, suprarenal, and pituitary, and the vagotrophic group comprising the parathyroids and the pancreas.

EXPERIMENTAL CONSIDERATIONS.

Extirpation of the thyro-parathyroids produces a reduction in the phagocytic properties of the leucocytes in a normal man; extirpation in monkeys produces increased liability to infection, and in some animals there is frequently a fatal result.¹ Extirpation of the suprarenals appears to be invariably fatal. With regard to the pituitary Cushing² states: "My own impression has been that total extirpation often provokes a peculiar train of symptoms passing from cachexia to coma and ultimate death." Smith³ says that if the pituitary gland of rats is sucked out by the buccal route an invariable syndrome occurs. In young animals there is cessation of growth, and in adult animals there is, *inter alia*, physical impairment with atrophy of the thyroid, parathyroids, and suprarenal cortex.

Since extirpation of one or other of these glands may produce such serious results, surely it is only sound common sense to assume that a severe infection in the blood stream, attacking each and every gland simultaneously, may produce a result which may easily be fatal. To swamp an endocrine gland with a potent toxin must surely cripple its function, sometimes to an extent which may be equivalent to extirpation.

In profound toxæmias or infections in childhood hæmorrhage may take place into the adrenal medulla, producing profound collapse, associated with convulsions, vomiting, and purpura, a fatal result generally occurring within twenty-four hours.⁴ Elliott and Tucker have shown the lack of chromaffin substances in the adrenals of fatal cases of diphtheria,⁵ and, in certain fulminating cases of cerebrospinal meningitis, hæmorrhage into the suprarenals has been associated with the fatal termination.

Unless the infection is overwhelming in potency, the glands may withstand its effects to some extent and for some time, and may hold out until the invasion has been quelled successfully. But in certain individuals what appears to be a comparatively mild disease may be fatal, and I suggest that the following are among the explanations in many such cases.

(a) The patient may have started life with faultily constructed glands.¹²

(b) He may have a natural tendency to faulty balance in the endocrine glands. Cammidge,¹³ working with Howard, has carried out experiments with mice, and has found that in some apparently healthy animals a low blood sugar is a recessive character, transmitted in accordance with Mendel's theory of heredity, and he suggests that it seems probable from the experiments that the hypoglycaemia is due to hyperfunction of the pancreas relative to the glands of the opposite group—that is, thyroid, suprarenals, and pituitary.

This hereditary dysfunction or lack of balance is interesting, since the percentage of sugar in the blood of the peripheral circulation appears to be similar under like conditions in all mammals. It seems not unreasonable, therefore, to suppose that human beings may exhibit similar hereditary tendencies to imperfect balance of endocrine secretions. Such endocrine imbalance would explain certain cases of diabetes mellitus in young children; it would explain also the tendency to diabetes as a racial or hereditary condition, and would, in my opinion, explain a low grade of resistance to infection in some young children.

(c) Some gross or, more usually, subtle toxin from the apices of the teeth, from the nasal sinuses, from the tonsils, from the digestive tract (as in alcoholism) or from elsewhere may have been at work, undermining the healthy activities of his endocrine glands. Such a condition may be cumulative in old persons and may account for senility, sometimes of premature type, also for the glycosuria which occasionally develops in later life, and for the ease with which elderly people succumb to acute infections.

(d) He may be the victim of unsuspected vitamin deficiency. McCarrison has shown that vitamin defects lead to endocrine changes; the adrenals and pituitary enlarge, while other endocrine glands show more or less atrophy.⁶ Hence there must be a resultant imbalance. Pighini¹⁴ has found that if tadpoles are fed on thyroid gland from pigeons in which beri-beri had been induced by an exclusive diet of polished rice, the tadpoles failed to show the acceleration of growth which is produced by administering the thyroid gland from normal pigeons. This suggestive experiment points to the assumption that a deficiency of vitamin B has a definitely inhibitory effect on the healthy functioning of the thyroid gland. For more than a century before the vitamin content of cod-liver oil was discovered the oil was used on account of its known beneficial effect in raising the patient's resistance, particularly to pulmonary affections.

(e) Emotional causes may lead to a disturbance of balance in the endocrine glands, as witness the well-known fact that cases of exophthalmic goitre showed a great increase during enemy air raids on London. It seems reasonable to think that prolonged anxiety or psychological trauma of any kind may cause or be associated with endocrine imbalance. The gonads are sympathicotrophic, and it is possible that sexual affairs may play a part in producing such an imbalance.

(f) Exposure to cold and damp and other adverse climatic conditions, such as lack of sunlight and fresh air, may cause a lowering of resistance and allow of the advent of certain diseases. In this case it is possible that impulses may pass from the nerve endings near to the surface of the skin and produce imbalance by way of the sympathetic nervous system. It is well known that in hyperthyroidism there is an intolerance to heat and in hypothyroidism to cold.

The position may be summed up in the words of Langdon Brown:⁷ "The balanced life is like a well-planned herbaceous border—to each season its appropriate flowers. For the due ordering of this we are much beholden to the endocrine system." I believe that the simile may be carried a stage further—if a sudden storm sweeps down on the herbaceous border there is an equally sudden strain thrown on the endocrine glands to stand up to the emergency.

Having outlined certain experimental points in connexion with the theory of endocrine resistance, I wish to mention some clinical details; but before doing so it is desirable to touch on physiological and other features associated with the two groups of endocrine glands.

1. The sympathicotrophic group, katabolic in function, comprises the thyroid, suprarenal, and pituitary, and this group responds to invasion of an external foe by activating fight or flight, and of an internal foe (bacterial invasion) by pyrexia and other reactions. It is clearly recognized that these glands, associated with the sympathetic nervous system, are therefore concerned with the activation of the body for defence.⁸

The chief secretory impulses of the thyroid are under

the control of the sympathetic nervous system, but it is innervated also from the superior and inferior laryngeal branches of the vagus. It alone of all the secretions of the body contains iodine.⁹ Thyroid secretion accelerates the heart without augmenting the strength of the beat; increased secretion diminishes sugar tolerance and may raise the basal metabolic rate. It is well known that hyperthyroidism may follow sepsis in tonsils or teeth in adult life. Adrenaline stimulates the force and frequency of the heart-beats, raises the blood pressure, and may excite glycosuria, because it tends to empty the glycogen reservoirs of the liver.

The anterior portion of the pituitary gland is concerned chiefly with the mechanisms of growth, and it is believed to influence the basal metabolic rate. As regards growth, it is interesting to note that when a child has recovered from some acute infection its bodily growth is often thought to have been accelerated, a point which suggests a stimulation of the anterior portion of the pituitary gland.

Dott¹⁵ considers that the hypothalamic region of the brain, which has been called the head ganglion of the sympathetic nervous system, acts in conjunction with the anterior portion of the pituitary gland in its metabolism-regulating function. The internal secretion of the posterior portion of the pituitary gland has the property of slowing the pulse and raising the blood pressure. It appears to have a balancing effect on carbohydrate metabolism, inhibiting, for example, the effect of drugs which tend to change the blood sugar concentration away from normal in either direction.¹⁰ Its overaction leads to glycosuria, and stimulation may play a part also in raising the basal metabolic rate.¹⁶ It appears to be an antidiuretic, and its action on uterine and other muscle is well known.

It will be noticed that little mention has been made of another set of endocrine glands—namely, the gonads, which are linked up with the sympathicotrophic group. Extirpation of the gonads, or their dysfunction as a result of disease or involution, may and does cause certain disturbances in the body processes, but I am unable to produce evidence that they play any important part in resistance to acute infection. For a similar reason no mention is made of the pineal body.

2. The vagotropic glands, anabolic in function, comprise the parathyroids and the pancreas.

Vines's¹⁷ work has emphasized the importance of the parathyroids. He has demonstrated that parathyroid substance is a physiological adjuvant to the normal mechanisms of defence, and he states that there is some evidence that the production of antibodies may depend on the functional integrity of the parathyroid glands. He has shown that the calcium content of the blood is lowered in infective conditions. The parathyroid glands probably act as regulators of calcium metabolism, and, further, the giving of parathyroid gland may raise the polymorphonuclear leucocyte count in infections. The extract has a sedative effect on the nervous tissues; its diminution may lead to irritability, restlessness, and even to tetany.

The pancreas is probably activated, to some extent at least, by the parathyroids, and in the present connexion it is only of moment in relation to its function in helping to raise sugar tolerance.

CLINICAL ASPECTS.

I wish now to consider the clinical aspects of a patient who is in the early stages of an acute infection such as puerperal or other septicaemia, and to correlate the signs and symptoms with special reference to the sympathicotrophic and vagotropic glands.

I desire to stress the point that the first effect of a severe bacterial invasion is a stimulation, obviously protective in character, of the sympathetic nervous system, in relation to which are the sympathicotrophic glands. At the same time there is an inhibition of the vagotropic group, which may be actually paralytic in character—that is, it may result from imperfect response in these glands—or it may be a relative effect, by which I mean that the vigorous response of the sympathicotrophic group may swamp the activities of the vagotropic group, thus causing imbalance in the whole endocrine mechanism.

Side by side with the clinical findings at an early stage of the infection I tabulate the findings in a case which is rapidly approaching a fatal termination.

Clinical Findings:	Early.	Late.
Facial aspect ...	Usually flushed	Pale or cyanosed.
Skin	Hot and dry, but sometimes perspiring	Cold and clammy.
Musculature ...	Movements may be strong and may even require restraint	Limp. Feeble, irregular movements may occur.
Mental state ...	Variable, sometimes clear, often delirious	Usually lethargic.
Temperature ...	High and swinging in type	May fall suddenly before death.
Pulse	Rapid, generally regular	Rapid, frequently irregular.
Respiration ...	Rapid and regular	Rapid and shallow, with irregular sighing before death.
Blood pressure ...	Often slightly above normal for patient's age	Low.
Urinary secretion	Merging from normal to scanty	Scanty.
Basal metabolic rate	Raised	Not determined, owing to seriousness of patient's condition.
Sugar tolerance ...	Tends to be lowered	Blood sugar immediately before death averages 0.15 per cent. (see below).
Blood calcium ...	Lowered	—

As examples of the initial stimulating effect on the thyroid it may be noted that the face is flushed, the pulse and respirations are rapid, the basal metabolic rate is raised, and the sugar tolerance is lowered.

The tendency towards raised blood pressure seems to point to stimulation of the suprarenals, and their increased secretion may also be a factor in producing the rise in blood sugar. It has been said that the pituitary plays a part in connexion with the regulation of the basal metabolic rate, and the increased functioning of the posterior lobe of the pituitary may be one factor in the scanty urinary secretion.

The question of fever in connexion with the endocrine glands calls for some comment. The mechanism of the production of fever is by no means certain. It seems likely that the heat-regulating centre resides in the tuber cinereum, and that this centre is stimulated as a result of bacterial invasion. Fraser¹⁹ states that fever is now believed to be closely associated with hyperthyroidism due to the reaction produced in the thyroid gland by toxins. Langdon Brown¹¹ points out that fever increases the secretory activity of the adrenals and thyroid. This floods the blood with sugar, the increased oxidation of which increases the production of heat, and if the oxidative mechanisms are intact glycosuria is not excited. Miller²⁰ states that increased adrenaline inhibits loss of heat, and it follows that such suprarenal activity would be of great use in the initial stages of an acute infection by raising the internal temperature to a point unfavourable to the growth of micro-organisms in the blood and other tissues. The low blood calcium and low sugar tolerance point to inefficient functioning of the vagotropic glands.

Turning now to the signs and symptoms which accompany the approach of a fatal termination, many features seem to indicate a failure of glandular activity. The drop in blood pressure points to cessation of suprarenal secretion, perhaps with that of thyroid and post-pituitary.

It is a significant fact that limp musculature is an outstanding event, prior to death, in experimental removal of the pituitary gland. There is also, of course, the important fact that, in an acute infection, the muscles themselves have been inundated with the toxins produced by the invading organism. But may not this muscular condition be due in part to failure of the suprarenals to supply a substance acting on the myo-neural junction and maintaining muscle tone in normal circumstances? The irritability and restlessness may be due in part to parathyroid failure as well as to the direct effect of the toxin on the brain cells.

Dr. E. L. Carter, one of the resident staff of the Blackburn Royal Infirmary, is carrying out observations on the blood sugar immediately prior to death. His work is

not yet published, but he allows me to say that his average figure at present is 0.15 per cent. in patients dying from such conditions as lobar pneumonia and septicaemia. This is an isolated figure, as it is impossible, for obvious reasons, to carry out a complete sugar-tolerance test in a dying patient.

The points which have been mentioned are not convincing, but they are suggestive if they are regarded in an unbiased way. I do not for one moment imply that the sole cause of death is to be found in the failure of endocrine glands, but merely that these may play a highly important part, which may have been hitherto insufficiently stressed in relation to treatment. What of the heart itself, and what part does essential heart failure play in determining the lowering of blood pressure and a fatal result?

Pembrey²¹ points out that the heart is very susceptible to a rise of temperature and malnutrition. Perhaps this malnutrition may consist in a lack of normally balanced constituents of the blood, so that the heart, like other muscles, is thereby enfeebled. In this connexion it is interesting to note that adrenaline actually dilates the coronary vessels in spite of its general vaso-constrictive action. If the blood supply to the myocardium is thus increased by dilatation of the coronary arteries this may explain to some extent the prompt action of intracardiac injection of adrenaline when the heart has apparently ceased to beat. If adrenaline is lessening in the blood during the later stages of an acute infection the coronary arteries will fail to allow of the passage of an adequate blood supply. Further, it is possible that the blood which does reach the myocardium is wanting in other constituents which should normally be supplied by the endocrine glands.

Many physicians consider that digitalis is of little use as a heart stimulant during an acute infection such as pneumonia. As Mackenzie²² said: "The factors exciting the heart, such as high temperature, toxins, or the invasion of the heart by specific organisms, exert an influence over the heart which digitalis cannot overcome."

The modern tendency is to combat heart failure in these and other conditions by such substances as post-pituitary or suprarenal extract, and in this way we may be feeling our way towards the right path. Indeed, it is possible that when such a remedy succeeds it may do so, in part at least, by supplying a specific deficiency in the blood. One medical friend of mine to whom I had mentioned this matter treated a moribund case of lobar pneumonia with massive doses of thyroid extract, and to his astonishment the patient recovered.

To make use of an everyday expression, it is no good blaming the carburettor for the faulty running of an engine if the real cause of the trouble is petrol which is inadequate in character.

It is possible that the liver may play a not inconsiderable part in resistance to acute infection, but the means for assessing hepatic function in such a condition are somewhat inadequate. In like manner I know of no explanation, which has been proved hitherto, as to why the phagocytes flag in power under certain conditions, and I suggest that this may be due to deficiency of endocrine gland secretions in the blood stream. Reference to this point has already been made in connexion with the parathyroids. As regards agglutinins, opsonins, precipitins, and bacteriolysins, Dudgeon states:²³ "Unfortunately the presence of all these substances, even in large amounts in the blood, is not necessarily an indication of the patient's resistance to a bacterial invasion."

If, then, it can be accepted that exhaustion and failure of many of the endocrine glands may be an important factor in allowing the patient to be overcome by the invading organism, I submit that it is a rational procedure to supply him, from the beginning of the infection, with gland products to aid the tissues in their fight.

I suggest that the initial stimulation of the thyroid, parathyroids, and suprarenals throws a sudden strain on these glands, and it has been pointed out that the glands may be faulty from congenital causes, from the effect of toxins, or as a result of avitaminosis and other factors. We have little means of estimating the efficiency of the glandular response until the time of testing comes. They

may fail or they may withstand the strain, but it is hardly safe to wait. An acute appendicitis may subside without the surgeon's aid, yet it is usually more prudent to call for that aid. By supplying gland products we attempt to reinforce the natural defensive mechanism and thus to prevent its exhaustion. Just as insulin may be said to rest an overstrained pancreas, so other glandular extracts may co-operate with the natural secretions and allow them to continue their work without depletion of their strength. In the words of Vines:¹⁸

"The aim of treatment should be fundamentally to encourage the re-establishment of normal conditions (that is, a metabolic balance), and since the endocrine glands are the regulators of normal metabolism it is to them that we should look for a means of obtaining the end in view."

Geikie Cobb has called these the glands of destiny, and it is probable that the personality or "make-up" of each member of the human race is influenced profoundly by the predominance of one or more of the endocrine glands. This implies that there is an endocrine balance which is normal for each individual, though differing widely in different individuals; and I believe that health depends upon the maintenance of that normal balance, with slight but constantly occurring deviations according to the needs of the moment.

Departure from health, on the other hand, probably depends on, or is associated with, an unusual deviation from the normal balance. The compass is deflected more or less violently. If, for some of the reasons which have been given—for example, faulty diet or long-continued emotional strain—the endocrine glands become temporarily inefficient, acute disease may result from the advent of a virulent organism in the system, which then responds well or badly, according to the underlying condition of the endocrine glands. If the latter are only slightly under-

mined as regards their efficiency, they will respond adequately to the sudden stimulus of an acute infective condition, in which case recovery is likely after a struggle. An example of this is lobar pneumonia. If the endocrine response is poor, death is probable. Even if the invading organism is one of comparatively mild virulence, death may result if the endocrine glands are inefficient. An example of this would be the occurrence of boils, or a scratch on the skin, followed by septicaemia in a sufferer from diabetes; or the occurrence of puerperal septicaemia in a patient with apical infection of the teeth, presupposing that no interference with the normal course of labour has been necessary.

According to this view subacute and chronic diseases represent varying degrees of virulence in attack, balanced by varying degrees of endocrine response in defence. Having set forth the theory, I am carrying out work at present in connexion with its application to treatment in acute infective conditions.

REFERENCES.

- ¹ Vincent, Swale: *Internal Secretions and the Ductless Glands*, p. 320.
- ² Cushing, Harvey: *British Medical Journal*, July 2nd, 1927, p. 5.
- ³ Smith, F. E.: Disabilities caused by Hypophysectomy and their Repair, *Journ. Amer. Med. Assoc.*, 1927, lxxxviii, 158.
- ⁴ Brown, W. Langdon: *The Endocrines in General Medicine*, p. 22; p. 74; p. 2; p. 26; p. 99; p. 27; p. 55; p. 99.
- ⁵ McClure, J. C.: *Proc. Roy. Soc. Med.*, March 3rd, 1927, p. 1027.
- ⁶ Cammidge, P. J.: *Practitioner*, August, 1927, p. 105.
- ⁷ Pighini, G.: *Biochimica e Terapia Sperimentale*, 1927, July 31st, p. 249.
- ⁸ Dott, N. M.: *Medical Annual*, 1927, p. 374.
- ⁹ Cushing, Harvey: *British Medical Journal*, July 9th, 1927, p. 52.
- ¹⁰ Vines, H. W. C.: *Parathyroid Glands in Relation to Disease*.
- ¹¹ *Ibid.*, p. 89.
- ¹² Fraser, K.: On Iodine. Appendix to Annual Report of the School Medical Officer, Cumberland, 1925.
- ¹³ Miller, E.: *Types of Mind and Body*, Psyche Miniatures, Medical Series, p. 60.
- ¹⁴ Pembrey, M. S.: Price's *Textbook of the Practice of Medicine*, p. 8.
- ¹⁵ Mackenzie, J.: *Diseases of the Heart*, third edition, p. 379.
- ¹⁶ Dudgeon, L. S.: *Bacterial Vaccines and their Position in Therapeutics*, p. 4.

SIMPLE ILEO-CAECAL LYMPHADENITIS.

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THE subject of mesenteric lymphadenitis has from time to time attracted attention, probably, however, not to the extent it merits. Considerable confusion still exists apparently, to judge from the writings that appear now and again, as to the forms in which the condition occurs. The disease may be purely tuberculous, purely non-tuberculous, or a mixture. Tuberculosis of the glands may be diffused, but much more frequently is localized to the ileo-caecal angle; its late effects here, particularly as a cause of obscure chronic right-sided pain, are familiar to all. It is not uncommon to find a septic exacerbation of the process, just as is so often seen in connexion with cervical adenitis.

It is desired here to call attention to the existence of a simple adenitis, which may be either an acute inflammation or a chronic hyperplasia. Occasionally a chronic hyperplasia is found throughout the mesentery of the small bowel—usually in younger children affected by some definite chronic enteritis; much commoner, however, is the condition here described, which is localized to the ileo-caecal lymphatics.

CLINICAL FEATURES.

The disease is seen chiefly between the ages of 5 and 15, and is more frequent in girls. Acute attacks may or may not have been preceded by other acute attacks or by chronic abdominal symptoms—for example, dyspepsia. A large proportion of patients conform to type: the child is a "finicky" feeder, is listless, easily tired, sallow; is liable to acidosis; the musculature is hypotonic. There is sometimes antecedent bowel irregularity, and acute attacks may be preceded by a definite catarrhal condition. It is notable also that a considerable proportion show cervical gland hyperplasia in addition; frequently there has been tonsillar trouble, as if the "stopping power" of the (alleged) first line of lymphatic defences of the mucosae were in general deficient.

Acute Attacks.

The pain is rapid in onset and nearly always right-sided, but sometimes is epigastric or diffuse at the beginning. Vomiting and nausea are relatively infrequent; the appetite may be retained throughout, and the tongue remains moist and only slightly furred. Fever is early and often much higher than in appendicitis, not uncommonly reaching even 102° to 103° F. The bowels may be loose or constipated; the breath often smells of acetone. True abdominal rigidity is not found, though the right side may not move freely; deep tenderness is present over a band about an inch wide under the outer and middle part of the rectus muscle, above McBurney's point. Swollen glands are commonly palpable with a conscious patient, practically always under anaesthesia. Pain is often elicited by either active or passive movement of the hip.

The disease is as rapid in recovery as in onset: two or three days usually sees it end, while the pain itself may last only a few hours. It will be obvious that while there is a superficial resemblance to appendicitis, the two conditions are sharply cut clinically.

Chronic Cases.

The patient comes under attention for repeated attacks, usually very short, of abdominal pain; there may be dyspepsia, and the case usually agrees with the type already described. The pain may be right-sided, umbilical, or epigastric; there may sometimes be persistent aching, worse on movement, on the right side. In a great number of cases the lumpy glands can be felt—the patient has a thin lax parietes; only slight tenderness is present.

PATHOLOGY.

The glandular condition is localized to the ileo-colic chain, and affects the ileo-caecal group in particular. In some cases glands were enlarged up to and in contact with the duodenum, and the lowest were commonly adherent in the ileo-caecal angle; nodes are frequent on the anterior and posterior aspect of the ileo-caecal junction, and in the meso-appendix. In the acute cases the swelling is considerable, the retroperitoneal tissue is oedematous and abnormally vascular, and there may be a small amount of clear colourless fluid in the peritoneal cavity. In the chronic cases the glands are firm, nearly always discrete,