severe cases, for others a more satisfactory method than either of those mentioned was suggested to me by a case in which the crusts were confined to the left nostril. In this girl, aged 14, the right nostril was occluded by a cone-like deviation of the quadrangular cartilage. The left nostril was extremely wide, at least an inch between septum and antral wall below the anterior end of the middle turbinal; turbinal atrophy was marked. The cone was resected through the right nostril with the idea of replacing it with its apex inverted towards the left nostril. Unfortunately the cone broke, but the pieces were pushed under the muco-perichondrium of the left side of the septum. The improvement that resulted was probably due to the freeing of the right nostril, as the left is still too wide, and it has been agreed to do the operation now to be described—transplantation of rib cartilage.

Flakes of cartilage are, with a wide, sharp gouge, taken from the seventh or eighth costal cartilage, and after incision and elevation of the muco-perichondrium of one side of the septum over the greater part of its extent and similar elevation on the inner antral walls between middle and lower turbinates, these flakes are introduced by suitable forceps—the swan-billed septal punch is excellent—into the pockets thus created, which are packed full. The incisions are closed by careful packing with bismuth gauze, which is left for four or five days.

Attention to the following points helps to guard against risks of septic infection, an important consideration in such cases with a closed wound.

Local anaesthesia should, if possible, be used. An intranasal douche with normal saline should be given before the anaesthesia till no trace of pus can be seen. The nose and face must be cleansed with spirit, and the vestibule painted with iodine tincture. The costal wound is closed with an anchor dressing before the nasal operation is begun. If local anaesthesia is used, then, before the costal operation is begun, the nose is anaesthetized with cocaine and an injection, at the vestibule, of eucaine or novocain solution. The pieces of cartilage as removed are laid on

sterile gauze and not touched except with instruments. They may be an inch or more long but neither broad nor thick; for to secure primary union and retention of the grafts the nasal wounds are best not more than 1 cm. long, placed vertically and just posterior to the skin margin, where they can be wiped with iodine.

I have done this operation in three patients, aged 15, 18, and 60 respectively—once under chloroform, twice under local anaesthesia—with primary union and retention of all the grafts in each case. All the patients expressed themselves pleased with the results in respect of comfort and diminution of discharge; but I am not satisfied, as the cavities are still too wide, though the result is better than could be got by any other method known to me. It is surprising how much cartilage can be packed into the pockets; the cubic capacity of the nasal airway is relatively great. To avoid necrosis of the septum the perichondrium can be safely elevated from one side only at one operation; this leaves the other nostril too wide. This defect will be met in future by storing the excess of costal cartilage in a subcutaneous pocket, whence it can be easily taken a month later for transplantation on to the second side of the septum.

Where only one nostril need be narrowed the operation would probably be better done from the other, narrower, nostril; a bevelled incision, longer than that indicated and carried right through septal cartilage but stopping short of perichondrium of the wider nostril, would allow complete elevation of the latter, thorough packing with transplants, and complete closure of the septal window owing to its bevelled edges and elasticity.

Any treatment of atrophic rhinitis, the ciliary brush being gone, can be only palliative. All that is claimed for the method here suggested is that in well developed cases it is more efficient than paraffin injection and less formidable than Halle's operation. The cases are too few and recent to judge results. The method is published that others who have more opportunity than I now have may try it and report.

ACUTE OTITIS MEDIA WITH JUGULAR BULB THROMBOSIS.

BY

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THE following case presents some very uncommon features, especially the absence of any of the usual signs of mastoid disease. The notes given explain the main features.

C. G., a girl aged 10, on February 11th, 1923, complained of sore throat, vomiting, and some right earache, beginning suddenly on the previous day. I saw her on the following day; she then had flushed face, dry lips, very furred tongue, and slight tonsillitis; both tympanic membranes were pink but not bulging. There was a little pain in the right ear, and on deep pressure in the postaural sulcus some tenderness, such as an acute myringitis might produce, but no tenderness over the mastoid process, no redness nor swelling there. Constipation was present, the vomiting had ceased. Her appearance was suggestive of scarlet fever. Temperature 102°, pulse 126, respiration 24.

On February 13th, the right ear was discharging. There was epistaxis in the morning and some vomiting; constipation continued. The child looked and felt better. There were no mastoid signs; the tenderness in the post-aural sulcus had disappeared. Temperature 99°.

Left myringitis occurred on February 15th, with rise of temperature to 102.6°. There was discharge from the ear on February 16th; on this day the vomiting ceased.

On February 18th, vomiting began again, with some diarrhoea, general abdominal discomfort, and tenderness, but no distension.

Both ears were discharging; there was occasional slight right earache; no mastoid signs. The tongue and lips were very dirty and dry. The child was not well although the temperature was down to 99°. The case appeared to be possibly abdominal, and enteric was considered.

On February 21st, the temperature, after remaining down one day, rose again, and the child had a rigor in the morning. Still there were no signs of mastoid abscess; but after consultation, in the absence of any other reasonable possibilities, I decided to operate on the right mastoid, as there had once or twice been earache on that side. With the patient under ether, I performed the Schwartze operation. The mastoid cells and bone were absolutely normal; the antrum contained a very little mucoid material. I therefore exposed the ascending part of the lateral sinus; it was normal in colour and contained fluid blood; the wound was packed and left open. Lumbar puncture gave perfectly clear fluid under slightly raised tension: 15 c.cm. were withdrawn. Pathological report: Slight increase of cells and globulin, no organisms, culture sterile.

On February 24th the temperature remained high and another rigor occurred. Ether was therefore again given, and I exposed the internal jugular vein; it was perfectly normal. The lateral sinus, however, was filled with recent clot to a distance of two inches behind the genu. This was curetted out, as also the jugular bulb, and free bleeding was obtained from both ends. The sinus was packed off, and the jugular vein tied and divided. A small perisinus abscess was found superficial to the sinus, close to the bulb, and was drained. The bone surface was lightly rubbed with "bipp" and the wound packed. The pathological report stated that both clot and abscess contents grew streptococcus. Later that day the patient's condition was good.

Although subsequently the temperature rose again and I feared that the mischief was going to extend, it fell later by lysis, and convalescence was quite uneventful. Hearing was good in both ears. The child was discharged from the infirmary ward on April 3rd.

The course of events is on retrospect fairly clear: the jugular bulb was probably infected through the floor of the middle ear, the disease sparing the mastoid antrum and process. The result was a case very obscure right down to the discovery of the perisinous abscess, which did not exceed a pea in volume. I was not free from anxiety for some days after the second operation. The case well illustrates the necessity for operating sometimes on very slight local indications in the absence of other explanation of a severe infection.

For permission to publish these notes my thanks are due to Mr. J. P. I. Hart, F.R.C.S., who has recorded a somewhat similar case (*Bristol Med.-Chir. Journ.*, 143, September, 1921, p. 78).

ADRENALINE AS A POTENTIAL FACTOR IN HYPERTHYROIDISM.

BY

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In a former article (*BRITISH MEDICAL JOURNAL*, March 31st, 1923) I considered the relationship of the putrefactive group of intestinal organisms to the production of thyroxin. My present purpose is to consider the activity of the same group among the intestinal flora as a potential causative factor in regulating the supply of adrenaline. Berthelot isolated from the intestine a bacillus (*B. phenologenus*) which converts a solution of tyrosin into phenol. This represents a considerable advance in our knowledge of the intestinal flora, for although it had been known for many years that there existed in the intestine bacteria capable of converting the aromatic products of digestion into phenol, no specific function had been attributed to any specific and isolated species among these organisms. This observation suggests a concept which may be found to account for the production of adrenaline either from tyrosin, or else (more probably) from some hitherto unidentified aromatic body closely allied to tyrosin, and, like tyrosin, a product of digestion. Can we in the light of our present knowledge conceive of adrenaline as being produced from tyrosin?

The Amine Producers.

These organisms have the power of removing COO from the COOH group of an amino-acid, converting it into an amine containing one carbon group less than the amino-acid from which it was produced. Apply this to tyrosin. If we limit the function of the amine producers to effecting the change just mentioned, tyrosin becomes, not a body of the adrenaline group, but tyramin, a lower member of the series.

Adrenaline is represented by the formula—

that is, it differs from tyrosin in containing an extra carbon group. If we still regard it as being a derivative of tyrosin, we must attribute an additional function to the amine producers—that of elevating the series by the addition of another carbon group; or else we must postulate a similar synthetic process as being carried out by some other agency, such as the cells of the adrenal glands. Now, as far as the amine producers are concerned, such a view of the question presupposes a type of activity which is, generally speaking, exceptional rather than characteristic among the metabolic processes brought about by micro-organisms. Their function is mainly katabolic; they split up complex into simpler bodies, the building up of complex from simpler bodies being relegated to various types of living body cells. If, then, we are to regard adrenaline as being produced from tyrosin, we

must assume the elaborative activity of some body cell (for example, the cells of the adrenal medulla) to be a factor in its production. On the other hand, if we are to regard adrenaline as being more directly a product of bacterial activity in the intestine, we must postulate the existence of some precursor other than tyrosin. We must seek for its source in some unidentified amino-acid which is, like tyrosin, a product of digestion, and structurally allied to tyrosin (so closely allied to tyrosin as to escape detection by the analytical processes hitherto employed), but which is higher placed in the series than tyrosin. Such a substance will be represented by the corresponding amino-butyric acid, with the formula—

or, better still,

Both the above could be direct precursors of adrenaline, needing only the intervention of the amine producers to effect the change; but as regards the catechol derivative it is important to note that Ebstein and Müller have demonstrated the presence of catechol sulphate in the urine. Such a concept of the origin of adrenaline traces it to a common source with the other endocrine secretion, thyroxin. As explained in my former article, thyroxin is regarded as being derived from tryptophane. Let us consider what happens if this common source (that is, the products of digestion) is interfered with. Such an interference arises when the putrefactive organisms disappear from the intestine. This state of affairs makes itself manifest biochemically, by the diminution or disappearance of indican and all other organic sulphates from the urine, and, clinically, by the development of the familiar syndrome of hyperthyroidism. But what is significant from the point of view of the present investigation is that hyperthyroidism implies not merely changes attributable to thyroxin, but equally important changes attributable to adrenaline, a state of affairs which we should expect if adrenaline and thyroxin are traceable to a common source—the end products of digestion. The absence of all the organic sulphates shows, in fact, that tryptophane and all the aromatic amino-acids are regulated by bacterial action. We should expect, therefore, a part of the syndrome of hyperthyroidism to be really hyperadrenalism. How far is this the case?

It enunciates no new theory to state that hyperactivity of the thyroid gland is accompanied by a corresponding activity of the adrenals. Hitherto this has been explained on the ground of an interaction between the thyroid and the adrenals, the so-called balancing of endocrine activities. From the foregoing paragraphs, however, the phenomenon is more rationally explained, not by some recondite interaction, but by a simple increase in the source of their particular secretions. To emphasize this I would quote the following pregnant paragraph from Professor Swale Vincent's *Internal Secretion and the Ductless Glands*:

"It is doubtful whether a condition strongly resembling Graves's disease has ever been produced by the administration of thyroid substance to animals. But there can be little doubt that definite toxic effects can be produced by thyroid feeding. These are loss of body weight, gastro-enteritis, and diarrhoea, rather than tachycardia, nervousness, and exophthalmos."

A further important question to be considered in relation to hyperthyroidism is that of the increase in the basal metabolic rate, which is now so generally recognized to be an outstanding feature of the condition. There are two main factors affecting this increase. In the first place, it is due to the greater amount of amino-acids rendered available for absorption by the disappearance, or lessened activity, of the putrefactive group of intestinal organisms. In the ordinary course of events a part of the amino-acids would be converted into phenol, indol, and skatol, and would be excreted in the urine as aromatic sulphates. By being