affect the main point of my thesis, which is intended to show that the functional element probably enters more largely into the etiology of anosmia than most authorities are prepared to admit.

Whether Reuter be correct in his distinction between anosmia due to neuritis and the respiratory form, I am unable to say. It is possible that careful employment of the olfactometer may eventually aid us to distinguish the purely functional form, only it must be remembered that the apparatus can only be of use where smell is defective but not quite absent. In the cases I have brought forward, facts have been adduced which seem to me to prove that loss of smell in the presence of nasal obstruction is by no means always due to this cause. If the obstruction be very marked, operative measures will be indicated on general principles. The difficulty in arriving at a decision is pronounced in those cases in which the middle turbinates are somewhat enlarged, and may give rise to the symptom. It might be thought, as indeed I did think until recently, that where the sense of smell returned after measures calculated to diminish the enlargement, we have sufficient evidences to justify an expectation of great benefit to be derived from operative procedure. The cases I have detailed—more particularly the first—however, show that this is not so. At one time it seemed to me that, given a case of anosmia with retention of the power of appreciating the flavours of food, and an obstructive condition detected by anterior rhinoscopy which might interfere with the entrance of odorous particles, we were justified in concluding that the removal of the obstruction would benefit, or even restore, the sense of smell. It appeared probable that the preservation of the power of tasting in the above sense indicated a healthy condition of the olfactory nerve. An analysis of my cases, however, negatives this view.

I am not aware that physiology has contributed anything towards the solution of the question. Of course, we are all familiar with the fact that an expiratory current is required for the appreciation of flavours; but it must be borne in mind that the class of cases which present difficulty are those in which expiration, and indeed respiration, as a whole are not interfered with. It would be tempting to theorize as to the possibility of there being different sets of fibres or nerve endings concerned in smell proper and taste in so far as it is dependent upon the olfactory nerve, but definite proof seems to me impossible to obtain at present. I have spoken of the condition of the olfactory nerve present in these cases as functional for want of a better term. It will, however, at once occur to the reader that in its obstinacy and in the uncertainty which attends its prognosis, the defect in the patients I have referred to differs from many other affections which we are accustomed to classify thus. I have therefore written this paper not only with a view to calling attention to a form of anosmia which seems to me to have been insufficiently studied, but also in the hope that others—whether rhinologists or neurologists—may be induced to throw more light upon a matter which, I must confess, seems to me at present somewhat obscure.

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CONGENITAL SYPHILITIC OBSTRUCTION OF THE COMMON BILE DUCT.

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This case appears to be worth recording because of its rarity, and also on account of its relation to the more usual form of congenital obliteration of the bile ducts.

A female child, aged 3 weeks, was admitted to the Victoria Hospital for Children, under my care, on January 9th, 1906, for jaundice, melaena, and wasting, and died twelve hours later.

Family History.—The father, a retired soldier, lost his voice in 1904. The mother had a bad throat two years ago, and was treated for three months; a year ago she had a child born dead at the seventh month; no other miscarriage; she presents slight scarring with a serpiginous outline about the fauces and soft palate; she says she is subject to jaundice.

History.—This child was born with instrumental assistance at full term, and weighed 7 lb. at birth; artificial respiration was necessary for half an hour after birth. It was not

jaundiced at birth, and did not become so till seven days later. The motions were green, and since the onset of jaundice were offensive; for the last week of life blood was passed by the bowel. The child was breast-fed, and vomited curds for the last week of life. "Snuffles" were noticed during the last two days of life only.

When admitted to the hospital the infant was emaciated, much jaundiced, collapsed, with ulceration of the mouth and a dry tongue, pulse 76, irregular; respirations 40, temperature too low to be registered, except on one occasion when it was 95.4° F. The abdomen was retracted, the liver came two and a half fingerbreadths below the costal margin, and felt hard. No tumour could be felt. The spleen was just palpable. The urine contained bile and blood, but it might have been contaminated with faecal matter. During the twelve hours that the infant was in the hospital the bowels acted six times, the motions being green, offensive, and containing curd and blood. The treatment consisted in stimulation by brandy and heat applied in the form of hot bottles and baths. For these notes I am indebted to Mr. G. E. Friend, House-Physician.

The autopsy was performed thirty-five hours after death by Dr. A. J. Jex-Blake, who kindly preserved the abdominal viscera intact, with the exception of the intestines, for me to dissect. The skin was jaundiced. The thoracic organs were natural, except for bile-staining of the aorta. The liver (4 oz.) was bile-stained. The surface was smooth, not particularly firm, and did not convey the impression of cirrhosis, though in one or two places there were fibrous bands surrounding the

Fig. 1.—Transverse section of obstructed common bile duct, the obstruction being due to syphilitic granulation tissue.

superficial veins. There was no perihepatitis and no adhesions. On section there was an increased amount of fibrous tissue in the portal canals, but the liver did not appear specially firm or cirrhotic. On section of the transverse fissure the lumen of the bile ducts was visible, and was stained yellow; there was some dilatation of the ducts in this position, and the walls were thickened. The gall-bladder showed no pericholecystic adhesions, but its outer surface was white, as if slightly thickened; it contained bile-stained mucus with a white, flaky precipitate, but no calculi. There was some excess of fibrous tissue round the cystic duct, and on transverse section its lumen showed up as a brown spot. The common hepatic and common bile ducts were much thickened by pericholangitic fibrosis; a probe passed freely down from the hilum of the liver towards the duodenum until a point about half an inch from the biliary papilla where it was arrested: the distal part of the common bile duct formed a thick white cord about the size of an adult's vas deferens, and ran down to the biliary papilla without any definite adhesions in the neighbourhood. On transverse section the thickened common bile duct resembled the cross-section of the stem of a clay pipe, there being a minute round hole in the centre with a thick wide rim. On opening the duodenum the orifice of Santorini's duct was visible. The biliary papilla was very prominent and swollen; pressure on the gall-bladder did not drive any bile out of the papilla. Prolonged pressure on the papilla led to the exit of some whitish opaque fluid. There was some bile-staining of the duodenum. The small intestine was natural but there was some colitis and there was haemorrhage into the mucous membrane of the sigmoid flexure. The pancreas was enlarged, firm, and white; on section of the head of the pancreas there was some interstitial fat necrosis; no dilatation of Wirsung's duct. The spleen weighed 2 oz.; a splenunculus was present. Kidneys, 1½ oz. each, were bile-stained, but otherwise normal.

Microscopic Examination.—The thickened lower end of the common bile duct shows in transverse section (Fig. 1) the marked thickening described above. This is due to granulation tissue situated mainly inside the muscular coats, but in places passing through into the outer fibrous coat, and surrounding the mucous glands in that situation. The granulation tissue is composed of round and spindle-shaped cells; there are no giant cells and no caseation. The lumen of the duct shows a little epithelial debris.

The liver shows pericellular cirrhosis and in addition some increased fibrosis around the portal spaces, and here and there plugs of bile-stained mucus in the bile capillaries. The plugs are no doubt due to the biliary obstruction. In short, the liver shows the changes characteristic of congenital syphilis with the effects of biliary obstruction. After hardening in the usual manner sections were stained for the *Treponema pallidum*, but with a negative result.

The pancreas presents a high grade of interstitial fibrosis, the lobules of the gland being invaded and broken up and the acini destroyed or widely separated by connective tissue in various stages of development—round mononuclear cells resembling lymphocytes, plasma cells, spindle cells, and fibrous tissue. Some of the arteries show periarteritis but not endarteritis. The islands of Langerhans are well preserved and prominent, as can be seen in the photomicrograph (Fig. 2) most kindly shown by Dr. Cammidge. The ducts of the gland are not dilated. The interstitial pancreatitis is so advanced that the normal appearance of the gland is quite obscured. It is important to note that this diffuse interacinous pancreatitis is quite different from the interlobular pancreatitis described by Emanuel¹ in the usual form of congenital obliteration of

Fig. 2—Section of pancreas showing interacinous pancreatitis due to congenital syphilis. The islands of Langerhans are prominent. (Photomicrograph by Dr. P. J. Cammidge.)

the bile ducts. For, as shown by John Thomson,² congenital obliteration of the bile ducts (that is, the form presenting mixed portal and monolobular hepatic cirrhosis) is not connected with syphilis.

REMARKS.

There are two possible views as to the nature of this case, first that it is syphilitic, secondly that it is an early stage of congenital obliteration of the bile ducts. In favour of the syphilitic origin of the lesion are the following points: The definite history of syphilis and the syphilitic character of the associated morbid changes on the liver and in the pancreas. I have hesitated to publish this case because the *Treponema pallidum* was not found in the hardened sections; but this failure is probably to be attributed to the tissues having been hardened in the ordinary way. Against the view that the granulomatous change in the common bile duct is an early stage of congenital obliteration of the bile ducts is the absence of the hepatic changes (a mixed monolobular and multilobular cirrhosis) met with in that disease; further, as already pointed out, the form of pancreatitis present in this case is quite different from that figured by Emanuel in congenital obliteration of the bile ducts. I believe, therefore, that this is a case of congenital syphilitic obstructive cholangitis. This condition has been very rarely recognized. The only case like it that I know of is that reported by Beck,³ though possibly some of the reported cases of stricture of the common bile duct in early life may be a later result of this process.

This case supports the view that several different conditions may give rise to obstruction or obliteration of the larger bile ducts on the newborn. Thus, the ducts may

be narrowed as the result of peritoneal adhesions set up by fetal peritonitis; the ducts may be obstructed by syphilitic inflammation, as in this instance; or, as in the majority of cases, there may be a mixed form of hepatic cirrhosis, which is followed by a descending and obliterative cholangitis, the disease usually called congenital obliteration of the bile ducts. Further, it may be pointed out that the recognition of congenital syphilitic obstruction of the common bile duct offers an explanation of recovery under mercurial treatment of cases which appear to belong to the well-recognized group of congenital obliteration of the bile ducts. Poynton⁴ has recorded a case in point, but interpreted the obstruction as due to peculiarly tenacious bile.

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A MODIFICATION OF LEISHMAN'S METHOD OF ESTIMATING THE OPSONIC INDEX.

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LEISHMAN'S original method of estimating the opsonic index is so much simpler and more rapid than Wright's that it seemed desirable to experiment with it in the direction of greater accuracy. Several estimations were made from blood mixed direct with a citrated emulsion of tubercle bacilli, and the results showed marked discrepancies with estimations made by Wright's method, a normal index working out, for instance, to 2.0 or to 0.5, and seldom exactly to 1.0. After trials in various directions it appeared that the inconstant factor vitiating results was the differing number of leucocytes in the respective bloods. Counts of the leucocytes, estimations of the index by Wright's and by Leishman's method modified were then made, and it was found that the figures, expressing the index by Leishman's method and the number of leucocytes, varied inversely, the emulsion being constant.

By a simple formula, based on the above proposition, several apparent indices were corrected, and were then found to agree closely with those estimated by Wright's method; but still discrepancies occurred, and for the following reason. It is usually convenient to obtain blood by the pressure of an elastic or other band; if the same drop of blood thus obtained is used both for the index and leucocyte count, good results are produced. But this needs agility, and in practice would require an assistant. If different drops of blood are used, the haemocytometer and incubation pipettes may contain differing numbers of leucocytes; it is inconvenient to get blood without pressure as a deep puncture has then to be made, possibly daily. Finally, a simple plan was hit upon; after mixing blood direct from the finger—under any pressure, with the bacillary emulsion, the number of leucocytes in the mixture itself was counted, either before or after incubation, each batch being done in the same way. For this the red blood corpuscle pipette serves, and the process is more rapid if there is one for each blood; but any pipette and any dilution desired may be used by drawing up successive volumes of the mixture and diluent as recommended by Wright.¹

After using a drop or two to make a film the mixture is kept in its capillary tube till required for the leucocyte count. The total number of leucocytes need not be estimated, the figure expressing the average for the fields sufficing. A fallacy exists that the phagocytes are not in constant proportion to the total number of leucocytes.

The actual modification of Leishman's method is very simple. The capillary pipette is that in ordinary use; it is convenient to make a bend in it 2 or 3 in. from its distal end. A rubber tube, as in the haemocytometer pipette, is easier to manage than a rubber teat, or a Wright's throttled pipette may be used. A mark is made above the bend, giving a long column of blood and emulsion, making for greater accuracy of dilution and providing enough of the mixture for its two purposes—namely, for making a film and for leucocyte count.

The emulsion consists of (tubercle) bacilli suspended in citrated normal saline—say, 0.75 per cent. each of