free of carbohydrate, in the form of sugar, from a previously combined state.

If, now, we give a comprehensive glance at the position of our knowledge of the conditions standing at the foundation of diabetes, it may be said that the effect of modern research is to carry us away from the view that there is a functional transit of the carbohydrate matter of our food as sugar through the general circulation to the tissues. Under such a view we are faced with insuperable difficulties in relation to the information we possess with regard to both health and diabetes, whilst under the view contended for in this communication everything satisfactorily fits in, and a rational working basis for the treatment of diabetes is supplied.

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THE FACE AND PUPIL IN ALCOHOLIC NEURITIS.

By SIR T. LAUDER BRUNTON, M.D., F.R.S.,
Physician to St. Bartholomew's Hospital, London.

ALCOHOLIC neuritis is so common, and the literature about it so extensive, that the signs I am about to mention have probably already been described before, yet I have not come across a description of them, and should be glad to learn if such a description has appeared.

The loss of knee-jerk and extreme hyperæsthesia of the skin in advanced cases are of course known to everyone, but before these occur, and while the knee-jerk may be only sluggish or may even be unaltered, a peculiar expression of the face is sometimes noticeable. The face becomes mask-like and expressionless, the lips appear to move apart from the cheeks, but what is sometimes still more extraordinary the lips themselves may seem very mobile. The eyebrows and eyes may move in accordance with the lips, but a fixed and unexpressed band stretches across the nose and cheeks between the eyes and lips, the skin upon the cheeks remaining motionless and unwrinkled, while the lips, eyebrows, and forehead may be moving freely. I was much struck by this condition on seeing it one morning in two patients successively. In the first patient I had no difficulty in obtaining a history of alcoholism. As the second one entered the room I was struck by the similarity between the appearance of his face and that of his predecessor, but I could only elicit a history of excessive smoking both from himself and his wife; they both assured me that while the patient smoked excessively he was exceedingly temperate in regard to alcohol. A few days afterwards, however, I had a letter from the patient's partner saying, "I suppose So-and-so told you that he had been taking a great deal more spirit than was good for him?"

Another point is the condition of the pupil reflex, which is just the converse of the Argyll-Robertson phenomenon. In a number of cases of alcoholic neuritis I have noticed that the reflex of the pupil to light is rapid and extensive, whereas the contraction of the pupil on accommodation to a near object is slight and sluggish or entirely wanting. Indeed in one or two cases I have observed a dilatation instead of contraction on accommodation.

EXPLOSION OF ACETYLENE GAS.—An accident that might have been attended by much more serious consequences occurred a few days ago at Chapel-en-le-Frith, Derbyshire. One of the hotels in the town had been fitted up with acetylene gas. A representative of Husson's Acetylene Syndicate happening to call at the hotel he was asked to inspect the generator, which was supposed to be leaking. He must have known that acetylene is more explosive than coal gas. If so familiarity had bred contempt. On lighting a match a loud explosion occurred, during which the representative of the syndicate was injured in the face and hands. At first it was thought his eyesight was destroyed. Fortunately none of the injuries were of such a nature as to prevent the traveller leaving the district two days afterwards. It should be remembered that while acetylene is an excellent illuminant it is a highly explosive gas.

ON THE CAUSATION OF THE CONGENITAL STRIDOR OF INFANTS.*

(With Illustrations on Special Plate.)

BY
JOHN THOMSON, M.D., F.R.C.P. Edin., and A. LOGAN TURNER, M.D., F.R.C.S. Edin.,
Physician to the Royal Hospital for Sick Children, Edinburgh. Surgeon for Diseases of the Ear and Throat, Deaconess Hospital, Edinburgh.

THE clinical features of a typical and simple case of congenital stridor may be summarised as follows:

The infant, who appears normal in other respects, is noticed shortly after birth to have noisy breathing. The noise consists of a croaking sound accompanying inspiration, which rises to a high-pitched crow when a longer or more vigorous breath is taken; expiration may be accompanied by a short croak when the stridor is loud, but at other times it is noiseless. Even in the most severe cases there are occasional brief intervals during which there is no sound audible; but, with this exception, the stridor goes on constantly while the child is awake, and sometimes even when he is asleep. Any emotional excitement or any physical cause of deeper breathing, such as exposure to colder air or to the exertion of sucking, is apt to intensify the sound. The child's power of crying and coughing is unaffected. The breathing, although noisy, is not accompanied by the slightest distress, and there is no cyanosis. There is, however, always marked inspiratory indrawing of the thoracic and abdominal walls, except in the mildest cases.

The stridor increases in loudness during the first few months, and, after remaining about the same for a few more months, gradually lessens and disappears during the course of the second year. Long after it ceases to occur habitually, however, it is occasionally set up by emotional causes.

Considerable difference of opinion has been expressed as to the causation of these cases. The symptoms have been attributed to posticus paralysis (Robertson) or to abductor spasm, due to adenoids or to some other source of irritation (E. Smith, Lövi). Some again have blamed an enlarged thymus (Avellis), and others a congenital malformation of the upper aperture of the larynx (Lees, Sutherland and Lack, Variot, Refslund). Attention has also been drawn to the soft collapsible character of the laryngeal structures in the infant (Goodhart, etc.), and one of us (Thomson) has already suggested the view which we confirm and further elaborate in this paper. This view is that the primary cause of the obstruction is probably an ill-co-ordinated spasmodic action of the muscles of respiration—a choreiform respiratory spasm—analogue to stammering—as distinguished from a laryngeal spasm.

The site of the obstruction which causes the noise has variously been thought to be in the pharynx, at the upper aperture of the larynx, at the cords, and in the trachea at the level of the thymus. In the presence of so much difference of opinion it seemed to us worth while to make a further attempt to ascertain what facts can be established with regard to these cases, and to what extent the various theories of causation are in keeping with them.

As the condition is entirely confined to young infants, it will be well to begin by referring to the peculiarities of the larynx and of the breathing at this time of life.

THE LARYNX IN THE YOUNG INFANT.

Our observations upon the anatomy of the larynx in infants and young children have been based upon the examination of fifty specimens removed from subjects in whom no obstruction of the upper respiratory passages existed. For the majority of these we are indebted to the kindness of Dr. Theodore Shennan, pathologist to the Royal Hospital for Sick Children and to the staff of the Royal Maternity Hospital. The larynges of the stillborn infants were in many cases examined *in situ* by means of a dissection which exposed them from behind; the remainder, taken from children up to the tenth year of life, were examined after removal from the body by the ordinary *post-mortem* method. Our attention was directed mainly to the upper aperture of the larynx. This

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opening may be defined anatomically as the space bounded in front by the epiglottis, behind by the summits of the arytenoid cartilages with the intervening fold of mucous membrane, and laterally by the two ary-epiglottic ligaments. When the term "upper aperture" is employed in this paper, the space enclosed by these structures is signified.

In the infant the structures bounding this opening are much softer and more collapsible than they are in the adult; further, the aperture is relatively narrower from side to side, owing to the folding backwards and inwards of the epiglottis with the consequent drawing together of the ary-epiglottic folds. This infantile type, which has been described by Merkel, Luschka, and other anatomists is represented in Figs. 1, 2, and 3 (special plate), reproduced from the most typical of our own preparations. In them, the epiglottis is seen to be distinctly gutter-shaped on its posterior aspect, while the soft and yielding ary-epiglottic ligaments are approximated so as to narrow the upper aperture. As the child grows, a change takes place in the form of these parts; the epiglottis becomes more expanded, the ary-epiglottic folds separated, and the aperture in this way broadened. Fig. 4 (special plate) represents the larynx of a child aged $3\frac{1}{2}$ years; in it the opening is wider; while in Fig. 5 (special plate) is depicted the larynx of a boy, 9 years of age, in which the infantile type has almost entirely disappeared. Fig. 6 (special plate) shows the adult type of the same region, which demonstrates the more expanded leaf-like form of the epiglottis, the greater separation of the ary-epiglottic folds, and the consequently increased breadth of the intervening aperture.

While varieties are met with in different infantile larynges at the same period of life, all of them possess in a more or less marked degree the characteristics above described. These peculiarities combine to make the upper aperture of the larynx liable to constant changes in form under the action of influences which have no corresponding effect on the adult organ. The most important influence of this kind is the sudden drawing of air into the larynx. In the larynges of newborn children and very young infants, the effect of this can be easily demonstrated after death by the following simple experiment. A bent metal tube, 18 inches in length, is introduced into the trachea, and, in order to keep the chink of the glottis open, a small piece of rubber tubing is at the same time placed between the vocal cords. forcible inspirations are then made through the tube, and it is found that with each a very striking alteration in the form of the upper aperture of the larynx occurs. The exact change, however, varies in different cases; we have noticed three main types:

1. In some cases the sides of the opening were sucked together so as to form a narrow mesial slit.
2. In others the epiglottis was drawn backwards and downwards so as to act as a lid.
3. In others, again, the arytenoid cartilages met in the middle line, while the margins of the anterior portion of the aperture remained unaffected.

Figs. 7 and 8 (special plate) show the upper aperture of the larynx of a stillborn infant before and during the carrying out of such an experiment. In Fig. 7 the open aperture is seen before the experiment, while in Fig. 8 the epiglottis is markedly folded backwards, and the ary-epiglottic ligaments are in contact mesially.

In two of our experiments the upper aperture assumed a form almost exactly similar to that figured by Refslund (Fig. 10, special plate), but unfortunately we have not succeeded in securing a photograph of this result.

When this experiment is repeated several times with the same larynx, we find that its upper aperture retains to a very considerable degree the deformity which the suction has produced. Thus, when the upper aperture collapses on suction so as to form a vertical slit, the ary-epiglottic folds will be found after a few experiments to lie permanently nearer one another.

The objection will doubtless be raised by some that such experiments conducted upon the cadaver cannot be regarded as entirely trustworthy, because the exact conditions which exist during life are not present. We would point out, however, that the ary-epiglottic folds, which are mainly concerned in the production of the deformity, contain only a few muscular fibres, and are thin, lax folds of mucous membrane which are readily acted upon by such a mechanical force as

the sucking in of air between them. Further, it must be sufficiently well-known to all who have attempted to examine during life the larynx of healthy infants and young children that the ary-epiglottic folds tend to become approximated when the child forcibly inspires during the examination; that is to say that these structures are capable of assuming in the living body the same position that they can be made to take up in the cadaver, and as a result of a similar cause.

RESPIRATION IN THE YOUNG INFANT.

The respiration under normal conditions in very young children is peculiar in being irregular in rhythm and in force, as if the co-ordination involved in its movements were not as yet under full control. There often occur, therefore, relatively sudden respiratory efforts. When a young baby is excited, it is no uncommon thing for it to develop a temporary crowing noise with inspiration, although it has none at other times. A similar, but louder and more persistent crowing, which indeed exactly resembles that in a case of infantile stridor, is not infrequently observed in young infants while they are beginning to come out of chloroform narcosis; and a very loud crowing is a common accompaniment of operations on the genital organs, owing to the sudden forcible inspiration which the stimulation of the nerves of these parts is apt to occasion. Along with crowing, there is always a degree of indrawing of the lower chest wall proportionate to the loudness of the sound.

We must now turn to consider the conformation of the larynx and the character of the respiration in children who are the subjects of congenital stridor.

THE LARYNX IN STRIDOR CASES.

A peculiarity of form of the upper aperture of the larynx, which differs in degree, but is always essentially the same in nature, has been described and figured by various writers (Figs. 9 and 10). We believe that some degree of this is always present in cases of congenital stridor. It may be said to consist in an exaggeration of the normal infantile type, and it is practically the same as that which can be induced *post-mortem* in the larynges of many newborn children by suction through a tube. In two of the published cases the upper aperture was found to be reduced to a mere slit. In both of these the patient died from respiratory disease, and presumably had severe dyspnoea immediately prior to death.

Fig. 9 (special plate) shows the larynx in the fatal case of congenital stridor described by Dr. Lees, and we are indebted to him for kind permission to reproduce it here. The patient, an infant of a year old, had suffered from stridor since birth, and died of laryngeal diphtheria.

Refslund's patient (Fig. 10) was an infant aged $2\frac{1}{2}$ months who had been the subject of persistent crowing from birth. Death took place from pneumonia, and an examination of the larynx showed the upper laryngeal aperture reduced to a mesial slit. Variot's patient was a boy aged 1 year and 10 months who began to suffer from inspiratory stridor shortly after birth. He died from scarlet fever, and the upper aperture of the larynx showed a lesser degree of the same deformity.

The peculiar conformation may continue long after the breathing ceases to be noisy. We have examined five children between 7 and 9 years of age whom we had previously seen with congenital stridor, and in all of them the deformity still persisted to some extent.

PECULIARITY OF RESPIRATION IN STRIDOR CASES.

The respiration in these cases differs from that in normal babies in being accompanied by inspiratory croaking and crowing sounds, which vary in loudness according to the vigour of the inspiration, and by the occurrence of a corresponding amount of thoracic indrawing. The stridor is nearly constant in well-marked cases, but never entirely so, there always being occasional free intervals. When the inspiratory crow has lasted some time, and is loud, there is apt to be expiratory croaking also. We have thus in these cases the habitual presence of a condition of stridulous breathing, the same as occurs temporarily during recovery from chloroform narcosis.

CONCLUSIONS.

Our most important conclusions may be stated as follows:

1. That the primary element in the causation of this condi-

Fig. 2.—Child, aged 3 months.

Fig. 1.—Child, aged 10 weeks.

Fig. 3.—Child, aged 3 months

Fig. 4.—Child, aged 3½ years

Fig. 6.—Adult larynx.

Fig. 5.—Boy aged 9 years.

(7) Before experiment.

(8) During experiment.

Fig. 9.—Lee's case of infantile stridor, child, aged 1 year, death from diphtheria.

Figs. 7 and 8.—Larynx of newborn child. Fig. 10.—Refslund's case of infantile stridor, child aged 2½ months, death from pneumonia.

tion is a disturbance of the co-ordination of the respiratory movements, probably due to some developmental backwardness of the cortical structures which control them.

2. That the change of form found is merely an exaggeration of the normal infantile type, and is mainly if not entirely the result of a constantly recurring sucking-in of the upper aperture of the soft larynx, which is induced by the ill co-ordinated and spasmodic nature of the breathing. That it is, in fact, an acquired deformity strictly analogous to pigeon breast.

3. That there is no proof that any congenital malformation of the upper laryngeal aperture exists in these cases.

4. That the supposition of a congenital deformity is not essential to account for the symptoms, inasmuch as normal babies crow in a very similar manner when they are coming out of chloroform.

5. That the sounds are not produced in the pharynx. This is proved by the high-pitched phonic character of the crow, and also by the fact that the stridor persists, not only when the nostrils are closed, but equally when the mouth is occluded by the nipple, when the tongue is depressed by a spatula, and during yawning. That they are not produced in the trachea by compression exerted by a large thymus or enlarged lymphatic glands, we conclude on two grounds. First, because in none of the fatal cases of congenital stridor recorded have we seen enlargement of the thymus or lymphatic glands noted; and, secondly, because in two cases which we have examined, in which compression of the trachea by enlarged bronchial glands was found after death, the symptoms were of a very different type. In these cases the stridor was mainly expiratory, the larynx did not move up and down with respiration, as it does in cases of intra-laryngeal obstruction, and there was much greater respiratory distress. The stridor is probably produced partly in the larynx proper, and partly at the abnormally approximated ary-epiglottic folds.

6. That the neurosis causing the symptoms has not in our experience seemed to depend on the presence of adenoid growths or other obvious causes of reflex irritation.

It may be well that we should add a few words to define as clearly as possible how our position differs from that held by Drs. Sutherland and Lack in their paper already referred to, which seems to us by far the most important contribution made to the subject. We are cordially at one with them when they say "that the affection depends on a valvular action of the upper aperture of the larynx, a falling inwards of its lateral walls during inspiration," and that this is due partly to "the flaccidity of the parts in infants." We join issue with them altogether, however, when they ascribe primary etiological importance to the peculiar conformation of the upper aperture found in these cases, and regard it as a congenital malformation. We consider it an acquired deformity, and hold that it, as well as the stridor, are the result of the ill co-ordinated stammering breathing.

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AN INFANT POISONED BY ITS MOTHER'S MEDICINE.—As reported in the *Essex Weekly News*, an inquest was held recently at Southminster, on the infant child of a labourer who had died, presumably as the result of opium poisoning, on the day after its birth. The child had screamed very much during the early hours of its life, and was given a few drops of its mother's medicine by a friendly neighbour. The medicine contained opium, and it was probable that an enormous dose had been given for so young a child. From the evidence it appeared that it was no uncommon thing for nurses and midwives in that district to administer to infants doses of the mixtures prescribed for their mothers. The jury added a rider to their verdict calling attention to the practice.

CASES OF ACROMEGALY AND INFANTILE MYXŒDEMA OCCURRING RESPECTIVELY IN FATHER AND DAUGHTER.*

(With Illustrations on Special Plate.)

BY

F. M. POPE,

and ASTLEY V. CLARKE,

M.B., M.R.C.P.,

M.D.,

Senior Physician to the Leicester Infirmary; Assistant Physician to the Leicester Infirmary.

As far as we can ascertain no case of direct hereditary connection between acromegaly and myxœdema has yet been reported. The following cases are at present under observation at the Leicester Infirmary:—

CASE I.—G. D., aged 38, married and with family, was sent to one of us by Dr. Hewlings.

Family History.—Good. No history of constitutional disease. The eldest child, a girl, is Case II.

Personal History.—Generally healthy; has had a hydatid cyst (?) opened in the abdomen 9 years ago, and recently had an abscess in the right lumbar region; no evidence of hydatid present. He sought advice in December, 1890, for weakness, rheumatic pains, stiffness of the hands and inability to clench the fists, dating from four years earlier and getting worse. He complained of some headache; he had had no sickness and there was no history of any chronic chest trouble.

Condition on Examination.—The hands are broad, massive, with rather lengthened fingers, the fists cannot be clenched, the palms and flexor surfaces of the fingers are thickly padded by tough skin and increased subcutaneous tissue. The sausage-shape of the fingers makes them appear relatively shorter than they are. The ends of the fingers are bulbous. He is subject to "dead fingers." The wrists are enlarged and the lower parts of the radius and ulna are massive. These conditions are symmetrical. The feet are large. He has noticed that he has had to get larger boots lately. The face (Fig. 1, Special Plate) is somewhat elongated; the superciliary ridges are prominent, and the lower jaw slightly elongated. This is most noticeable when comparison is made with a photograph taken sixteen years ago. The alveolar processes not thickened. There was no marked increase in size of any other bones; no joints were enlarged. The tongue is larger than normal. The thyroid gland apparently normal. The chest shows no abnormal physical signs either cardiac or pulmonary. The attitude in the erect position shows shoulders rounded (a slight cervico-dorsal curve). As to the special senses there is bitemporal hemianopsia. The field of vision more contracted in left eye. Hearing, smell, and taste are normal. The skin presents no warts; he sweats profusely on slightest exertion. The hair is thin on the head, and he is slightly bald; the beard is lanky. Under treatment with thyroid extract the patient has considerably improved in certain details: the pains are less, the fingers can be flexed firmly into the palms, the general appearance is more healthy, the weakness less prominent.

The diagnosis of acromegaly rests on the presence of the following phenomena: The occurrence of a localised symmetrical hyperplasia of both bones and soft parts of the forearms, wrists, hands, and feet; the bitemporal hemianopsia, the lassitude and the rheumatic pains of long duration, the absence of vomiting, optic neuritis, and giddiness; the gradually progressive nature of the first set of these symptoms.

The club-shaped extremities of the fingers—well shown in the photograph—are rather against acromegaly, but then there are no signs or symptoms of any chronic chest trouble, which makes a diagnosis of hypertrophic osteo-arthritis untenable.

CASE II.—A. D., aged 20, a girl, the eldest child of Case I, has been under observation of one of us since October, 1896.

Personal History.—The only illness has been pertussis. She was apparently healthy until 5 years of age, when the parents noticed that she did not seem to be developing properly either physically or mentally. She came for treatment at the age of 16. She was then 3 feet 4½ inches high; weight 43 lbs. Her intelligence was about equal to a child of 4 or 5 years old. She had a heavy expression and malar flush, the lips were thick, the nose flat, and the skin dry. The hands were spade-like, and there were well marked pads of fat above the clavicles. The usual solid œdema was present about the body. The thyroid gland could not be felt. The temperature was often 96.4°; the pulse about 60. The photograph (Fig. 4, Special Plate) shows the patient shortly after the treatment began. Thyroid treatment was carried out for some time, but any improvement which was at first noticed was not permanent, perhaps because the parents would not persevere with the treatment.

It will be seen that Case II is probably one of infantile myxœdema; it can hardly be termed cretinism, as the symptoms did not come on till the patient was nearly 5 years old. The clinical symptoms do not in any way correspond with those of acromegaly.

That there may be an hereditary predisposition to either of these diseases is shown by the fact mentioned by many observers that cases of each have occurred in more than one member of the same family. Instances of this nature have naturally—owing to the greater rarity of the disease—been less common with regard to acromegaly than to myxœdema.

* Read before the Midland Branch of the British Medical Association.