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.

REFERENCES.

- ¹ *Journal of Physiology*, xxiv, p. 486. ² Baisch, *Zett. f. physiol. Chem.*, xx, 1895, p. 249. ³ *Skandinavisk Archiv für Physiologie*, ix, 1899, p. 336. ⁴ *Lancet*, vol. i, 1900.

THE FACE AND PUPIL IN ALCOHOLIC NEURITIS.

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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
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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

* Read in the Section of Diseases of Children at the annual meeting of the British Medical Association, Ipswich, July-August, 1900.