as to formulate this into a so-called "law," "a positive reaction denotes active syphilis." As a general statement, however, this cannot be correct. Tabes and general paralysis can scarcely be described as active syphilis, yet, as we have seen, the serum in these diseases gives a positive reaction in 70 to 90 per cent. of cases. In the spinal fluid it may be held even to be injurious rather than beneficial, since it has a definite affinity for so important a constituent of normal nervous tissue as lecithin. Further, its occurrence in the serum and spinal fluid of general paralysis and tabes, while it adds another argument to the clinical evidence connecting these diseases with syphilis, in no sense proves that connexion incontestably.

We have pleasure in thanking Dr. Mott for the interest he has taken in our work, and the assistance he has rendered us throughout. We also acknowledge with pleasure the kind assistance given us in obtaining material by Dr. Robert Jones, the Medical Superintendent, and Dr. Hughes, Medical Officer of the London County Asylum at Claybury; Dr. Bond, Medical Superintendent of Long Grove Asylum; Dr. Ingram and other medical officers of the London infirmaries; Mr. Gibbs, Surgeon at the London Lock Hospital; and Dr. Nepean Longridge, of Queen Charlotte's Lying-in Hospital for Women.

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PNEUMONIA MIGRANS INVOLVING THE WHOLE OF BOTH LUNGS, FOLLOWED BY EMPYEMA: RECOVERY.

By JAMES BANNERMAN, M.B., C.M.EDIN.,
STANHOPE, CO. DURHAM.

THE rarity of acute pneumonia lasting for over five weeks and involving the whole lung area justifies a record of the following case, in which the disease had affected *seriatim* the three lobes on the right side, commencing at the top, and the two lobes on the left side, commencing with the lower, the last involvement, that of the left upper lobe, being preceded by a localized pleuritic effusion. Such a protracted and extensive pneumonia must be a rare occurrence, and it was a matter of surprise to me that even the last phase of the disease ran quite an ordinary course.

On January 4th, 1909, I was asked to see a girl, M. D. F., aged 3½ years, and found her to have quite indefinite symptoms, without any rise of temperature. I was suspicious of pneumonia, as her father was suffering from it in the same house. The next day she had a high temperature (103° F.), but still I could not be sure of pneumonia. On January 6th I detected pneumonia, with all its typical signs, in the right upper lobe. The case progressed in an ordinary way till January 11th, when I observed an extension of the inflammation to the right middle lobe. Two days later much anxiety was caused by signs of heart failure, but happily the heart reacted well to strychnine. The following day (January 14th) there was well-marked pneumonic involvement of the lower lobe on the right side. I noted on January 15th a clearing up of the upper lobe; by January 18th it appeared to be normal. The temperature had kept up till now, varying from 101° to 105° F., but on the evening of January 19th it dropped to normal. The following day it rose to 104.6° F., with clear evidence of pneumonia in the left lower lobe. This fresh attack ran an ordinary course, the temperature only falling on January 29th.

On January 27th I had noted that the right base appeared practically normal. The next spread of the disease was on January 30th, when there were indefinite signs of trouble over the left upper lobe with an increase of temperature. The breath sounds were tubular and distant and percussion was dull; there was no increase of vocal fremitus or resonance. Over the left lower lobe there was undoubtedly no fluid, the breath sounds being tubular and loud with vocal fremitus and

resonance both increased. But I could not satisfy myself as to whether the condition in the left upper lobe was pleuritic effusion, or pneumonia, or both. This was cleared up on February 3rd by exploration through the third interspace in the anterior axillary region when I drew off some perfectly clear yellowish-green fluid. Exploration over the left lower lobe by sterilized needles at different places gave negative results. The child's breathing being very embarrassed, I aspirated the localized effusion over the left upper lobe, withdrawing 12 oz. of clear fluid. This gave much relief and the left upper lobe after the aspiration was quite resonant on percussion. But the temperature never came below 100.4° F., and on the morning of February 4th there was conclusive evidence of pneumonia in the left upper lobe, the resonance being impaired and the breath sounds assuming a high-pitched tubular type. This last attack of pneumonia also ran an ordinary course till February 10th, when the temperature fell to 98° F. after having been up almost uninterruptedly for thirty-six days.

With the child shockingly reduced by the long illness, I vainly hoped that some degree of acquired immunity would succeed in cutting short the latter attacks. The only comfort was that there was no other lobe to become involved. For a week the temperature remained down, but the child was in a pitiful state of weakness. Sickness supervened on February 17th, with a rise of temperature, and the child began to go downhill rapidly. The left base had never regained its resonance, but now it became absolutely dull, with distant bronchial breath sounds. On February 22nd the child's condition was extremely grave, but as the temperature kept down I waited and watched for a day or two longer. An increase of dyspnoea on February 25th induced me to explore the left base, when I found pus. The following day, with the kind assistance of my neighbour, Dr. Menzies of Wolsingham, I resected 1½ in. of the eighth rib in the posterior axillary line under local anaesthesia. I avoided a general anaesthetic on account of the weak and septic condition of the child, and I was more than satisfied with the efficacy of the local anaesthetic. The child lay still without making a cry when I excised the rib. A large quantity of pus was evacuated and two parallel rubber drainage tubes inserted. The case progressed favourably until a week after operation, when defective drainage caused some rise of temperature. I then irrigated the cavity daily for a few days with weak perchloride solution, followed by saline solution, and this brought away considerable masses of stinking pus clot. The after-progress was uninterrupted. Six weeks after operation she was healed up, strong, and running about with an expanded left lung.

I regret that nothing was attempted in the way of vaccine treatment, but in a scattered country practice such a refinement is hardly practicable.

THE CAUSES OF OBSTRUCTION PRODUCING CYANOSIS DURING THE NASAL ADMINISTRATION OF NITROUS OXIDE.

By F. TREWBY, M.R.C.S.,

ASSISTANT ANAESTHETIST TO ST. BARTHOLOMEW'S HOSPITAL; HOUSE-ANAESTHETIST TO THE ROYAL DENTAL HOSPITAL.

AFTER careful observation and examination of some thousands of gas administrations I have come to the conclusion that cyanosis in ordinary cases is merely mechanical, being mainly due to the size and shape of the palate and tongue.

The tongue varies from the large flabby to the compact muscular variety. While at rest, when the tip of the tongue is on the posterior surface of the lower incisors, the base of the tongue in the former variety may be ½ in. above the level of the grinding surface of the second molars, in the latter it may be well below the junction of the tooth and gum margin. The importance of the soft palate is its variable length; it may overlap the base of the tongue by an inch, or it may be separated by an aperture of an inch.

I. When these structures give rise to the greatest obstruction to free breathing, the patient has a large flabby tongue and a long, overlapping soft palate; the distance between the two is slight, or they may be in contact. Even to start with, the patient may find some difficulty during expiration, as the oro-pharyngeal aperture (by this I mean the aperture bounded above by the soft palate, at the sides by the pillars of the fauces, below by the base of the tongue) is small. Immediately the soft palate loses its tone, being placed at a sloping angle with the base of the tongue, it acts as an inspiratory valve, and is pressed down on the base of the tongue during expiration. This state of affairs may easily be brought about much earlier if gas is given under much pressure through the nose-piece, as the gas passing back into the

naso-pharynx forces the soft palate against the base of the tongue, thus making expiration through the mouth impossible. At this stage, with the soft palate and base of the tongue in contact, if there is no valve at the upper part of the bag, nasal "to and fro" breathing into the bag commences, the patient gradually becoming cyanosed. If there is a valve to prevent this rebreathing, then the only possibility of respiration is by forcing the nose-piece slightly off the face, thus allowing the expired air to escape around it; this obstruction to respiration quickly causes cyanosis. It is quite easy to keep this class of patient unconscious with the ordinary nose-piece, but only at the expense of cyanosis.

II. In a patient who has a small, compact tongue, a short soft palate, the two being widely separated and therefore giving rise to practically no obstruction to respiration, one obtains an ordinary gas anaesthesia; but remove the mouth cover, and continued nasal administration will have very little effect. Even if nitrous oxide is passed rapidly through the nose, the rate of diffusion is so rapid that the patient obtains a mixture of gas and air, which is insufficient to produce anaesthesia; these patients can be controlled by placing an expiratory valve in the fauces.

Between these two extremes are varieties of tongues and soft palate which during the administration give rise to more or less obstruction to respiration, with a corresponding amount of cyanosis or gas anaesthesia.

The most important point dependent on the construction of the fauces is the occurrence of nasal "to and fro" breathing when the soft palate comes in contact with the base of the tongue; this may be brought about by other factors. In cases in which the soft palate does not reach the base of the tongue the slight cyanosis of the gas administration causes the tongue to swell up sufficiently to fill up the space between them. If the oro-pharyngeal aperture is of the largest type, the tongue during an ordinary administration will not swell up sufficiently to reach the edge of the soft palate, and nasal "to and fro" breathing does not occur; but in the average nasal administration during the continuation one observes that the patient is kept cyanosed and the tongue swells up and lessens the oro-pharyngeal aperture; in this way the oral air-way is occluded, and the patient must obtain pure gas by the nose.

It has been stated that "after normal consciousness is lost, a period follows in which psychical disturbances ensue; at this stage there is frequently a change from 'naso-oral' breathing to 'to and fro' nasal breathing, which is evidenced by escape of expired gases around the nose-piece. This is so frequent that it has been taken as a useful sign of unconsciousness and approaching anaesthesia; even the firm application of the mouth cover throughout the inhalation had little if any effect in modifying this phase." The following case will explain the reason:

The patient had a large flabby tongue with an overlapping palate; when anaesthetized with the ordinary nose-piece, the mouth cover being placed in position, for three breaths the valve worked; then "to and fro" nasal breathing commenced, and expired gases tended to escape round the nose-piece, and no air was expired through the mouth. Now, according to the above statement, after four breaths this patient should have been unconscious and nearly ready for operation. What had happened was this: The space between the base of the tongue and soft palate being narrow, directly expiration occurred the soft palate was first pushed against the base of the tongue, and then bulged forwards into the mouth, forcing some of the air out of the mouth and through the valve of the mouth cover. When inspiration started, a slight *minus* pressure formed in the mouth, shown by the valve of the mouth cover being slightly drawn in; as neither gas nor air could enter the mouth, the soft palate was kept fixed down on the base of the tongue. I should describe this as "false action" of the mouth cover, giving rise to early nasal "to and fro" breathing, putting the valve of the mouth cover out of action, and completely obstructing breathing through the mouth.

Whether gas is given by the nasal or ordinary method, the fact remains that the oro-pharyngeal aperture is always obliterated, or nearly so, before the naso-pharyngeal aper-

ture is in any way affected, and the breathing gradually tends to become nasal. This can be easily demonstrated; if gas be given by the face-piece method, when one is near the end of the administration, the face-piece is still kept on, and at the same time the nose is compressed, unless the palate is of the short variety, it gives rise to obstruction to expiration.

Besides the condition of the fauces causing obstruction and giving rise to cyanosis, cyanosis in itself gives rise to obstruction of free breathing in the later stages, the swelling and falling back of the tongue giving rise to false stertor; I have noticed that this trouble occurs to a marked degree in patients with receding chins.

Cyanosis causes distension of the large veins of the neck; and, as is well known to every one, a collar or piece of tape, which was quite loose at the beginning is, if the administration is pushed until the patient jactitates, found to be pressing tightly round the neck, the measurement of the neck having increased $\frac{1}{2}$ in. in circumference. This is clearly due to distension of the superficial veins of the neck, as it is not likely that a structure like the cervical fascia can stretch much. In cases of oedema or suppuration beneath the deep cervical fascia, one is struck by the severe dyspnoea that occurs from pressure beneath this structure without any apparent general swelling of the neck.

If the superficial veins external to the cervical fascia be compared with the deep veins of the neck, one realizes what the pressure in these huge veins must be, on the soft tissue, beneath a fixed structure like the cervical fascia.

The parts likely to be affected are the laryngeal opening, which may get pressed back against the wall of the pharynx, and the space just above the larynx, where the tissue is extremely vascular, and it is quite easy to imagine how, with the increased tension within the cervical fascia occurring in asphyxia, the air-way here may be completely obliterated.

It has been stated that "by compressing the nose at the end of an ordinary administration the patient is more likely to stay under for several extra seconds." This is a general experience; we can conclude that the compression causes obstruction of free breathing, if at the end of an administration instead of compressing the nose we place an ordinary nose-piece over the nose, we completely obstruct expiration through the nose, the nasal air-way being at this stage of the administration the chief means the patient has of breathing.

From the fact of the occurrence of nasal "to and fro" breathing during the induction of anaesthesia by nitrous oxide, and that in the later stages of the induction the respiration is almost entirely by the nose, I draw the conclusion that any nose-piece without an expiratory valve on it causes unnecessary obstruction and cyanosis, not only while anaesthesia is being induced, but also during the continued administration.

THE TREATMENT OF SOME CHRONIC INFLAMMATIONS OF THE EYE.

By C. G. RUSS WOOD, F.R.C.S., L.R.C.P.,

SURGEON TO THE SHROPSHIRE EYE, EAR, AND THROAT HOSPITAL, ETC.

SOME three years ago Professor Burnham of Toronto published a small book¹ describing in detail a method of treatment of chronic ocular inflammatory conditions, especially irido-cyclitis and sympathetic ophthalmia. My attention was drawn to this by Mr. Stanford Morton, who advised its use in a case, which we were seeing together, of severe irido-cyclitis, with nearly total loss of the anterior corneal layers.

The "combined" treatment, as it is termed by Professor Burnham, consists in the administration of mercury and potassium iodide, combined with the daily hypodermic injections of pilocarpin, his hypothesis being that the pilocarpin arouses and stimulates the ordinary physiological processes to activity in excess of the normal, so that the tissues are more easily acted upon by the mercury and potassium iodide. It is, of course, in the experience of every one that in certain chronic inflammatory