A similar bacteriological table seems obvious for the broncho-pneumonias separate from the lobar pneumonias. This is especially desired by anybody interested in the streptococcus, the importance of which has been increasingly realized with each succeeding month of the past winter and spring. It would bring out more clearly the true extent of the supposed *relation between broncho-pneumonia and streptococci*. This relation is of the greatest importance. It can be traced only by using thoroughly reliable data. The use of any other would yield results of apparent value but really only misleading. It is felt that our data, while serviceable for the purposes of the bacteriological tables presented, are not, however, sufficiently reliable for the determination of the relationship under discussion, because:

a. Too many *patients* showing diffuse râles followed later by clinical and x-ray signs of consolidation not definitely either lobar or lobular, were not followed up. Looking back we can only guess. We cannot justly say whether there was:

- i. Bronchitis developing broncho-pneumonic patches.
- ii. Bronchitis developing confluent-lobular broncho-pneumonia.
- iii. Bronchitis developing lobar pneumonia.

b. Too many *sputa* were:

- i. Never sent to the laboratory.
- ii. There considered "defective specimens."
- iii. Not examined for any streptococci.
- iv. Not classified as to whether the streptococci were hemolytic or innocent.
- v. Reported under a different classification on two occasions without any evidence in the clinical record of co-operation between the ward and the laboratory in deciding whether this divergence was due to:
 - i. Faulty technic.
 - ii. Reinfection with a different organism.
 - iii. Super-infection with a different organism.

(To be continued.)

Clinical Department.

REPORT OF A CASE OF CONGENITAL ATRESIA OF THE OESOPHAGUS.*

BY JAMES LINCOLN HUNTINGTON, M.D., J. HERBERT YOUNG, M.D., AND N. CHANDLER FOOT, M.D.,
BOSTON.

THE obstetrical history in this case is as follows: The mother is 39 years old, with a negative family and past history; date of last catamenia was January 9, 1918. Previously had had two normal labors, the children are alive and well; one miscarriage, followed by uneventful convalescence. The history of this pregnancy is in no way remarkable, the patient started in labor with rupture of the membranes at 2.20 A.M., on November 8th: under gas and oxygen, followed by primary ether at 10.25 A.M. she was delivered of a female child, weighing eight pounds, eight ounces. The delivery was in every way normal. The child breathed at once, but seemed to be secreting a great deal of mucus, requiring constant attention for the first two hours, turning blue repeatedly and choking. Every time there was respiratory difficulty the evacuation of a drachm or two of thick, tenacious, slightly yellow mucus by the mouth would give temporary relief. Between these attacks the child seemed normal.

When first seen by the surgeon, the infant was six hours old. She was well developed and nourished, rather pale for a new-born baby and slightly cyanotic. Her breathing was noisy, but not labored. The head and fontanelles were normal. The mouth and throat were filled with thick, tenacious mucus. The heart was normal in size and position, heart-sounds of good quality, no murmurs. Although the child had not cried lustily since birth, air apparently entered all parts of both lungs; but auscultation was difficult because of the noise made by the mucus in the throat. The abdomen and extremities were normal. There was no paralysis and no evident malformation. At the first examination nothing unusual was suspected. The child appeared normal, except for the large amount of mucus in the throat.

When seen the next day the amount of mucus in the throat was still considerable. Attacks of choking and cyanosis were frequent, sometimes three or four within one hour, and even with oxygen it was difficult at times to get the child breathing again. She had had normal

* Read before the Boston Obstetrical Society, January 28, 1919.

meconium movements and had passed urine. When given water from a medicine dropper and when put to the breast, she would take a mouthful, choke and stop breathing. When the attempt was made to give a tube feeding, the tube met an obstruction five inches below the gums and no fluid would pass this obstruction. Although the tube caused no discomfort when the attempt was made to pour in fluid, respiration would cease. The diagnosis of complete obstruction of the oesophagus was evident. Surgical treatment was not justifiable because of the poor physical condition of the patient, who died on the fourth day, death being due to exhaustion and an inhalation pneumonia which was revealed only at autopsy.

At autopsy the following facts were noted, the protocol will be given in somewhat abbreviated form. The body is 53 cm. in length, well developed and nourished; rigor mortis present in lower jaw, absent in extremities. Post-mortem lividity marked in dependent portions. There are no external abnormalities, the pupils are equal, 2 m.m. in diameter. Upon making the primary incision from suprasternal notch to mons pubis the subcutaneous fat is found to be well developed, the diaphragm is in its proper relationship to the ribs. The umbilical vein is patent throughout, from the healing umbilicus to the liver, 2 m.m. in diameter. Upon opening the thorax the lungs do not collapse.

The heart shows no abnormalities, excepting that it contains a small amount of fluid blood, indicating asphyxia. The right lung lies free, its surface is everywhere smooth and shining and is mottled purplish red and bright salmon-pink. The upper, middle and lower lobes are, for a great part, consolidated, the consolidation being of a patchy nature. The lower anterior margins of the upper and lower lobes show normal aeration and are of a salmon-pink color. On cut section this organ is found to be filled with areas of consolidation averaging 3-5 m.m. in diameter. Throughout these areas there is very little air content, the fluid content is slightly increased, as is the blood content. The bronchi are everywhere markedly congested and contain a slight amount of mucus. The left lung is very similar, the upper lobe being almost normal in color and consistence, with the exception of a few areas of very dark red hemorrhage, averaging 1-2 m.m. in diameter. The lower lobe is markedly mottled and contains numerous patches of consolidation which are

sparsely scattered throughout its substance. On cut section it is for the greater part normal in the upper lobe, except for the hemorrhagic areas just described; cut section of the lower lobe shows similar changes to those seen in the right lung, but much less marked. Its bronchi are exactly like those of its fellow.

Microscopically, the lungs show edema and hemorrhage into the alveolar spaces, with very marked congestion of the capillaries in the alveolar walls, areas of true broncho-pneumonia in which the exudate is composed chiefly of polymorphonuclear leucocytes and red cells. Some alveoli contain a thick, pink-staining mass somewhat suggestive of milk. Fibrin is present in the exudate in small amounts. Compensatory emphysema is noted in the least affected parts of the lungs. The bronchi are filled with pus and there is considerable sub-pleural hemorrhage.

The spleen, liver, pancreas, adrenals, kidneys and internal genitalia are in no way remarkable.

Alimentary Tract. The oesophagus, beginning at the pharynx, extends downward to a point 3.5 cm. below the glottis, where it terminates abruptly in a rounded, slightly tapering, blind extremity not unlike the thumb of a glove. It is 1.5 cm. in diameter, has thickened walls, 2 m.m. diameter, and contains a good deal of thick, tenacious mucus. From its extremity a small musculo-fibrous cord continues to a lower oesophageal segment 2.5 cm. further down. This cord measures 1 m.m. in thickness. The lower segment of the oesophagus opens out of the trachea, just at its point of bifurcation. It appears to be similar to a normal oesophagus in its general structure and size, being one centimeter in diameter, with its walls of normal thickness and color. It terminates normally in the cardiac end of the stomach. The opening into the trachea is a small, transverse slit, about 5 m.m. in width, which communicates with the posterior surface of the trachea at a point directly behind the bifurcation. There is a suggestion of a funnel-shaped bulging of the posterior tracheal wall for 1.5 cm. above this communication.

Microscopic examination of cross sections of the connecting cord, taken at different levels, show no trace of epithelial tissue; it is made up of longitudinally arranged muscle fibres and intermuscular connective tissue. The muscle is striated.

In the accompanying illustration, Fig. 1 is

the posterior view of the specimen, *a*, the upper segment; *b*, the cord, and *c*, the lower segment. Fig. 2 gives the anterior view of the communication of the lower segment with the trachea.

The stomach and intestines present no abnormalities, the former contains a small amount of thick, bile-stained mucus; the latter are partly collapsed and contain a small amount of meconium and some gas. The cause of death was evidently respiratory embarrassment, due to the anomaly and to the inspiration pneumonia that resulted therefrom.

We come now to a consideration of the history of and the explanations for a condition which is common enough to warrant much more mention than has been accorded it in the average system of medicine or surgery; Albutt, Keene, Osler, Pfaundler, and Schlossmann (La Fetra's translation) all dismiss it with a paragraph or two and even special pathologies, like Kaufmann's, give but brief accounts of this anomaly. Lewis, in Keibel and Mall's *Embryology*¹, devotes much time to atresia of the

oesophagus at birth and puts special emphasis on the most common type, that represented by our case. He outlines the theory of its origin, without going into the subject at length, and gives a valuable bibliography.

A review of three of the authors named in this bibliography will give one a good idea of the work done on this subject and of the various theories to explain the different forms of the anomaly encountered. These three are Forssner², Kreuter³, and Giffhorn⁴. Giffhorn's article is of interest chiefly for its review of the literature in years past; he describes two cases and advances a theory as to the reasons for their existence. Forssner's article takes up the subject of atresiae of the gastro-intestinal tract as a whole, including those of the oesophagus and gives so excellent a summary of Kreuter's paper, that it is unnecessary to refer to it any further.

Summing up the material of these three articles we find that the condition has been known since 1838, when Schoeller described a number of cases of congenital atresiae of the oesophagus, stating that the type represented by our case is the most common. In 1884 MacKenzie described forty-five cases. Kreuter collected no less than one hundred and eleven. In discussing this last series, Forssner ruled out eighteen for various reasons; total absence of oesophagus, double oesophagus, other gross abnormalities present, insufficient data, etc. Of the remaining ninety-three he notes the following facts:

Sixty-four are connected with respiratory tract, sixty-one of them with the trachea, three with the bronchi. Four of those connected with the trachea opened into it in its upper portion, forty-one at the bifurcation. In sixty-one the upper segment ended blindly with a cord connecting it with the lower segment in thirty-three, no cord in five, and no data in twenty-six. There was complete interruption of the oesophagus, with two segments ending blindly, in eleven cases. Of these, six had a connecting cord, five had none. There was membranous closure of the oesophagus in three cases, and a stenosis in fifteen.

Thus we see that the most common type, comprising roughly two-thirds of all congenital atresiae, is one connected with the trachea at its bifurcation, where the lower segment takes its origin; the upper segment ending blindly and

being connected with the lower by a cord of muscular tissue.

There are several theories to explain the anomaly and we shall consider them chronologically. The oldest is that of Klebs and MacKenzie: that too much material was used up in forming the respiratory tract, the oesophagus being interrupted at a point where its substance was, so to speak, appropriated by the respiratory tract. Foerster and Giffhorn took the view that it was to be traced to the period when the oesophagus was still a solid cord and failed to vacuolate and open up properly; a theory based upon comparative embryology and, as has been subsequently shown, quite erroneous, as the human oesophagus is at all times patent, under conditions of normal development, unlike that of some of the lower animals. Kraus combines these ideas, advancing three hypotheses: *a.* That, in the type where the oesophagus is for the greater part obliterated, the respiratory tract has used up the material (Klebs' view). *b.* That those cases with membranous closure "entice one to think of epithelial occlusions." *c.* That the common type is due to pressure exerted by neighboring vessels, with resulting pressure-atrophy, an hypothesis also advanced by Keith, who found anomalies in the branching of the left subclavian artery ("low origin") in three cases where the oesophageal deformity was also present, but which he had to reject later, upon finding several cases where this did not hold true. Kreuter supported Foerster's view.

Forssner's explanation is more logical than any of these, but fails completely to explain the condition. He rejects the theories just cited, as they fail to explain the formation of the fistula which exists in most of the cases. He rejects the epithelial proliferation theory on the grounds that the separation of the respiratory from the alimentary tracts takes place at a period much in advance of that in which the oesophageal epithelium becomes actively proliferative; the two tracts separate before the four millimeter stage is reached, or shortly before, while the proliferative phenomena take place when the embryo is about 19 m.m. in length. That he is correct in this surmise is proved by the presence of a well-formed oesophageal atresia of the common type in an eighteen millimeter embryo in the Harvard collection, which corresponds perfectly with our case, excepting that there is a sacculated dilata-

tion of the middle third of the lower segment. Forssner's explanation is that the fusion of the outgrowing lips of the tracheal groove is incomplete at a point corresponding to the bifurcation of the future trachea and that this interruption persists as a fistula, which stimulates later proliferation of the epithelium above, or below it with resultant occlusion at the end of one or both segments; if either end be closed off, a fistula will persist. The characteristic dilatation of the upper segment, such as seen in our specimen, he thinks results from the swallowing of liquor amnii and that this brings about an axial shortening of the cul-de-sac which may cause the rupture and disappearance of the cord connecting the segments.

The best explanation of all is outlined by Lewis, in Keibel and Mall. When the trachea and oesophagus are becoming separated, at the 4 m.m. stage, two processes or wings of the body-cavity project up on either side of the latter, normally exerting enough pressure to groove its outer surface, these grooves running from below upward and from behind forward. These grooves are readily demonstrated in a reconstructed model of a 4 m.m. embryo. Now, supposing this pressure were increased by an increase in the size of these processes, due to an abnormal amount of fluid within them, or to cellular overgrowth, the grooves might become deep enough to mould the oesophagus into the trachea at that point, cutting off an upper and a lower segment, the former usually ending blindly, the latter being switched into the trachea and communicating with it. That portion of the oesophagus between the two wings of body-cavity would be pressed together and ultimately atrophy, giving rise to the cord. It would be well to bear this in mind in the future and investigate such cords microscopically to determine the presence or absence of epithelium.

To recapitulate: the best theory is that, at an early period, the walls of the foregut grow towards one another, forming two folds. Anterior to these folds is the future trachea, posteriorly the future oesophagus. If, while this is taking place, pressure is exerted at a point near the bifurcation of the respiratory tract by the impinging processes of body-cavity already described, the fusion will take place along new lines at this point, resulting in a shunting-out of the lower two-thirds of the oesophagus from the foregut into the respiratory tract and an obliteration of the lower extremity of the upper

third of the oesophagus, with the formation of an upper blind segment, or cul-de-sac communication with the mouth. It is easy to see how such an anomaly, with numerous mucous glands in the pharynx and upper cul-de-sac actively secreting (and possibly hypertrophied in the cul-de-sac) could cause much mucus to collect in the mouth and upper air passages. It could not be swallowed and if inhaled might excite further secretion in the trachea and bronchi from the resulting irritation, establishing a vicious circle.

In closing we wish to express our thanks to Professor Lewis for his kind assistance in the interpretation of this phenomenon.

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⁵ Keith: See Lewis.

Selected Papers.

CHOREA: ARSENIC AS A REMEDY.*

By GEORGE FOY, M.D., F.R.C.S.I.

"ROUGH NOTES ON REMEDIES," by Dr. William Murray, published in 1896, was one of the most successful books of its year. Its introduction at once produced a favorable impression, and the opening chapter "On arsenic in diabetes, chorea, and asthma," met and satisfied the wants of the great majority of his professional brethren. We have no intention of quoting theories on the etiology or pathology of chorea, either Sydenham's or the so-called *Chorea magna sive Germanorum*. The disease is described under the title Saint Vitus' dance by Sydenham; and we read that Saint Vitus was removed from Sicily when a boy, at the time of the Diocletian persecution of the Christians in the year 303, and suffered martyrdom in Florence. His mortal remains, after many removals, are said to have been interred in the cloister of Koovey in 836.

"At Strasburg many hundred began
 To dance and leap, both wife and man,
 In open market, lane and street,
 By day and night; many did not eat
 Until to an end their madness came;
 Saint Vitus' they did it name."

* Reprinted from the *Medical Press* of January 22, 1919.

Gregory Horstius, the distinguished professor of medicine at Wittenberg, and afterwards a public physician and president of the University at Ulm, informs us that in the opening years of the seventeenth century some women, whom he described as being disordered in their minds and affected with a peculiar kind of restlessness in their bodies, repaired once a year to the Chapel of Saint Vitus, near Ulm, and continued there night and day, leaping and dancing, till they were so exhausted as to drop down almost dead. Thus, continues he, they were restored, and continued well till the following May, when the same propensity returned, and required a similar course of exercise to remove it. From this tradition the convulsive disease to which children are so liable derives its name. This condition of convulsive neurasthenia is, in some cases, difficult to differentiate from Sydenham's chorea. Hirsh reminds us that the dancing mania is still commemorated in the grotesque procession of the "jumping saints" which is held every Whitsuntide at Echternach. These manifestations are evidently symptoms of hysterical neurasthenia in weak-minded individuals, and, as we see from the older writers, were sometimes successfully treated by purgatives and bleeding. A different condition is found in Sydenham's type, even though only in degree. There are, however, some cases intermediate between these two in which the arsenic treatment is peculiarly successful. Of these Mr. Salter, of Poole, put some cases on record one hundred years ago, and successfully treated them in the manner Dr. Murrell so happily, and unconsciously, re-introduced. Eliza H., aged 17, has had chorea Sancti Viti for three months. "The muscles of the arms are most affected with the irregular and involuntary motions, but those of the trunk and lower extremities are considerably under the influence of the disease; indeed, the whole body is frequently thrown into the most grotesque attitudes." She was put on four-drop doses of liquor arsenicalis three times a day, with directions to add one drop to each dose every day. Ten days after she was taking fourteen drops in each dose, "and is apparently getting rapidly well." She is directed not to increase the dose any further. Three months later "there has not been any return of the chorea." Miss P., nine years of age, affected with chorea, September 6, 1817. Fear is supposed to be the exciting cause. On the 24th of the month the symptoms