for one from 2 to 3 years old, and from 1 to 3 grains (0.065 to 0.2 gm.) for older subjects, every two hours, suffice. It should always be given with as much water as the patient will take. *Liquor ammonii acetatis* is another valuable agent, given in doses varying from 5 to 30 drops—also in considerable water—according to age.

The onset of collapse is not only advanced, but may be actually caused by the absence of sodium chloride in, or lowered alkalinity of, the blood, or both of these conditions. *Hypodermoclysis* promptly restores the patient—even when he is approaching the moribund state, in some cases. In the infant, 4 to 6 ounces (120 to 180 gms.) of normal saline solution may be injected slowly under the scapula, and renewed if necessary. Hypodermic injections of *aromatic spirits of ammonia,* 2 drachms (8 gms.), being injected into the arm, repeated as needed, have also been found useful.

Still better than all these measures is to avoid the need of them by using alkaline beverages from the start, thus keeping the blood supplied with its normal salts.* The measures indicated on page 1367 may be utilized, reducing quantities according to age. *Saline enemata, i.e.,* rectal injections of warm saline solution, are also of very great value, especially in young children.

I cannot sufficiently emphasize the importance of not waiting until the disease is far advanced, to resort to the use of alkaline beverages. Their use should begin when the patient is first seen. Quite as important is to supply the patient with fresh air, *i.e.,* air not partially deprived of any of its oxygen, and with all the water he wants to drink.

The use of normal saline solution in this disease has been found very advantageous by J. Madison Taylor[127] when used as ordinary beverage. Lemaire[128] used hypodermoclysis in 11 cases of infantile bronchopneumonia, 6 ounces (180 gm.) being injected under the skin of the abdomen or thigh in children 3 years old and over. Under that age the injections were 2 ounces (60 gm.) three times a day. All the cases recovered. Injections of the aromatic spirits of ammonia were found very effective, though somewhat painful, by H. Morell.[129] He states that the action of the remedy is noticed almost immediately, the face losing its livid color and becoming flushed.

* *Author's conclusion.*
[127] J. Madison Taylor: Medical Record, Jan. 13, 1906.
[128] Lemaire: *Loc. cit.*
[129] H. Morell: N. Y. Med. Jour., Sept. 7, 1895.

CHAPTER XXIX.

THE INTERNAL SECRETIONS IN THEIR RELATIONS TO PATHOGENESIS AND THERAPEUTICS (*Continued*).

THE ADRENAL SYSTEM IN THE CATARRHAL AND NERVOUS DISORDERS OF THE RESPIRATORY TRACT.

The four diseases studied in the present chapter are intended to exemplify the manner in which the adrenal system reacts when exogenous or endogenous irritants assail the mucosa of the respiratory tract. Under Acute Bronchitis I submit the manner in which the tracheo-bronchial mucous membrane becomes the seat of an acute inflammatory process through the operation of a factor whose pathogenic influence has been abundantly confirmed but not explained. Bronchial asthma illustrates a complication which endows the disease with its autonomy as a morbid process, namely, hypersensitiveness of the vagal center in the pituitary body—and the manner in which stricto-dilation (the mode of action of all motor-nerves) provokes muscular contraction—the minute bronchial muscles, in this instance. A kindred disorder, hay-fever, serves to indicate how the same process operates in the nasal mucosa, where the stricto-dilators regulate the flow of blood into sinuses, which thus become engorged with blood, causing the copious secretion, marked obstruction, etc., observed in this disease. This process prevails as well in acute coryza and in other disorders of the upper respiratory tract, which cannot be treated in full in this volume. The fourth disease analyzed, pertussis, exemplifies the manner in which cough is elicited by irritation: a reflex excitation of the vagal center (or trigeminal center, if the nasal field is involved), the violence of which is such in this disease as practically to exhaust the lungs of air, thus necessitating sudden and violent inspiration, the characteristic "whoop."

ACUTE BRONCHITIS.

SYNONYMS.—*Tracheobronchitis; Acute Tracheobronchitis; Acute Bronchial Catarrh.*

Definition.—Acute bronchitis, an acute catarrhal inflammation of the tracheo-bronchial mucosa, is the expression of a local protective process characterized by an increase of auto-antitoxin and phagocytes in the secretions, having for its purpose the removal or destruction of irritants carried to the mucosa either by the air (dust, vapors, etc.), or through the blood (toxic wastes, antitoxins, toxins, etc.).*

Symptoms and Pathology.—Three types of acute bronchitis may be distinguished. The first of these is the *afebrile* form, starting, as a rule, with an acute coryza, soon followed by a feeling of oppression behind the upper part of the sternum, more or less headache, languor, and sometimes muscular pains. The expectoration is at first viscid, then opaque and purulent. There is usually some hoarseness and in some cases aphonia. In normal adults this form is quite benign, but in children it may lead to broncho-pneumonia. In aged or debilitated subjects (especially if scoliotic) the congestion of the bronchial mucosa and the imperfect elimination of the pulmonary secretions may sufficiently embarrass the smaller bronchi to cause asphyxia.

The second is the *febrile* form, in which the phenomena are all much more marked, being those of a true infection, viz., severe headache and malaise, repeated chills followed by fever, which may reach 104° F. (40° C.) in the afternoon or evening. Then follow the thoracic symptoms: dyspnœa and tightness about the chest, retrosternal rawness and pain traceable upward along the trachea to the larynx. Here a sensation of pricking or tickling provokes paroxysms of severe, dry cough, attended with little or no expectoration, and very distressing on that account. Soon, however, the sputa are brought up with more ease, and finally in abundance, being at first viscid, then muco-purulent, and finally purulent. The tongue is coated and there is usually anorexia or dyspepsia. Such an attack, left to itself, usually lasts from ten days to two weeks.

* *Author's definition.*

The third, or *secondary* form, as its name implies, occurs as a complication of other diseases, measles, typhoid fever, variola, influenza, erysipelas, scarlatina, diphtheria, etc. It differs in no way from the second or febrile form as to its symptomatology, the various phenomena enumerated being merged in with those of the primary disease.

The physical signs in all three forms vary with the intensity of the morbid process. In the afebrile form but slight, if any, change from the normal is discernible unless the case be somewhat severe and approximate the febrile form. In the latter, the dyspnœa is attended by a compensative increase of respiratory movements, and bronchial fremitus. On auscultation, the respiratory sounds are exaggerated and differ according to the caliber of the bronchi from which it originates, the larger bronchi producing a grave note resembling snoring, the smaller, a sharp whistling or sibilant note, both during respiration and expiration. As the secretion increases, the râles become moist, as when air is blown through water. These sounds are best heard posteriorly all over the chest and on both sides. In secondary acute bronchitis, the moist or subcrepitant râles begin at the base of the lungs, where dullness on percussion is noticeable, especially in certain areas.

The predominating pathological feature is hyperæmia of the capillaries of the bronchial tubes. In severe cases this may be sufficiently marked to cause tumefication and infiltration of the bronchial mucosa, *i.e.*, a true œdema capable of materially reducing the caliber of the smaller bronchi, and even of causing asphyxia.

This is only partly due, as shown below, to a local inflammatory process caused by a vicarious elimination of pathogenic elements. The local hyperæmia coincides with the dry cough, but this is soon succeeded by increased activity of the mucous glands and the abundant secretion produced soon gives rise to a free expectoration. An abundant local leucocytosis occurring simultaneously to rid the respiratory tract of any detritus, the sputa acquire their muco-purulent character. This stage usually terminates a mild case. In the more severe cases desquamation of the ciliated epithelium takes place, followed by lesions of the deeper structures. In the bronchitis of certain infections, diphtheria, typhoid, etc., the hyperæmia may be followed by the local formation of a false membrane which, according to Cornil and Ranvier, is replete with micrococci.

Etiology and Pathogenesis.—The *exogenous* causes of acute bronchitis are those which, such as dust or irritating vapors,

reach the bronchial tubes and their ramifications with the air inhaled, and are capable of irritating mechanically or chemically their mucous membrane. This form is observed mainly among individuals who are exposed to such irritation in the course of their occupation, millers, knife grinders, etc.

The majority of cases met with, however, are due to *endogenous* causes, *i.e.,* the vicarious elimination by way of the lungs, of poisons formed in the body as a result of some morbid process.* Thus, exposure to cold and damp is a most prolific source of bronchitis. The warm—and perhaps perspiring—skin is exposed to conditions, a draught for instance, which suddenly reduce its temperature. Tissue catabolism requiring a certain temperature (without which the trypsin, the ferment upon which the process depends, will fail to act), it is materially impeded as long as the exposure lasts, and quantities of toxic products of imperfect metabolism are formed.* Being gradually transferred by the lymphatic circulation to the blood-stream, these toxic wastes soon reach all parts of the body and are gradually eliminated by the urine, sweat and mucous membranes. The mucosa of the upper respiratory tract taking part in this process and being readily irritated by the special poisons (as it is by iodine and other substances), it becomes inflamed, its most sensitive portion, that lining the nasal cavities, being affected first.* The bronchitis so frequently observed among gouty subjects, and the secondary bronchitis that complicates so many infectious diseases, and also malaria, Bright's disease, etc., are all due to the same cause, *i.e.,* auto-protective elimination of the poisons through the bronchial mucosa and the local irritation engendered thereby.*

The local phenomena incident to the auto-protective process are not those, however, that give rise to the untoward or dangerous features of the disease.* These are due to swelling and œdema of the mucosa, the causes of which vary according as to whether the bronchitis be of exogenous or endogenous origin.* In the former case, these phenomena are produced directly by the irritant. In endogenous bronchitis, they are partly due to this process, the irritants being the poisons derived from the blood, that are being vicariously eliminated through the bron-

* *Author's conclusion.*

chial mucous membrane. The principal cause of the local lesions, however, is a constriction of all the vessels of the body, which occurs as an incidental feature of the primary causative toxæmia—toxic wastes, disease toxins, etc. The blood-mass being thus forced into the capillary system, the capillaries of the bronchi are themselves engorged—sufficiently so in severe cases to provoke marked swelling of the bronchial mucosa.

The *exogenous form*—rarely met with—is well illustrated by six cases reported by J. N. Hall,[1] in which the causes were the inhalation respectively of chlorine gas, sulphurous-acid gas, formaldehyde, kerosene smoke and smoke containing undetermined irritants, and one by Thomas,[2] due to the inhalation of lime dust. To the same class would belong the "bacillary bronchitis" ascribed to the streptococcus by Forchheimer,[3] Patton[4] and others, and to the diplococcus pneumoniæ by Ritchie,[5] P. W. Williams[6] and other observers. Pharyngeal erysipelas and diphtheria may thus prove a source of acute bronchitis by extending downward. That under such conditions infiltration amounting to obstructive œdema may occur, is shown by the familiar lesions produced in the upper respiratory tract, the larynx, for instance, under similar circumstances. In infants it is especially apt to occur owing to the laxity of the cellular tissue.

The well-known fact that cases of gout and Bright's disease are liable to acute bronchitis and that it may appear in such subjects without exposure to cold, points to the *endogenous* cause of the disease in healthy subjects after such exposure, since both gout and Bright's disease are attended by an accumulation of toxic products of imperfect catabolism. Many bacterial infections act the same way; thus the secondary bronchitis of variola is accompanied by the specific pustular inflammation of the bronchial mucosa—a counterpart of the cutaneous inflammatory lesions due to elimination of the specific toxic elements of the disease. In some exanthems, scarlatina and measles for instance, the conjunctival, nasal, pharyngeal and bronchial mucosæ may even precede the cutaneous eruption. Lancereaux[7] contends, in fact, that "'few morbid agents leave the respiratory tract absolutely intact." As is well known, iodine, when given but slightly in excess of the quantity utilized by the body, causes "iodism," including cough in some cases, through a process similar in every respect to that which prevails in any form of endogenous bronchitis. In all these causative conditions the general blood-pressure is raised. In the febrile infections it is high owing to the fever itself, as shown by the flushed face, the excessive warmth of the skin, etc.; gout, Bright's disease and malaria, for example, are also attended, as is well known, by periodical elevations of the blood-pressure.

Treatment. — The two main indications in view of the pathogenesis of the disease are: (1) to counteract the general vasoconstriction and relieve the engorgement of the bronchial

[1] J. N. Hall: Phila. Med. Jour., Dec. 20, 1902.
[2] Thomas: Atlanta Med. & Surg. Jour., Dec., 1888.
[3] Forchheimer: Med. News, June 1, 1901.
[4] Patton: N. Y. Med. Jour., Mar. 28, 1903.
[5] Ritchie: Jour. Path. and Bact., vol. vii, p. 1, 1901.
[6] P. W. Williams: Bristol Med.-Chir. Jour., June, 1902.
[7] Lancereaux: Gaz. des hôpitaux, vol. lxviii, p. 1061, 1895.

capillaries;* (2) to promote elimination through normal channels, the kidneys, intestines and skin, of the toxic substances, which, by being vicariously eliminated by way of the lungs, provoke the bronchial hyperæmia.

Both these indications are materially facilitated if the patient can be kept in bed. The formation of wastes which attends physical exertion is thereby limited to its lowest degree, and what catabolic and oxidizing energy the blood can spare over and above that required for tissue metabolism, is entirely utilized for the destruction of the pathogenic elements. Even the vascular tension is favorably influenced, since the waste-products developed during muscular activity tend to raise the blood-pressure. The warmth of the bed also exercises a favorable influence by causing blood to circulate more freely in the skin, and by facilitating diaphoresis.

In the average case, remedies which lower directly the blood-pressure may be advantageously employed, especially in view of the fact that they tend to relieve simultaneously the harassing dry cough, and to reduce greatly the character of œdematous infiltration of the bronchial mucosa. The *sodium bromide,* 15 grains (1 gm.) and *chloral hydrate,* 10 grains (0.6 gm.), given jointly every three hours in a solution containing merely simple syrup or syrup of lemons (but no expectorant) soon procure these results and afford the patient considerable relief, by depressing the sensitiveness of the general vasomotor center.*

Some of my own cases were relieved after a couple of doses of this mixture, though the dyspnœa was already troublesome. Its use must be avoided in debilitated subjects, however, since their auto-protective functions would be further weakened. Persons otherwise in normal health are not materially influenced in this direction, and the pathogenic poisons in the body-fluids are gradually catabolized and eliminated by the normal paths.

When these agents cannot be employed, *veratrum viride,* 10 minims of the tincture (0.6 gm., 1905 U. S. P.) every hour, three times, then every two hours, may be given instead. Although its physiological effect is almost similar, it tends to cause general depression in some cases, and does not always moderate the cough. When there is oppression, an indication

* *Author's conclusion.*

that oxygenation is being hampered in the smaller bronchi, it replaces advantageously the chloral hydrate. *Aconite,* in drop doses (1905 U. S. P.) every hour, is preferred by some authors.

Stimulation, the use of balsams and expectorants, do more harm at this stage than good. A reliable preparation of veratrum viride, for instance, should be prescribed, as the tinctures obtained in the shops cannot always be depended upon.

When, as is usually the case, the patient cannot be prevailed upon to remain at home, the same results may be obtained by remedies which counteract the bronchial hyperæmia, by causing, through their stimulating action on the sympathetic center, constriction of all arterioles.* In this manner, the blood cannot reach the capillaries freely, and their engorgement is prevented.* Opiates are the most reliable agents of this kind. *Dover's powder,* 10 grains (0.6 gm.) taken at bedtime with a large tumberful of water, is especially advantageous, since its ipecacuanha counteracts the evil effects of the opium by promoting diaphoresis and hepatic activity—the main antitoxic resource of the organism. During the succeeding day, Dover's powder should not be used, however, since the diaphoresis produced exposes the patient to the effects of cold. The beneficial action on the lungs may be sustained, however, with *heroin,* $^1/_8$ grain (0.008 gm.) every three hours, and the Dover's powder resumed the second night, the dose being reduced, however, to 5 grains (0.3 gm.). *Codeine,* in doses of $^1/_4$ to $^1/_2$ grain (0.016 to 0.03 gm.), is preferred by some physicians. If constipation occurs (in adults) a full dose of *citrate of magnesia* is indicated to enhance the elimination of the pathogenic toxics by the intestinal canal.

In the acute bronchitis of infancy and childhood, opiates do not act as satisfactorily. *Calomel,* $^1/_6$ grain (0.01 gm.), rubbed up with sugar and milk and given every two or three hours, produces, after a few doses have been taken, a copious evacuation of the bowels and disappearance of the morbid symptoms, including fever, if the case belong to the febrile form Active stimulation of the adrenal center and a rapid increase of the auto-antitoxin, is obviously the effect produced under these conditions.*

* *Author's conclusion.*

In some cases, the dyspnœa is sufficiently marked to demand more active measures to lower the blood-pressure.* A few whiffs of *iodide of ethyl,* inhaled from a handkerchief upon which 10 drops of this remedy have been poured, relieve the distressing symptom in a few minutes. *Nitrite of sodium,* 1 grain (0.065 gm.), in a little water, produces a similar but more lasting effect. Inhalations of *oxygen* are of great value when a tendency to cyanosis occurs.

The importance of this symptom was emphasized by B. W. Richardson,[8] Bruce[9] and others. It comes on late in the course of the disease, and is complicated by the accumulation of fluids in the smaller bronchi. Many cases of acute bronchitis in which "shortness of breath" is complained of, are nearer asphyxia than they are thought to be by their physicians. Nitrite of sodium and nitroglycerin have been recommended by Frazer,[10] and iodide of ethyl by Main[11] and others. Oxygen is spoken of as a life-saving measure by Sinainski,[12] Langston,[13] Leech[14] and many other observers.

An important feature of the treatment of febrile bronchitis is the ingestion of alkaline fluids to facilitate the elimination of toxic wastes by the kidneys, and antagonize acidosis, as indicated on page 1367. When the acute stage recedes, the elimination of the bronchial detritus must also be facilitated by remedies which increase the alkalinity of the blood and facilitate general osmosis, including that of the peribronchial fluids.* *Ammonium chloride* is an active remedy of this kind, in 10-grain (0.6 gm.) doses every three hours, given in a mixture containing 30 minims (1.85 c.c.) of syrup of tolu to the dose.

If the convalescence is delayed, the chloride may be replaced by *ammonium iodide,* which stimulates somewhat the adrenal system, owing to the iodine it contains. More active in this connection, however, is a mixture containing *potassium iodide* and *ammonium carbonate,* 5 grains (0.3 gm.) of the former and 10 grains (0.6 gm.) of the latter to the dose. This combination is very effective in children, the dose, of course, being adjusted to the age of the little patient.

When after recovery from the more acute symptoms, cough and a muco-purulent expectoration persist, the fluid

* *Author's conclusion.*
[8] B. W. Richardson: Med. Press and Cir., Jan. 25, 1888.
[9] Bruce: Lancet, May 30, 1891.
[10] Frazer: Amer. Jour. Med. Sci., Feb., 1888.
[11] Main: Brit. Med. Jour., Nov. 30, 1889.
[12] Sinainski: Lancet, Sept. 1, 1888.
[13] Langston: Brit. Med. Jour., Jan. 30, 1892.
[14] Leech: Practitioner, May, 1898.

extract of *hydrastis,* 20 to 30 drops, four times daily, in a little sweetened water, is sometimes quite efficacious, acting much like the opiates without being attended by their untoward effects. *Caffeine,* 5 to 10 grains (0.3 to 0.6 gm.), in a solution containing 20 grains (1.3 gm.) of *potassium acetate,* administered on retiring, is to be preferred when the cough is troublesome at night. *Apomorphine,* in doses varying from $^1/_{30}$ to $^1/_2$ grain (0.002 to 0.03 gm.), given after meals to prevent nausea, is preferred by some authors.

In some cases, all these measures prove ineffectual, owing to general asthenia. *Quinine hydrochlorate,* 2 grains (0.13 gm.), given with *strychnine,* $^1/_{40}$ grain (0.0016 gm.), after each meal, is often effective in such cases, owing to its stimulating action on the general centers. A more lasting effect is obtained by means of small doses, *i.e.,* 1 grain (0.065 gm.) of *thyroid extract,* three times daily, after meals.* By enhancing physiologically the functional activity of the adrenal system it activates that of all functions and augments general nutrition.*

BRONCHIAL ASTHMA.

SYNONYMS.—*Asthma; Spasmodic Asthma.*

Definition.—A form of paroxysmal dyspnœa due to the concurrence of two pathogenic factors: (1) hyperexcitability of the general vagal center; (2) the presence within or upon the bronchial mucosa, of endogenous or exogenous irritants. The mucosa requiring for the expulsion of these irritants, reflex impulses derived from the general vagal center, the hyperexcitability of the latter causes it to project unusually violent impulses to all the elements of the bronchi, including their muscles, and these in turn, being inordinately contracted, they reduce the caliber of the bronchi and thus provoke asthma.**

Symptoms.—An attack of asthma may be preceded by one or more premonitory symptoms: slight gastro-intestinal malaise, flatulency, intellectual torpor or exuberance, depression of spirits, pruritus, especially of the trunk and chin, sneezing, accompanied sometimes by a free flow of watery mucus, epistaxis, and in most cases a copious excretion of urine.

* *Author's conclusion.*
** *Author's definition.*

The access proper usually begins abruptly, often during the early morning hours. Marked restlessness is soon followed by dyspnœa of the most distressing kind, and a sensation of great depression about the chest. The patient soon finds it necessary to assume positions that favor the action of the respiratory muscles, which seem unable to cause depression of the thorax and expulsion of the air. Gradually, as this difficulty increases, the dyspnœa becomes more distressing until, after a couple of hours or more, the face becomes dusky and perhaps cyanotic, owing to imperfect oxygenation. In severe cases, this phenomenon is sufficiently marked to cause marked hypothermia, especially of the extremities. The signs of asphyxia are so intense in some cases, that they may suggest, even to the physician, an early lethal termination. The pulse is small and rapid, the brow is bedewed with sweat, the eyes stand out prominently, a deadly pallor replaces the cyanotic hue, etc.

The movements of the chest are suggestive. Though fully expanded, it fails to recede to any marked extent during expiration. The inspirations are short, but the expirations are considerably prolonged, thus reversing the normal relations between the respiratory acts. The percussion note is hyperresonant, sometimes almost tympanic, but highly-pitched. Auscultation confirms the objective respiratory phenomena: the expiration is greatly prolonged, and both inspiration and expiration are attended by sibilant, more or less high-pitched râles, throughout the entire chest, which resemble the chirping of a multitude of birds. The heart is often displaced, the apex being nearer the sternum and lower down than usual—obviously pushed in this unusual position by the distended left lung. The veins of the neck are, as a rule, considerably dilated.

When the paroxysm has reached its worst stage, the picture changes: the breathing becomes easier, and a spell of tight, harassing cough marks the onset of the period of resolution. At first the sputa are quite characteristic of asthma. They contain small, grayish-white gelatinous pellets, Laennec's "pearls," which are in reality small rolls of condensed mucus, containing two characteristic elements, Curschmann's spirals and the Charcot-Leyden crystals. The spirals are skeins of spirally-disposed mucin filaments enclosing many eosinophile leucocytes;

the crystals are similar to those found in the blood in leukæmia. An enormous number of free eosinophile leucocytes are also found in the sputum, as well as in the blood. Gradually, however, the expectoration assumes the ordinary mucoid type; as it becomes freer the cough becomes less harassing, and the attack passes off, leaving the patient considerably weakened.

The relief may be of short duration, a second attack following the first after a few hours. As a rule, however, the paroxysm does not occur until the succeeding night, more or less coughing and wheezing occurring in the interval. Paroxysms may thus reappear five or six nights in succession, the series constituting an "attack" of asthma. The patients may then remain free several weeks, or even months, and suddenly lapse into another period of suffering.

In true bronchial asthma, the intensity of the symptoms is independent of the duration of the disease. Thus, the attacks that occur soon after its first appearance may be very intense as to dyspnœa, while in individuals who have long suffered from the disease, this symptom may not be severe. In the former cases, however, the attack passes off entirely, leaving the patient perfectly normal during the intervals, while in the latter, the bronchial mucosa may become the seat of a chronic inflammatory process, *i.e., a true* chronic bronchitis, with all its possible complications.

Pathogenesis and Pathology.—An attack of bronchial asthma is due to vagal stricto-dilation of the bronchial arterioles.* An excess of blood being admitted into the bronchial muscle-elements, and into the mucosa, the caliber of the bronchi is reduced in two ways: (1) by contraction of their muscles, and (2) by congestive swelling of the bronchial mucous membrane. In severe attacks the lumina of the terminal bronchioles becomes sufficiently reduced to prevent the egress of air from the alveoli, hence the cyanosis and the inability of the thorax and the diaphragm to contract.

All the theories best sustained by experimental and clinical evidence implicate the nervous system in the morbid process. Whether with Lefèvre, Salter, Biernier, Trousseau and others we ascribe asthma to spasm of the bronchial muscles; with Hack, Daly and others to reflex irritation of the nasal cavities; with Parrot to reflex excitation of the

* *Author's conclusion.*

bronchial secretory elements; with Brissaud to hysteria; or with Weber to vasomotor turgescence of the bronchial mucosa, etc., the nervous system stands out prominently as an active factor at least of the salient phenomena of the disease, the bronchial obstruction. The unanimity ceases, however, when the identity of the system of nerves involved is sought. Are we dealing with a vagal neurosis, as taught by Trousseau and his school, or with a vasomotor neurosis, as taught by Weber and his followers?

The question of course is closely associated with the character of bronchial lesions. Here, again, the bulk of evidence points in the one direction, *i.e.*, Traube's view that the bronchial mucosa is the seat of a fluctionary hyperæmia which Sir Andrew Clark, Störch and others interpret as a diffuse "vaso-turgescence" or hyperæmic swelling. Not only was Fraenkel[15] able to verify this fact post-mortem, but he found the lesions peculiar to an advanced stage of local congestion, *i.e.*, of chronic bronchitis, widespread desquamation of epithelial cells. In a very interesting case recorded by J. S. Billings, Jr.,[16] such a vaso-neurotic swelling involved "the forearms and arms as high as the middle of the biceps muscles." Although the fingers were not swollen, they had been "cold and blue from the first." The "typical asthma, with nocturnal attacks of sudden onset, ending in violent paroxysms of coughing with scanty viscid expectoration," ceased, and the swelling of the arms and forearms disappeared under what, as we will see, I regard as the appropriate treatment for asthma. The pulmonary lesions and those of the extremities were evidently similar, *i.e.*, a fluctionary hyperæmia of nervous origin. That this vascular turgescence is general is suggested by the observation of Sihle[17] that in all asthmatics, both during the attacks and in the intervals, the inferior border of the liver is considerably lowered and sensitive to pressure.

The identity of the system of nerves involved at once suggests itself, *i.e.*, the vasomotor system, in accord with the views of Weber and many other observers. When, however, the manner in which such widespread results must be produced by this system is analyzed in the face of experimental evidence, it fails to meet the needs of the morbid process. In order thus to influence the general vascular system, the original cause of the disease, whether it be "uric acid" or any other systemic poison, should stimulate the vasomotor center, and produce general vaso-constriction; more blood being projected into the capillary system, the bronchial capillaries among others would become engorged, i.e., hyperæmic. That such a general vaso-constriction does not occur, however, is shown by the fact that the blood-pressure, which is raised when the systemic vessels contract, is on the contrary lowered, as shown by Sihle, the average being from 70 to 100 in a large number of asthmatics as compared to the average of 80 to 130 in normal individuals. Again, if the data upon which the vasomotor theory is based are carefully scrutinized, they are found wanting. Stimulation of the pulmonary vasomotors produces the reverse of hyperæmia, *i.e.*, ischæmia, and the many symptoms other than asthma observed that are ascribed to vasomotor influence, rhinorrhœa, ptyalism, lachrymation, diarrhœa, etc., are in reality due to overstimulation of the respective organs by their secreto-motor nerves—all of which, as I have shown in the eighteenth chapter, fulfills functions identical to those of the vagus.

Quite another picture is presented, however, when the asthma, in accord with Trousseau's interpretation, is ascribed to the vagus. Brodie and Dixon[18] recently confirmed experimentally the well-known fact that

[15] Fraenkel: Zeit. f. klin. Med., Bd. xxxv, S. 559, 1898.
[16] J. S. Billings, Jr.: N. Y. Med. Jour., May 22, 1897.
[17] Sihle: Wiener klin. Woch., Bd. xvi, S. 85, 1903.
[18] Brodie and Dixon: Trans. Pathol. Soc. of London, vol. liv, p. 17, 1903.

stimulation of the vagus causes a marked diminution of the volume of air entering and leaving the corresponding lung, owing to contraction of the bronchial muscles. Kingscote[19] states, moreover, that this procedure causes simultaneously spasmodic contraction of the diaphragmatic muscle. As stated by Loque,[20] moreover, a similar effect is produced when the pulmonary ends of the nerve, *i.e.*, the bronchial muscles *per se*, are excited.

The manner in which these phenomena are produced is made evident when the observations of Sihle,[21] that this procedure produces bronchostenosis, "the efferent path being certainly the vagi;" and that of W. Blair Bell,[22] that pulmonary terminations produce *active* vasodilation of the bronchial arteries, are interpreted from my standpoint, *i.e.*, with the vagus as a *stricto-dilator* nerve in common with all motor nerves. Indeed, stricto-dilation means, as I have shown, constriction of the vasa vasorum of the arterioles that supply muscles (as well as any other organ); the walls of these arterioles being supplied with less blood, they dilate, admitting in turn more blood into the muscular elements. Now we have in its action upon the diaphragm, proof that the vagus causes muscular contraction. Hence, in accord with the interpretation of Lefèvre, Salter, Trousseau, C. J. B. Williams and many others, the spasmodic contraction of the bronchial muscles which, by greatly narrowing the caliber of the bronchi, provokes the asthma. Hence, also, the "hyperæmic swelling" upon which Traube, Sir Andrew Clarke and others have laid stress, since stricto-dilation of the bronchial arterioles produces congestion of all the bronchial elements.

As emphasized by H. L. Swain,[23] the researches of W. S. Miller have shown that venous trunks are practically absent in the bronchial mucosa, which is thus supplied only with capillaries. The readiness with which the mucosa can become hyperæmic, and conversely the rapidity with which the capillaries can be depleted as soon as the excess of blood supplied to them is reduced (by sympathetic constriction of the arterioles as shown below), is self-evident.

The predisposing cause of bronchial asthma is hypersensitiveness of the vagal center in the posterior pituitary body.* Under normal conditions, that is to say, when the bronchial mucous membrane is not irritated by some substance brought to it by the air current or by the blood, this hypersensitiveness gives rise to no untoward phenomena. When, however, irritating particles are inhaled and reach the lungs, or when catabolism is imperfect and toxic products of hypocatabolism are eliminated (in part) by the bronchial mucosa, asthma occurs.*

The muscular constriction of the bronchi and the swelling of the mucosa which give rise to asthma, are not caused, however, by a direct action of these poisons upon the bronchial muscles, or the epithelial or secretory elements of the mucosa,

* *Author's conclusion.*
[19] Kingscote: Brit. Med. Jour., Oct. 13, 1900.
[20] Loque: Jour. des sci. méd. de Lille, Jan. 19, 1895.
[21] Sihle: *Loc. cit.*
[22] W. Blair Bell: Edinburgh Med. Jour., Oct., 1899.
[23] H. L. Swain: Yale Med. Jour., Aug., 1900.