A Résumé of Recent Literature.

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A really comprehensive abstract dealing with the neuroses of the respiratory tract should be the product of "team-work." The team should consist of a physiologist, a pathological chemist, a neurologist, an internist, and a rhinologist. Such a team, difficult to collect in peace time, is at present out of the question. With this apology the abstractor presents some recent views—mainly those of American writers—regarding the respiratory neuroses, which may perhaps be better described as sensitisations of the respiratory tract."

The nasal mucous membrane consists of several layers. If we take the inferior turbinals as an example we find that next to the bony core of the turbinal there is a layer of periosteum containing numerous elastic fibres. Just outside this lie the larger blood-vessels and nerves. To a microscopist not accustomed to the examination of the nose the nasal blood-vessels appear to have remarkably thick walls. In the delicate submucous connective tissue we find a large number of thin-walled blood sinuses, which compose the erectile tissue of the turbinal. Along with these there are numerous mucous glands, the ducts of which open at various points on the surface. The superficial epithelium consists of several layers of cells, the most superficial of which is of the ciliated columnar variety, while the deeper cells are spindle-shaped and cubical. The latter lie on a delicate basement membrane. The elastic fibres from the periosteum radiate out and surround the blood sinuses and mucous gland; they end in a fine network beneath the basement membrane. These fibres no doubt aid in the shrinkage of the nasal mucosa when the vascular engorgement of the blood sinuses is over. Although the structure of the mucous membrane lining the nasal accessory sinuses and the lower portions of the respiratory tract is less complicated than that covering the turbinals, the difference is only one of degree.

The nasal mucosa appears to be constantly passing through three stages—(1) engorgement, (2) stage of secretion, followed by (3) a resting stage. If we test our nasal passages by stopping each
nostril alternately and breathing through the other, most of us will find that at any given moment we have one more or less patent nostril and one somewhat obstructed. If we investigate again after a varying period we may find that the previously clear side is now obstructed and vice versa.

In the milder varieties of nasal neurosis only the upper part of the respiratory tract is involved. The patients complain of intermittent blocking of the nasal passages, especially at night. This condition is often seen in overworked people, especially in brain workers, and is not infrequently met with in medical students studying for examinations. In the second degree of nasal neurosis we have increased secretion, and frequently attacks of sneezing along with the nasal obstruction. The sneezing attacks are often of a violent and prolonged character. In the last and most severe form the lower portion of the respiratory tract is also involved, and we have well-developed attacks of asthma and increased bronchial secretion.

The nasal neuroses have long been the opprobrium of rhinologists. This is not to be wondered at, considering our ignorance of the anatomical, physiological, and pathological conditions concerned in their production. Many of the nasal neuroses are erroneously attributed by patients to the effects of chronic or recurrent "colds" in the head—that is to say, to attacks of rhinitis or coryza. It must be obvious to us that many of these cases are not really due to infectious rhinitis, i.e. they are not caused by bacterial infection of the nasal or respiratory mucous membrane. For instance, a patient complains that, on getting out of bed in the morning, he immediately suffers from a sudden attack of sneezing. Here the reflex is much too quick to be due to any bacterial or toxic action.

That the condition of the nasal mucosa is under nerve control is shown by the effect of certain emotional states. This is well illustrated in the common phrase "open-mouthed with astonishment." The astonished person opens his mouth because his nose is obstructed by reflex engorgement of the nasal mucosa. The nerve mechanism involved does not belong to the voluntary or "animal" portion of the nervous system, but to the involuntary or "vegetative" system (see later). From the point of view of development, as well as from that of staining reaction, there appears to be a close connection between the vegetative nervous system and certain of the ductless glands. As our knowledge of the endocrine glands increases it is to be hoped that we may arrive
at a more rational treatment of the nasal neuroses as well as of many other difficult and obscure conditions. It must, however, be admitted that at present treatment of the nasal neuroses by the administration of extracts of the ductless glands is apt to partake of the nature of "blunderbuss" therapy.

It is a remarkable fact that the nasal reflex neuroses are much more common among the so-called "better" classes than among hospital patients. This fact is said to have led certain observers who were treating hay fever by the injection of the serum of horses immunised to pollen toxin to use only thoroughbred horses in their experiments.

Shurley\(^9\) holds that the so-called "nasal neuroses" are really sensitisations of the respiratory tract. They should be classified as follows:—I. Cases sensitised to the pollen of certain grasses and plants; II. Those sensitised to certain kinds of food; and III. Cases which are sensitised to a particular microbe or microbes, \(i.e\). patients liable to recurrent attacks of coryza or "cold in the head."

**Physiology.**

*The Nerve Mechanism Involved.*—Fetterolf,\(^8\) following the work of Eppinger and Hess, states that the nervous system may be divided into two sections—(1) the animal, and (2) the vegetative. (1) The animal group consists of sensory and motor fibres, and supplies the organs of sensation and the voluntary muscles. (2) The vegetative system, on the other hand, supplies all glands that have ducts and all involuntary muscle tissue. The vegetative system consists of two distinct parts—\((a)\) the autonomic, and \((b)\) the sympathetic systems. These two cannot be separated by dissection, but through various tests their individuality has been established beyond question.

The place of origin of the vegetative fibres is the brain and spinal cord. The sympathetic portion arises from the cord alone, beginning at the second thoracic nerve and continuing down to and including the second lumbar.

The fibres which comprise the autonomic portion arise in part from the brain stem and in part from the sacral region of the cord. The former—with which we are concerned in this abstract—are contained in the third, seventh, ninth, tenth, and eleventh cranial nerves. Those of the third nerve go to the ciliary ganglion, whence other neurons take up the impulses and carry them to the ciliary muscle and the sphincter of the iris.
The autonomic fibres of the seventh nerve (pars intermedia of Wrisberg) with those from the ninth, tenth, and eleventh go to sundry ganglia whence secondary or post-ganglionic fibres emerge. These innervate the blood-vessels of the mucous membrane of the mouth, throat, nose, and paranasal sinuses, the salivary glands, the heart muscle, the glands and musculature of the trachea and bronchi, the glands and musculature of the digestive tube from the oesophagus to the colon, as well as the blood-vessels and tubules of the kidney. In action the two groups—the autonomic and the sympathetic—are antagonistic, and it is generally conceded that all glands which possess ducts and all involuntary muscles receive a supply from both. Upon the maintenance of a proper balance between the two depends the normal functionating of the structures to which they go. If the autonomic supply is irritable, the organ will over-functionate in one direction. We then have the condition of "vagotonia," i.e. hypertonus of the above-mentioned group of nerves called the "extended vagus." If the sympathetic is in a condition of hypertonus—"sympatheticotonia"—the excessive action will be in the other direction. The results are manifested by hyper- or hypo-secretion on the one hand, or by spasm or relaxation on the other, i.e. in vagotonia we have hypersecretion and spasm, while in sympatheticotonia we have hyposecretion and relaxation.

The control of the entire vegetative system, both autonomic and sympathetic, is believed to lie in the cerebro-spinal axis, where possibly a regulating centre exists, and in the glands of internal secretion, the so-called "endocrinous glands." Adrenin has been shown to act generally as a stimulator to the entire distribution of the sympathetic group. For the autonomic system no drug has been found which acts uniformly upon all parts. The nearest approach to such a drug is atropin, which is a sedative to practically the entire autonomic system, dilating the pupil, checking the flow of saliva and sweat, and relaxing contracted involuntary muscle. Pilocarpin has a powerful effect upon the salivary and sweat glands, producing over-secretion, and hence it is clearly to be regarded as a stimulator of part of the autonomic system. Pilocarpin (grs. \(\frac{1}{20}-\frac{1}{6}\) hypodermically) is used in testing for the presence of vagotonia. If the test is positive we get salivation, lacrimation, sweating, cardio-respiratory arrhythmia, and hyperperistalsis—all these out of proportion to the size of the dose. In vagotonia we also have a relative decrease of the reaction to a sympathetic stimulant, such as adrenin. The symptoms of vago-
tonia in general are those of stimulation of the autonomic system, e.g. cramp of the ciliary muscle, spasm of accommodation and widening of the palpebral fissure (otic ganglion), salivation, congestion and hypersecretion in the nose and pharynx (sphenopalatine and submaxillary ganglia), bronchial asthma and hypersecretion, laryngeal crisis, bradycardia, gastric crisis, hyperperistalsis, and excessive gastric and intestinal secretion (vagus). The blood-picture shows an excess of eosinophiles.

The medical treatment of vagotonia may take one of two forms—(1) sedative treatment of the autonomic system; or (2) stimulation of the sympathetic. For example, spasmodic bronchial asthma may be treated by quieting the autonomic system with atropin or by stimulating the sympathetic by the local or hypodermic use of adrenin.

Brubacker points out that the nasal mucous membrane is thick and spongy and is provided with a large number of glands, both mucous and serous. In order that the glands may be enabled to elaborate the necessary amount of secretion it is essential that the vascular supply should be varied from time to time. The secretion of the glands as well as the blood-supply impart to the nasal chambers that degree of moisture and temperature necessary to prepare the inspired air for its entrance into the lower respiratory tract. The nerves distributed to the blood-vessels and glands belong to the autonomic system and hence consist of two consecutively arranged neurons.

The Efferent Nerve Mechanism.—This consists of pre-ganglionic and post-ganglionic fibres. The latter directly excite to activity the epithelium of the glands and relax the muscle fibres of the blood-vessel. They originate in the sphenopalatine (Meckel's) ganglion, situated in the sphenomaxillary fossa. [The otic, sphenopalatine and submaxillary ganglia, though anatomically associated with the trigeminal nerve, are not integral parts of it; they belong to the autonomic system, and are associated with the vasomotor and secretory system of nerves. They possess neither sensory nor motor functions.] From their origin in Meckel's ganglion the nerve fibres pass to the glands and blood-vessels of the nasal chambers, palate, and upper portion of the pharynx. The ganglion cells, though irritable, are devoid of automatic action, and must be excited by nerve impulses discharged by the nerve cells in the central nervous system and transmitted by the pre-ganglionic fibres. These fibres originate in nerve cells situated
in the grey matter beneath the floor of the fourth ventricle. From this they pass forwards and emerge from the side of the medulla between the facial and acoustic nerves as the *pars intermedia* of Wrisberg, of which they constitute a part. The nerve of Wrisberg accompanies the facial as far as the geniculate ganglion, but then leaves it to pass forward to the sphenopalatine ganglion as the great petrosal nerve. In this way nerve impulses discharged by the central cells are transmitted by the nerve of Wrisberg and the great petrosal to the cells of the sphenopalatine ganglion, which in turn discharge impulses that are distributed in a spray-like manner to the glands and muscular walls of the arterioles. Experiment has shown that, if the great petrosal nerve is exposed and divided and the peripheral end stimulated, we get a dilatation of the blood-vessels and a discharge of secretion from the glands of the mucous membrane of the nose and associated structures. The great petrosal nerve therefore contains both secreto-motor and vasodilator fibres.

The degree of contraction of the blood-vessels of the nasal mucosa is regulated partly by vasoconstrictor nerves, which also consist of two consecutively arranged neurons and therefore belong to the autonomic system. The peripheral neuron arises in the superior cervical ganglion, from which post-ganglionic fibres pass upward and assist in the formation of the plexus on the internal carotid artery. At the point where the great petrosal nerve crosses this artery some of these sympathetic fibres enter its sheath and accompany its branches to their terminations in the blood-vessels and possibly also the glands. The pre-ganglionic fibres originate in the cells of the general vasoconstrictor centre beneath the floor of the fourth ventricle. From this the fibres descend the spinal cord to pass out in the ventral roots of the upper thoracic nerves, and thus reach the vertebral chain of sympathetic ganglia to pass upwards to the superior cervical ganglion. There is thus a rather circuitous pathway between the vasoconstrictor centre and the blood-vessels of the nasal mucous membrane. Experiment has shown that if the cervical sympathetic cord be divided the blood-vessels of the nasal chambers, as well as those of the entire side of the head and face, will markedly dilate for some days. If the peripheral end of the divided cervical sympathetic cord is stimulated the blood-vessels at once contract. It will thus be seen that the blood-supply of the nasal mucosa is under the control of two antagonistic groups of nerves—dilator and constrictor. The calibre of the vessels at
any moment is the resultant of the relative degree of activity of these two groups.

The Central Mechanism.—The nerve cells of the two groups—vasodilator and vasoconstrictor—possess a certain degree of tonicity. They may be excited or inhibited by nerve impulses (1) transmitted from the nasal chambers by the nasal and other branches of the trigeminal nerve and from the skin by the cutaneous nerves (reflex or peripheral stimulation); (2) descending from the cerebrum in consequence of psychic states of an emotional character (direct or central stimulation). It is evident from a study of pathological conditions that the tonus of one or more of these centres may be abnormally increased or decreased by changes in their nutrition caused by the toxic products of metabolism, e.g. dietetic or alcoholic excesses, insufficient physical exercise or fresh air. If the tonus of the central mechanism is impaired, peripheral causes, which at one time would produce little or no effect, now give rise to pronounced and distressing symptoms.

The afferent nerve mechanism is to be found in the branches of the trigeminal nerve. The cells of the semilunar (Gasserian) ganglion give origin to a short process which soon divides into two branches, one of which passes centrally, the other peripherally. The central branch forms the large or sensory route. The peripheral branches constitute the three main divisions of the nerve. The axons of the central branches after entering the pons ultimately arborise around the vaso-inhibitor and vasoconstrictor and secreto-motor centres. The peripheral branches pass forward to be distributed to the skin, mucus membrane, and other structures of the head and face. Other of the central branches arborise around “sensor-end-nuclei,” the axons of which pass upward to arborise around sensory nerve cells of the cerebral cortex. Stimulation of the first group calls forth reflex phenomena; stimulation of the latter group produces pain.

Peripheral Stimulation of the Central Mechanism.—The stimulus to the peripheral terminations of the nasal and cutaneous nerves is the variation in temperature and moisture of the air. The amount of heat radiated from the blood and the amount of secretion produced by the glands ought to vary with the varying temperature and moisture of the inspired air. The reactive adaptation of the individual varies with his age, sex, occupation, habits, and the stability of his nerve centres. If a fairly normal individual be subjected to a temperature of 60° F. it will be found that the
blood-vessels of the nose will possess a certain calibre, the resultant of the co-operative action of the vasodilator and vasoconstrictor nerve centres. This calibre will permit the passage of a definite volume of blood with a definite velocity in a unit of time carrying with it a certain volume of heat. If the external temperature should fall to 30° F. the arterioles of the nasal mucous membrane contract as the result of the now preponderant action of the vasoconstrictor centre due to the stimulation of the afferent nerve endings by the cold air. At first sight this would appear to diminish heat radiation, but it must not be forgotten that though the thickness of the blood-stream is diminished, its speed is increased, so that the temperature of the air is correspondingly raised. On the other hand, should the external temperature be raised to 90° F., the blood-vessels will dilate, the thickness of the blood-stream will be increased, and its speed diminished. With regard to the secretory mechanism, similar statements may be made.

I. Inhalation Anaphylaxis, Hay Fever or Pollinosis.

Pathology.—Manning defines hay fever as an exudative catarrh of the conjunctival, nasal, and tracheo-bronchial mucous membrane, produced in hypersensitive individuals by the sensitising and anaphylatoxic action of the pollen of certain plants. The mucous membrane of the respiratory tract affords a ready means of access for the entrance of certain foreign proteids into the body. Hay fever must not be confused with hyperaesthetic rhinitis, which may occur at any time of the year, and does not carry with it the general constitutional depression which is so characteristic of true hay fever.

Manning states that the bactericidal power of the nasal secretion is due to a proteolytic enzyme. The pollen protein reaches the nasal mucous membrane of all persons, and in most of them the enzyme gradually splits the pollen protein into harmless products—proteoses and amino acids. This occurs slowly, so that the absorption of protein is exceedingly minute. Certain obscure conditions interfere with the normal digestive function of the nasal mucosa. When these conditions arise, sufficient protein may be absorbed to lead to sensitisation. Whatever the cause of the disturbance, there occurs an intake of foreign protein which so injures the mucosa that it remains permanently in a state of increased permeability for the protein.

According to Cooke, any form of foreign protein introduced within the living body gives rise to the formation of a specific
immune or antibody, which exists either attached to certain cells or free. When union takes place between protein and free antibody there is no clinical evidence of a reaction, but when a union takes place between protein and fixed antibody a reaction takes place. When there is a large excess of antibody circulating free we have an immune state, and when there is little antibody, and that for the most part attached, we have the sensitised state (anaphylaxis). With cessation of protein injection the body returns to the anaphylactic state, i.e. the duration of immunity is strictly limited. We do not know why a certain group of individuals become and remain sensitised, though we know that the capacity is largely inherited (64 per cent. of cases).

Grayson\textsuperscript{12} states that we have not so far been able to arrange with accuracy the sequence of events which terminate in enfeeblement of vasomotor control. Grayson himself finds that two-thirds of his cases have given a history of inherited neurotic tendency. In all the other (acquired) cases there were reasons of an emotional, mental, or physical nature to account for that general nervous exhaustion of which the loss of vasomotor equilibrium was but a symptom and a sequel. Grayson finds that there is a third group of cases, which can be explained upon the theory of anaphylactic reaction. In these it is usual to find urticaria on the skin as well as other areas of angioneurotic oedema, besides that in the nose. Grayson holds that the internist, the neurologist, and the rhinologist are all concerned in the treatment of these cases. He believes that in a very large percentage the neurasthenia primarily and the nasal neurosis secondarily are the products of a long-continued violation of the laws of personal hygiene, particularly those concerning diet, exercise, work, and play. He regards the relationship of the vasomotor neuroses of the nose to abnormal metabolism as well established.

Oppenheimer and Gottlieb\textsuperscript{18} state that heredity plays a very important \textit{rôle} in the etiology of pollinosis. Over 90 per cent. of patients have other members of their family who suffer from allied ailments, such as urticaria, asthma, or other manifestations of anaphylaxis, following the ingestion of casein, egg-white, shell fish, or are sensitive to dust or the serum of horses and cats. The nasal factor should not be considered as too important; many patients have had considerable surgical treatment of the nose, and it has been in but the rarest instance that benefit has resulted. Marked stenosis or suppurative processes in the accessory nasal cavities should, however, be corrected. The abstractor has found that if a
patient suffering from a nasal neurosis presents a marked nasal abnormality, such as a very deflected septum with turbinal enlargement, all that one can safely say is that operation will ensure free nasal respiration. If the neurosis is benefited or cured, the patient must be thankful, but he must not count on this result.

Plants which are wind fertilised and therefore distribute a large amount of pollen in the air excite hay fever symptoms. There are more than fifty plants to the pollen of which hay fever subjects may be sensitive (Goodale). (Plants which are fertilised through the agency of insects need not be considered.) If hay fever symptoms occur previous to the flowering of the grasses we have to look for the exciting cause in the early flowering shrubs and trees, e.g. birch and willow. Hay fever due to the grasses usually begins in this country in the early part of June. Noon and Freeman have shown that the injection of a solution of the pollen from one species of grass can protect against other grasses. Goodale's experience confirms this observation. The composite may also give rise to hay fever, and here again the proteid of all members of this family is essentially the same. The active pollen of one species will, for diagnostic purposes, cover the whole order. According to Goodale the chief causes of hay fever are the grasses and the late composites.

Symptoms.—Wilson divides the symptoms of hay fever as follows:—(a) Characteristic symptoms—itching, redness and swelling of the mucous membrane of the eye, nose and throat, with lacrimation, sneezing, and rhinorrhea: hay fever patients, especially those who breathe through the mouth, inhale into their bronchial tubes large quantities of pollen, which cause swelling of the mucous membrane with the production of typical asthma. Asthmatic symptoms may be perpetuated throughout the year owing to secondary bacterial infection. (b) Miscellaneous symptoms—fever, malaise, and asthenia. Vomiting, diarrhoea, and cardiac troubles are occasionally met with.

Diagnosis.—Three methods may be employed for determining to which pollen a patient is anaphylactic—(1) The ocular; (2) the hypodermic; and (3) the cutaneous method. The ocular method is very convenient, and is advocated by some well-known commercial firms which put up a hay fever reaction outfit. Full instructions for the use of this outfit are enclosed, but briefly it may be said that the procedure is carried out on the lines of the Calmette ophthalmic-reaction for tuberculosis. Oppenheimer and Gottlieb advocate the cutaneous method. A very small scratch, not enough
to produce bleeding, is made on the arm, and a very minute quantity of pure pollen is gently rubbed in. In a few minutes a wheal develops. The swelling and redness are measured after fifteen minutes. Similar vaccinations are made during the course of treatment so as to determine whether the size of the wheal diminishes as immunisation progresses. The intensity of the skin reaction is not always proportionate to the clinical symptoms. In children the skin disturbances are less pronounced than in adults.

Treatment.—According to Grayson, no one will claim that the cauterisation of hyperesthetic areas of the nasal mucous membrane is a rational treatment of hay fever, because it is not directed against the cause of the disease.

Until now the treatment of the nasal neuroses has been almost purely empirical. We have attacked the nasal mucosa by the direct application of an extraordinary number and variety of remedies, we have cauterised and even removed it more or less extensively, but seldom with any better result than incomplete and temporary relief (Grayson).

1. Passive Immunisation.—One may attempt to combat hay fever by adding some substance to the organism which will neutralise the poisonous fraction of the split protein molecule. Dunbar’s “pollantin” is an attempt at passive immunisation. Dunbar injected horses with gradually increasing doses of ragweed pollen with the expectation that a specific antibody would be formed in the serum in sufficient amount for practical use. It must be admitted, however, that this method of treating hay fever has not proved a great success. Recently attempts have been made to revive this method in order to deal with cases which are not able to develop for themselves enough immune body. One large dose of blood-serum from a rabbit that has had large quantities of pollen injected at regular intervals for about five months is given to the patient. In this way anaphylaxis is avoided.

2. Active Immunisation.—One may try to develop in the tissues of the individual a substance which will neutralise the poisonous fraction. In 1910 Noon and Freeman made the first attempt at active immunisation, working with the pollen of grasses. The pollen is collected and dried, and a weighed portion is then ground with sand in an agate mortar together with a small quantity of saline solution. More salt solution is now added, and the whole incubated for twenty-four hours. The mixture is centrifugalised and the clear supernatant fluid used as a basis for
subsequent dilutions. The term "pollen-unit" means the soluble protein contained in 1/1,000,000 grm. of dried pollen. As regards dosage, it is quite safe to begin with one or two units. In many cases considerably more than 1000 units may be safely given after a sufficiently extended preliminary desensitisation. Injections should be given at intervals of three to five days.

Dilutions of the pollen extract are most conveniently made by adding a certain amount of the stock solution to alcohol of the same strength so as to make a 25 per cent., or a 10 per cent., or a 1 per cent. dilution. The initial dose is determined by the dilution which fails to excite a definite reaction in the eye or on the skin. The quantity of material injected hypodermically should not exceed five or ten drops. Before beginning the injections it is best to wait until the reaction from the skin tests has subsided. The prophylactic injections should be begun about two months before the hay fever season, and should be given at intervals of from five to seven days, increasing by a few drops at first, and later by the adoption of stronger percentages of the stock solution. To avoid anaphylactic disturbances, Goodale advances the strength at first very slowly. Hypodermic injection of the extract gives rise to a well-defined lump, varying from the size of a bean to that of a pigeon's egg, accompanied by moderate heat and itching, but the discomfort is not sufficient to cause the patient to interrupt treatment. Goodale has only had two cases of anaphylactic shock, i.e. faintness, nausea, and vomiting about half an hour after the administration of the extract. After the course of injections had been stopped most of the patients showed a distinct diminution in the intensity of the skin disturbance, caused by rubbing pollen extract into the scratch. Cases that only present themselves for treatment after the attacks of hay fever have begun are given minute doses of pollen extract on four successive days, and subsequent doses at intervals of from three to five days.

Results of Treatment by Active Immunisation.—Freeman reports 30 per cent. of cures, and 35 per cent. almost completely cured; Cooke 60 per cent. markedly improved, 30 per cent. improved, and 10 per cent. of failures. Oppenheimer and Gottlieb have treated fifty-two cases, of which fifteen were cured, twenty-five markedly improved, and twelve in no way modified. Goodale states that out of thirty-four cases, twenty-six were more or less relieved. Among twenty-one hay fever cases Wilson had only one cure and three markedly improved. Goodale concludes that pollen therapy in hay fever may be regarded at the present time
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as a promising method of treatment, but its value and the permanence of its results remain still to be definitely established.

II. INGESTION ANAPHYLAXIS AFFECTING THE RESPIRATORY TRACT.

Goodale states that an individual sensitised to a given proteid may exhibit a characteristic reaction if the proteid is brought in a soluble form in sufficient concentration into contact with a scratch or abrasion of the skin. The skin of the flexor surface of the forearm is first cleansed with soap and water, and then with alcohol. In the case of very sensitive individuals it is not necessary to scratch the skin, but in less sensitive cases the epidermis is scarified, care being taken to draw as little blood as possible. The test material, e.g. the white of a fresh egg, is then rubbed gently into one scarification while a second one is left for a control. After five to fifteen minutes the positive reactions are indicated by varying degrees of local disturbance. In some cases the first perceptible alteration consists in a sharply circumscribed white area, not elevated, bordering the scratch for a distance of 1 to 3 mm. In others the first manifestation consists of a slightly red and raised area. In more pronounced disturbances the area of swelling is more extensive, white in colour, but surrounded by hyperemia of varying size. This degree is usually accompanied by itching. In the course of thirty minutes to one hour the reaction fades. In the case of certain proteids on the following day the borders of the scratch become red, elevated, and firm, and later a trace of suppuration may be seen. These disturbances are not due to sepsis, and disappear in two or three days.

In many cases the special exciting proteid is indicated by the patient's history, e.g. intolerance to fish, milk, horses, eggs, etc. In the larger number, however, the causative agent is unknown to the patient.

Preparation of Material.—It is of importance to have the test material in the form in which it exists when exciting symptoms, e.g. raw or cooked. The material should be in a stable form, and as pure and concentrated as possible. One person may react to a proteid dilution of a given strength, while another sensitised to the same proteid may require 100 times greater strength of the solution to elicit an equivalent skin reaction. Extracts from epidermal structures are easily obtained by placing scrapings from the skin, etc., in 12 per cent. alcohol. The clear filtrates keep without deterioration. In the case of animal foods, crude but satisfactory material is readily prepared by expressing the juices
in a meat press, and subjecting them to rapid desiccation by a blower. Both animal and plant extracts may be preserved when thoroughly dried without deterioration, e.g. egg-albumen, casein, pollen, etc.

Talbot has studied eleven cases of egg-poisoning in children. Six of the eleven cases had attacks of asthma in addition to the usual symptoms of egg-poisoning—vomiting, diarrhoea, urticaria, and eczema. The skin reaction was tested in five of the cases and found to be positive. By giving minute doses of ovomucoid by the mouth Talbot was able to produce immunity and so cure the asthma in three cases. In one he had partial success, but failed in the other two. Talbot has found that beef-juice may produce the same condition as egg-albumen.

It would take too long to go into the conflicting theories as to the pathology of asthma—(a) spasmodic, and (b) congestive. Those interested in this subject should refer to the letters by Gibson, Watson Williams, Campbell, Hare and Adam, which appeared in the British Medical Journal in the latter part of 1911 and the beginning of 1912.

Auld holds that asthma is a reaction on the part of the lungs to a toxic substance either of pathological origin or else a product of normal metabolism which gradually accumulates in the blood. Auer and Lewis have shown that the lung of a guinea-pig in anaphylaxis presents conditions identical with those of bronchial asthma. The protein poison which causes the asthma is still unknown, but Auld thinks that peptone is nearly akin to the actual anaphylactic protein poison. This has been shown by Biedl and Kraus. In the treatment of asthma Auld advises the subcutaneous injection of a watery solution of peptone (Armour). He uses ½ grm. dissolved in 5 c.c. of distilled water at blood-heat, and injects this solution at intervals of three or four days during the first week of treatment. In the second week he gives two injections of ⅓ grm., and in the third week two injections of 1 grm. dissolved in 10 c.c. of water. In most cases this is enough, but some require further injections. There is no apparent constitutional reaction, and little or no local reaction. So far Auld’s results have been very promising.

III. ACUTE INFECTIOUS RHINITIS.

Hill says that “colds” are most common when the humidity of the atmosphere is great and the temperature variable. Men
living open-air lives are free from colds, e.g. sailors on long voyages and Arctic travellers. Thus exposure even to extreme cold by itself does not occasion "colds" or pneumonia. The health of the Navy at sea in war is better than in peace; the sailors are far less exposed to temptation to over-indulgence and to infection from the civilian population in port. When children, after the holidays, return to school, epidemics of "colds" usually begin. Propinquity is required in order that the infected spray of nasal or bronchial secretion sneezed or coughed out may be transferred from the "carrier" to another victim. Cool breezes blowing round the head, the radiant heat of the sun, and a warm ground to stand on are the ideal outdoor conditions. The mucous membrane of the nose in these circumstances appears pale and taut. Indoors, when the feet are chilled by a draught blowing over a cold floor, and the head is immersed in warm stagnant air, the nasal membrane appears swollen, congested, and covered with thick secretion. This causes a feeling of stuffiness in the head, and headache felt in crowded, over heated places of assembly. A room with a gas fire fitted with no flue, so that all the heated air escapes into the room and rises, provides exceedingly bad conditions. Inhaled bacteria are caught by the swollen mucous membrane, which is covered with thick secretion. The ideal method of warming and ventilating rooms would give us abundant radiant heat, a warm floor, and agreeable movements of cool air. Such conditions are found in one-storied foundries.

Cocks holds that the bad effects of poor ventilation are due to the inability of the body to cool itself because of the increased temperature and moisture of the surrounding air. The capacity for heat regulation depends largely upon the vasomotor system and the sweat glands. The amount of heat lost from the surface of the body by radiation and conduction depends upon the temperature of the surrounding air, the amount of heat lost by evaporation upon the humidity. Resistance to infection is influenced by cold. Pasteur found that, although the common fowl is not susceptible to anthrax, it becomes susceptible when made to stand overnight with its feet in cold water. Rabbits and guinea-pigs chilled in various ways are much more susceptible to inoculation with bacteria than control animals. Vasomotor contraction of the skin vessels due to cold is accomplished by reflex dilatation of vessels in other parts of the body. Severe muscular exertion in hot bad air leads to active dilatation of the vessels of the respiratory mucous membrane, and if there now
follows a sudden exposure to cold a condition predisposing to catarrhal inflammation is produced.

Cocks⁵ has experimented with two rooms so arranged that any desired degree of temperature and humidity could be secured. He considers the normal temperature of a room (U.S.A.) to be 68° F., normal relative humidity 50 per cent.; cold room, temperature 50° F., relative humidity 50; hot dry room 80° to 86° F., relative humidity 20 to 30; hot moist room 80° to 86° F., relative humidity 80. He found that a large proportion of workers in hot moist rooms (steam laundries) suffered from a shrunken condition of the nasal mucosa. In passing from a normal or a cold room into a hot room there usually results an increase in colour, moisture, and size of the inferior turbinates and of the nasal mucosa generally. Conversely, on going from a hot or normal room into a cold one, there is a decrease. A second series of experiments was made to demonstrate the effect of a current of air blown directly upon the face, i.e. to obtain information concerning the effects of draughts on the nasal mucosa. On going from a hot dry room to a cold room in which a draught was created there was an increase in the size and moisture of the nasal mucous membrane. As a result of his experiments Cocks concludes that the theory of bacterial infection as the sole cause of catarrhal inflammation of the upper air-passages is not tenable.

Those who regard acute infectious rhinitis as a sensitisation of the mucous membrane of the upper respiratory tract to a certain organism, or organisms, suggest that the skin reaction of a given patient should be tested as follows:—The skin of the flexor surface of the forearm is prepared as described above, and then scarified as an ordinary vaccination. Bacterial test material is made by growing cultures on agar slants, exposing them to ether vapour for four days, and transferring the colonies to a 12 per cent. dilution of alcohol in water. The quantity of bacteria should be about one-third that of the dilute alcohol. The test material is rubbed in to the scarification and the result noted.

It would take too long to deal with the treatment of acute infectious rhinitis by means of vaccines. Those interested in this subject should consult the paper by Logan Turner and Hay Bolton (Med. Press and Circular, 1913, ii. 583).

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