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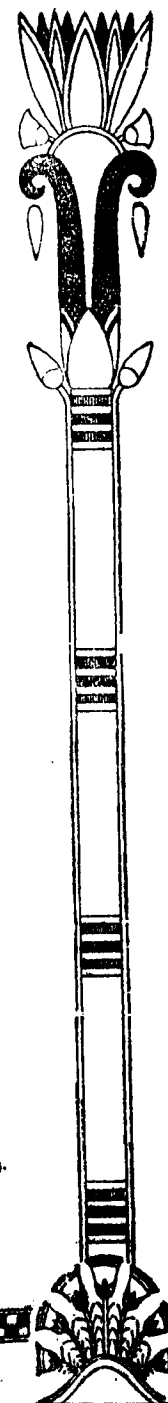
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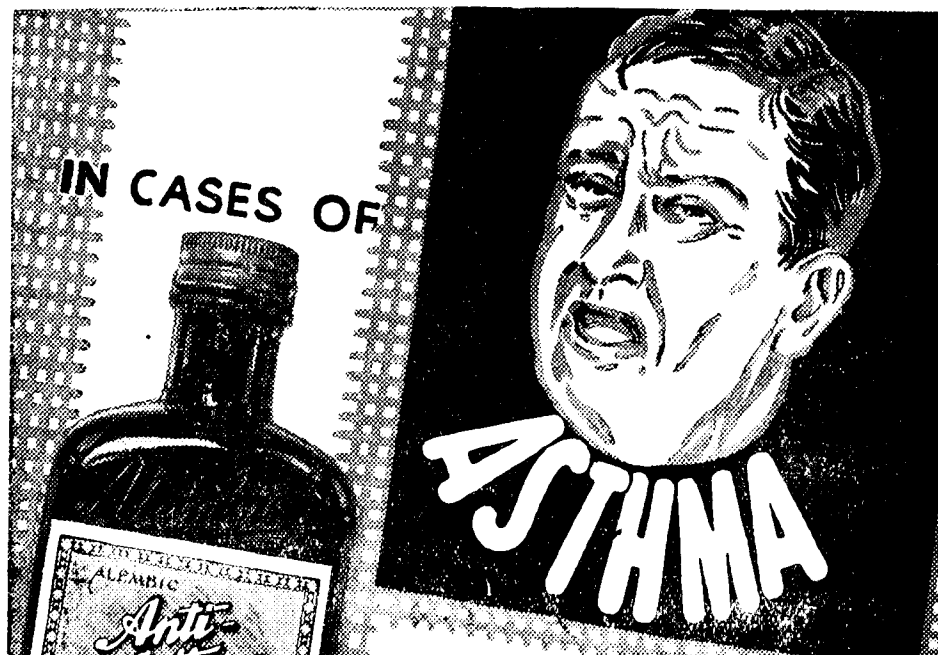
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VOL. IX

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No. 11

CONTENTS

ARTICLES—	PAGE.
The Significance of Hoarseness—By Walter Wells, M.D., and Washington, D.C. ...	319
Medical Treatment of Idiopathic Epilepsy—By Jyoti Dhar, B. Sc., M.B. ...	331
EDITORIAL Medical Associations of India ...	341
EXTRACTS ...	344

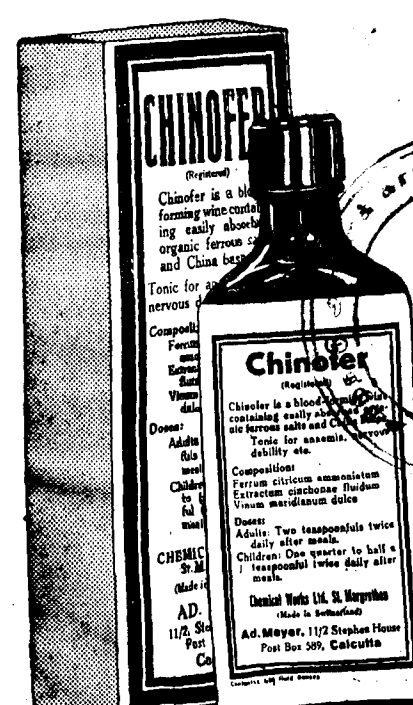
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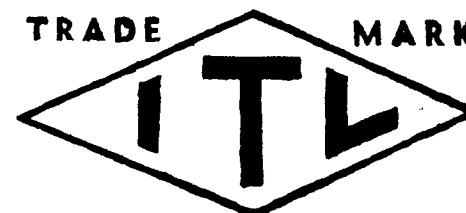
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in and out movement of the cords is made possible by an extraordinary arrangement of the arytenoid cartilages to which they are posteriorly attached. These are two small pyramidal-shaped cartilages, the bases of which form a capsulated articulation with the cricoid and in such a way as to permit a combined in and out sliding and at the same time a rotating movement about a vertical axis. To the anterior angles of the base of the little arytenoid the vocal cords are attached, and to the outer angles the minute muscles which run forward and backward to the cricoid cartilage just below.

When the backward running muscle (crico-arytenoideus posterior) is contracted, the effect is to cause an outward swing of the front angle to which the cords are attached, that is, abduction or opening of the glottis; when on the other hand, the forward running muscle (crico-arytenoideus lateralis) contracts the effect is to draw these points inward, thus bringing about the closing of the glottis by abduction of the cords.

While there is only the one muscle mentioned which serves the respiratory function by opening the cords there are several which, by bringing the cords together and making them tense, serve the functions of voice production. All these except one are supplied by the same nerve—the recurrent branch of the vagus.

There is one, the action of which anyone can verify by placing the fingers against the larynx in the interval between the cricoid and thyroid cartilages, while intoning notes of varied pitch. This is the crico-thyroid and it is supplied by the superior laryngeal nerve. When a high note is sounded you can feel the cricoid cartilage drawn upward against the thyroid, putting the cords on a stretch and increasing their tension, and by following with a low note an octave below, you can feel the cricoid dropping back into a low position, which relaxes the cords.

Very important for vocalization is the internal tensor (thyro-arytenoideus internus) muscle, the fibres of which run parallel with the cord and form a part of it. In fact, the vocal cords may be considered as the ligaments belonging to this muscle, and thus directly responsive to its contractions in sounding different notes. It is this highly developed internal tensor that gives the wonderful flexibility of the voice possessed by great singers.

The cords are two elastic bands, about three-quarters of an inch long in males and about half an inch in females, having normally smooth glistening white surfaces and even edges. As a result of disease they undergo a decided change in color, form and contour. The color may vary from pink or bright red in acute, to a dirty or slate-grey in chronic inflammation. The surface may become roughened and the edges uneven from exudate, infiltration, pseudo-membrane, ulceration or new growth, interfering with that nice approximation essential to the production of clear tones. Hoarseness

can occur, however, even with normal appearing cords, as when the approximation becomes impossible because of a paralysis or by a growth which gets between them.

Above the true vocal cords and somewhat external, lies the so-called false cords. They are not normally intended for voice production, but do exceptionally come into play in certain kinds of unnatural shouting, or in case of paralysis of the cord or their destruction by ulceration. The effect is a disagreeable raucous voice, produced by obvious great effort.

A fairly brisk current of air from the lungs is needed to throw the vocal cords into vibration; there is, therefore, something in the general view that one must have good lung power to have a strong voice.

The cords can vibrate only when they are in more or less close apposition, these vibrations causing the stream of air to be chopped in a succession of pulsations which give rise to sound. When the cords are very tense and near together, the vibration frequency rises, producing high pitched tones. When they are but loosely together, the tone is low pitched. The whisper is the result when the current of air passes between widely open cords, and is then whipped into speech by tongue, lips and teeth.

Though a whisper contains the essential elements of speech, it is lacking in the tonal qualities of the vibrating cord. It is, therefore, a sad substitute for vocal speech. For not only does it lack the carrying power often required, but it lacks emphasis and inflection, and the color of the voiced note. The vocal cords must come into play if the voice is to be expressive. The basic hum so produced, with its attendant harmonies emphasized in the resonant cavities of the nose, mouth and throat, give to it emotional qualities and musical charm.

It is sometimes necessary to warn our public speaking patients that mere loudness of the voice is not all that is needed to make themselves heard and understood. Clearness and intelligibility of speech depends to a great extent upon good articulation and accurate production and vowel resonance.

With proper vocal action, one can speak with little effort, yet be heard, for one thus gets full benefit of resonance. In this connection we might refer to the widespread belief that the deep or base register voice is due to chest resonance. The real purpose of the lungs is to serve as a bellows, and from a consideration of their physical qualities it is probable that they act as absorbers of sound rather than as resonators. As one writer expressed it, there is no more reason to expect the chest to have resonance than a bellows packed with a damp sponge.

To recapitulate, a good speaking or singing voice depends in the first place upon a good formation and healthy condition of the

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MEDICO-SURGICAL SUGGESTIONS

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NOVEMBER 1940

NO. 11

THE SIGNIFICANCE OF HOARSENESS.

WALTER WELLS, M. D., WASHINGTON, D.C.

When a symptom is one that has a definite and comparatively restricted indication, it may just as properly be made the subject of a medical discussion as the disease in which the symptom occurs.

Hoarseness is such a symptom. Unlike general manifestations such as fever, pain, etc., which arise from many causes, hoarseness occurs only when one particular organ, the larynx, is affected, and its presence is a sure indication of laryngeal trouble.

Furthermore, it is a symptom which must be accorded very special importance, because it is often the sole one present, and that, too, in cases in which the disease is one of extreme gravity.

For the better understanding of the subject of this paper, let us briefly review the structure and function of the larynx, particularly as concerned in voice production.

For the larynx, we must bear in mind, is an organ of a double function. Primarily it was a part of the organ of respiration, and its sole function was to serve as a passageway for air to and from the lungs. But when speech was needed, nature, the great economist, instead of creating a completely new and separate organ, engrafted one upon the already existent respiratory tract which was admirably adapted for the purpose. At the top of the airway she set the vocal cords, and she utilized the lungs as a convenient bellows to throw them into vibration.

The larynx, considered as a musical instrument, may be classed as a double lipped reed of the obœ type. The vocal cords, or lips, are two narrow ligamentous bands, running horizontally from before backwards. In front they are fixed close together at the center of the receding angle of the thyroid; posteriorly they are moveable, in a horizontal plane. When fully open for inspiration, they form two sides of a triangular space called the glottis; when brought together for the purpose of phonation they meet evenly in the midline. The

cords; second, upon a good development of the muscles that activate the cords; third, upon adequate lung power; fourth, upon a favorable development of the resonance cavities of the nose, mouth and throat; and fifth, upon proper methods of articulation.

The symptoms of hoarseness, though influenced more or less by all the factors of voice production, is essentially a trouble of the vocal cords. In former days when it was not possible to see the interior of the larynx during life, diseases of the vocal cords had to be diagnosed entirely by inference. Now, thanks to the arts of indirect and direct laryngoscopy, we can get a satisfactory view of the parts which are the seat of the disease.

Let us remind you at this point that practically all illustrations of the larynx by the indirect method are really of the image as seen reflected in the laryngoscopic mirror. As this differs from a view you would have by looking directly down upon the top of the larynx, you should know in what respect the difference exists. Remember that the image, and the illustration of it, is a view of the top of the larynx of the individual facing the observer. Therefore, the image as it appears in the mirror will show the epiglottis which is actually to the front as though it were to the back and farther away, and the arytenoids which are actually posterior, appear in front. The image in the mirror, you should know also, gives an illusory idea of the relative position of the true and false cords. They seem to be on the same level, whereas actually the true cords lie an inch below the false. The fore-shortening effect of the mirror furthermore gives a somewhat incorrect idea of the size of a growth situated on or about the cords, making it appear smaller or less extensive than it really is.

As hoarseness is a form of dysphonia due to an alteration involving the vocal cords, this alteration can for the most part be readily made out by laryngoscopic examination.

Leaving aside a few very rare diseases, we may briefly consider the following conditions in which it chiefly occurs: (1) Voice strain; (2) acute catarrhal laryngitis; (3) chronic catarrhal laryngitis; (4) acute edematous laryngitis; (5) acute spasmodic laryngitis (false croup); (6) diphtheria (true croup); (7) tuberculous diseases of the larynx; (8) syphilitic diseases of the larynx; (9) benign growths; (10) malignant growths.

Voice Strain: Hoarseness can develop as well from improper use as from excessive use of the voice. When it occurs suddenly after unusual exertion in the absence of other cause, we may justly say that it is due to strain.

Continuous improper use may not always be so evident, but should be suspected in those who complain of voice fatigue following singing exercises, or who speak in an unnatural cramped method, or who have poor resonance, and articulate indistinctly.

Objective changes are not always discoverable by the laryngoscopic examination. Violent strain may cause slight hemorrhages, usually shown as a red or later a brownish-yellow spot on the cord. In a chronically strained voice, we often note an elliptical space between the cords on abduction. This is due to paresis of the internal tensor muscle of the cord.

Singer's Node: A peculiar picture is sometimes seen in the larynx consisting in the formation of small nodules on the edges of the vocal cords, which are directly traceable to improper use of the voice. They are small, rounded and uniform, partially translucent, projections, often bilateral; found nearly always at the point of juncture of the anterior and middle third of the cord. They occur in singers who have been using improper methods, as for example, singing in a register beyond normal power, and producing tones in a way usually referred to as squeezing the voice. They seldom occur in those who sing regularly in the low registers, but mostly in tenors and sopranos.

They occur also in those who use speaking voice badly and to excess, as teachers, who teach in dusty rooms, public speakers, who speak in the open, or individuals who are compelled to talk a great deal to deaf persons who do not use hearing aids.

Pachydermia Laryngitis: This is also a condition in which abuse of the voice is considered as a chief cause, for it is most often found in street-hawkers, show barkers and auctioneers. It is marked by the heaping-up of the tissue at the posterior extremity of the glottis, in the inter-arytenoid space and on the vocal processes.

It is due to excessive proliferation of pavement epithelium and multiplication of papillae. A thickening of the one cord is often accompanied by a crater-like depression on the corresponding location of the other cord. These patients usually have a husky, deep voice, a disagreeable sensation and a constant desire to clear the throat.

Laryngitis, Acute: There are persons in whom every cold has a tendency to attack the larynx. The voice becomes husky, with a frequent desire to clear the throat, and sometimes slight tenderness in the region of the larynx. Expectoration is generally scanty, but marked by the expulsion of small pearls or pellets of mucus. Examination of the larynx will show the laryngeal mucous membrane congested, and the cords normally white will appear pink, red or scarlet, and may be covered with shreds of mucus.

Laryngitis, Chronic: Repeated attacks of acute laryngitis eventuate in the chronic form. Even without the acute attacks, it is common in those who breathe habitually through the mouth, or who are exposed to dust, smoke, or irritating fumes, also in smokers who inhale, drinkers who use to excess strong alcoholic drinks, and in those subject to suppurative diseases of the sinuses. Mouth

breathers will be discovered by a history of huskiness with tendency to increased clearing of the throat, particularly marked in the morning. In chronic laryngitis, there is a tendency to colds, accompanied by hoarseness. The cords will appear dull red, or dirty-grey, sometimes thickened and covered by sticky secretions.

Acute Edematous Laryngitis: This is an acute inflammation of the larynx, attended with edematous swelling of the membrane. When the swelling encroaches upon the glottis, difficult breathing is added to the hoarseness. In severe cases there is marked soreness of the larynx and painful swallowing. The condition is due to some virulent organism, generally the streptococcus. It is often preceded by a suppurative condition of the tonsil and peritonsillar tissue. Talking is painful, the voice is generally extremely hoarse, or may be aphonic. Examination shows the membrane swollen, partially translucent, sometimes red or purple. The swelling affects the epiglottis, arytenoid, or ary-epiglottic folds sometimes to an extent to render the anatomical landmark unrecognizable.

Acute Spasmodic Laryngitis (False Croup): This is a form of acute laryngitis peculiar to children. The little patient, hoarse during the day, wakes suddenly at night with paroxysms of difficult breathing and cough, the cough being characterized by a brassy ring. The attacks are repeated successive nights with comparative freedom during the day.

Laryngoscopy is not usually possible, but if it were, an edematous condition would be seen in the region below as well as above the cords.

Diphtheria (True Croup): When diphtheria involves the larynx, we have what is generally denominated true croup.

The hoarseness and difficult breathing are due to the location of the pseudo-membrane in the larynx. The symptoms are present day as well as night, and unless checked by antitoxin, the dyspnea becomes gradually worse, accompanied by inspiratory stridor and chest retraction, cyanosis and asphyxia.

Although laryngoscopy is not generally used, it can be in a certain proportion of cases. I have had occasion thus demonstrate the characteristic membrane in a number of cases in which none was visible in the upper throat thus clinching the diagnosis before the culture report could be obtained.

Tuberculosis of the Larynx: The question of whether or not laryngeal tuberculosis is ever primary we would leave to those who take delight in academical discussion; the important practical fact is that we do sometimes have it, and in outspoken form, in patients in whom the pulmonary lesion is slight or undiscoverable.

We should think of its possible presence in any anemic, emaciated patient, who continues to cough or has frequent attacks of hoarseness. The dysphonia varies with location, character and extent

of the lesion, but the voice is generally described as being weak, muffled, veiled or toneless. In the advanced stage, there will be pain in swallowing, often referred to the ear. The pain is quite marked in the swallowing of liquids, which is the point of difference from the dysphagia of cancer, which is more marked in the swallowing of solid food.

The laryngeal picture in an early stage may show nothing more than an abnormally pale mucous membrane, with perhaps a little swelling in the inter-arytenoid region. Later you will see evidence of infiltration of the arytenoids with papillary vegetation in the intervening area. When the ulcerative stage is reached, the cords will be involved and appear red and irregularly serrated.

Syphilis of the Larynx: Syphilitic lesions, known to appear in such various forms and in so many parts of the body, do not escape the larynx. And when the cords are affected, as they are very apt to be, a hoarseness develops, regarded by some observers as rather characteristic. It is therefore called *raucedo syphilitica*. The voice is harsh, raucous and presents a striking contrast with the voice of the tubercular, which, as we started, is generally weak, muffled but painful.

It is characteristic in general of syphilitic diseases of the larynx that it is relatively painless as compared with other serious conditions as for example, tuberculosis or cancer.

The form in which it most frequently occurs is the gummata. These are seen as smooth, round, reddish-yellow swellings. When they break down, ulceration follows, characterized by the deep, punched-out appearance, sharp edges, and surfaces covered by dirty looking pus or slough.

Benign Growth: Several kinds of benign growths occur in the larynx which may be responsible for a persistent hoarseness. The location of a growth has much to do with the occurrence of this symptom. A very small growth on the edge of the cords will produce considerable hoarseness, whereas a growth of greater size located elsewhere may be present for a long time without noticeable alteration in the voice.

Papilloma is the most common form of benign growth, especially in children in whom it develops to a size that will cause marked obstruction of breathing. It appears as a warty growth, pale pink in color, assuming generally a mulberry-like form.

The benign growths, named in order of their frequency, are: fibromata, myxomata, lipomata, chondromata. Cysts also occur, located generally in the epiglottis.

Cancer of the Larynx: The importance of hoarseness as a symptom cannot be better exemplified than in its occurrence in cancer of the larynx, for in this serious disease it is sometimes, in the early stages the only symptom present, and cancer is, of course, a disease in which early diagnosis is indeed of the utmost importance.

It is much more common in men than in women, and it is on this account that persistent hoarseness in elderly men may be very significant. The reason that hoarseness appears early is because laryngeal cancer of the intrinsic type has a predilection for the vocal cords, and because when a cord is thus involved, even though very slightly, infiltration of the underlying tissue affects its mobility. The reason that such a condition may persist quite a long time without other symptoms is due to the fact that cancer in this location shows little tendency to spread, owing to the isolated character of the intrinsic lymphatic circulation of the larynx.

On laryngoscopic examination we may see nothing more than a small greyish warty growth, or a small diffuse swollen area on the cord; but to the laryngologist, an appearance of this kind confined to one cord is very significant, more so if it is noted that the cord lags in movement as compared to its fellow of the opposite side.

A more typical form of epithelioma in the still early stage is a greyish fringe-like growth along the border of one of the cords. Later, when the neoplasm has broken down and the process extended, the diagnosis is unmistakable. The mirror now shows the presence of an irregular, sloughing growth; there is odor to the breath, intense pain in swallowing, emaciation and cachexia.

Laryngeal Paralysis: We might naturally expect the voice to be affected in cases of paralysis of muscles which serve the purpose of phonation. If, however, there is a complete paralysis of the abductors affecting both cords, instead of hoarseness there will be aphonia, because the cords cannot be approximated in a way to produce vibrations.

This is what we see in the hysterical form of aphonia, such as may occur following shock or emotional causes when the voice is suddenly reduced to a whisper, and later just as suddenly reappears.

When the external tensor, the one contraction of which can be felt on the front of the throat, is out of commission, we will see on the laryngoscopic mirror, as the patient attempts to phonate, a wave-like outline of the cords instead of a straight line. The voice is weak, feeble and easily tired.

The internal tensors, those directly connected with the cords, may be independently paralyzed as a result of local infiltration or severe acute inflammation or by voice strain. The voice in such a case is weak and toneless, and speaking is marked by so-called "phonetic waste," that is, escape of air between the insufficiently closed glottis. In the laryngoscopic mirror the picture is typical. In attempts at phonation the glottis takes the form of a narrow elliptical space.

Hoarseness may, also, be a symptom of a one-sided paralysis of the recurrent laryngeal nerve. This is not usually noted in the early

stage but later on, when the paralyzed cord has assumed the cadaveric position and the sound cord attempts to compensate by passing over the midline to approximate it in the act of phonation.

This is early recognized in the laryngeal mirror, when it is noticed that the paralyzed cord does not move with its fellow, and that on phonation you get an oblique instead of a straight midline between the cords.

In the foregoing discussion, we have assumed laryngoscopic examination as a matter of course. There are however, cases in which laryngoscopic examination is not possible, as in children and adults with very irritable throats, and in other situations in which for one reason or another it is not immediately available. In spite of its absence, however, it may be worth our while to attempt to draw some inference based on the occurrence of the hoarseness which even in itself may sometimes afford valuable diagnostic data. For example, in an adult the development of a marked hoarseness in connection with an acute inflammation of the respiratory tract (an acute cold), permits us to infer with reasonable certainty the existence of an acute laryngitis.

In such a case, if in addition to a severe hoarseness there develops a difficulty in breathing, with soreness in the laryngeal region and painful swallowing, we are justified in assuming that the acute laryngitis is of an edematous nature, and there is reason for growing alarm because of the dangerous tendency to dyspnea. In a child, hoarseness in connection with an acute catarrhal condition of the upper air passages, accompanied by difficult breathing, may mean either diphtheria or spasmodic laryngitis. If the difficult breathing grows, day and night, gradually worse, it is probably diphtheria; if the symptoms subside during the day and return in paroxysmal form during the night it is probably spasmodic laryngitis, so-called false croup.

In a child who has hoarseness and difficult breathing unassociated with a cold and of long duration, we may rightly suspect the presence of laryngeal polypus, the most frequent benign growth in the young and one which sometimes increases rapidly in size to the point of obstructing breathing.

In young adults, say between the ages of 20 and 30, the presence of persistent and recurrent hoarseness, should make us think of a laryngeal tuberculosis. Especially would this be so if the individual is anemic and has a chronic cough.

Persistent hoarseness in persons beyond the age of 40 should always arouse suspicion of malignancy, at least if not due to evident mouth breathing or excessive alcoholism.

The character of the hoarseness differs and sometimes is quite characteristic. In laryngeal tuberculosis the voice may be described as weak, veiled or muffled, and painful with much use; in syphilitic

laryngitis it is harsh and raucous, but painless. In bilateral tensor muscle paralysis it is hollow or toneless and easily tired; in unilateral cord paralysis it is unnaturally high pitched and easily tired.

From the standpoint of the laryngologist, diagnostic discussion of a laryngeal disease based solely on subjective symptoms is purely speculative, because the diagnosis cannot be considered as complete without a laryngoscopic examination.

However, speculative considerations are not without their value. They originate suggestions which tend to narrow the field of observation and to concentrate attention where attention particularly is needed.

The smart oil prospector looks for oil only when the right geological formation indicates its probable presence. Successful medicine depends in great measure on a knowledge of what to look for and where to look for it.

Doctors need to be reminded repeatedly of the indications of symptoms. Otherwise one is apt to hear too often such sad exclamations of disappointment and defect as, "Oh, I never thought of that." A fuller knowledge of the meaning of hoarseness would no doubt more frequently lead to the discovery of its cause.

The prognosis will naturally depend upon the underlying cause, and in the case of the grave infections or malignancies resolves itself into a question of the general as well as local extent to the disease.

When the hoarseness is the symptom of a self-limited acute disease such as acute laryngitis, we may expect to see it clear up as soon as the disease runs its course.

In case in which it can be rightly attributed to excessive or improper use of the voice, improvement will be in proportion to our success in restraining the excess or correcting the abuse.

When the hoarseness is due to a growth of any kind which prevents normal approximation of the cords, the prognosis for the voice is bad if the condition is not corrected, because of the extra strain put upon the laryngeal muscles in such case, in the effort to overcome the handicap. In the case, therefore, of singer's nodules due to voice strain, we have a real example of the so-called vicious circle. For the very thing that caused the trouble is in turn caused by it.

There is one kind of hoarseness that generally improves with time, that is, when it is due to vocal cord paralysis of one side; for after the paralyzed cord has finally settled down to a fixed position its immobility will be partially compensated by the overaction of the muscles of the other cord.

The hoarseness that we are all most frequently called on to treat is that due to an attack of acute laryngitis.

Outside of the general measures we adopt in treating any cold, there are some special indications for a cold in this location.

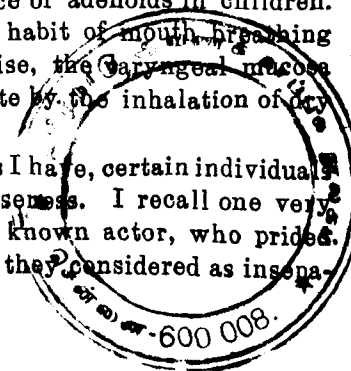
We believe in ice compresses, applied to the outside of the larynx, used within the first 48 hours, but not after that. During this early stage when the membranes are very sensitive we would not try to reach the interior of the larynx by astringent sprays or swabs. Later on mild sprays may be used, but none containing silver nitrate which is irritating to the larynx, and often produces severe, glottic spasms. Soothing sprays may be used, and steam inhalation containing benzoin, menthol and eucalyptol.

In acute laryngitis the prohibitions are more important than anything else. Smoking should be forbidden, especially inhaling. The patient should even avoid a room in which others are smoking, and any dust or fume laden atmosphere. It is important to avoid mouth breathing, and most important of all to avoid using the voice. This last is sometimes very difficult to enforce in the case of persons whose living depends upon their speaking or singing, and yet it is for these that it is particularly important.

A patient who is scheduled to give a lecture, or to go on in some theatrical act, will sometimes come to you and demand that you give him some spray or other treatment that will enable him to keep his engagement. Looking at the larynx, you see that the vocal cords are red and the whole laryngeal mucosa swollen and hyperemic. You know that to use the voice in such case is to increase and magnify the inflammation. You should, therefore, firmly forbid it, just as a surgeon would forbid the use of a tender, swollen joint in an acute arthritis. You should frankly tell the patient that there is no treatment, local or general, which under such circumstances will allow him to use his voice with impunity, and if he does, he runs the risk not only of prolonging the attack, but of becoming chronically hoarse. Chronic laryngitis and its attendant hoarseness is generally to be attributed to repeated and neglected acute attacks.

One should seek very carefully in such cases for a possible supporting cause in the condition of the nasal organ - as a suppurating sinus, with a constant draining of pus down the posterior wall of the pharynx in adults, and the presence of adenoids in children. Any nasal obstruction that induces the habit of mouth breathing should, of course, be corrected. Otherwise, the laryngeal mucosa will be kept constantly in an irritated state by the inhalation of dry and improperly prepared air.

Of course, you may meet sometimes, as I have, certain individuals who don't want to be cured of their hoarseness. I recall one very well known actress and one very well known actor, who prided themselves on their deep register, which they considered as inseparable from their stage personalities.



As a matter of fact, we have little doubt but that certain stage celebrities would in a measure lose their popularity if they lost their hoarse voices. Imagine the disappointment and disgust of the young gallery gods if Donald Duck were made to speak in clear musical tones. The gate receipts would, I fear, soon fall to the vanishing point.

Only a very few people have, however, a reason not to be cured of their hoarseness, for the hoarse voice is, generally speaking, a disagreeable voice; besides that it usually tires more easily than a normal voice, and grows worse with much usage. If it is done due to improper methods of voice production, the cure, of course, lies in the correction of those methods. This may require the individual to put himself or herself under the tutelage of a competent teacher. One can, in any case, do much for one's self by cultivating a natural manner of speaking. One must leave the throat relaxed, instead of cramping the muscles, and practice careful distinct articulation on all occasions.

Finally, in certain cases, the cure or improvement in the hoarseness is of importance as an indication of recovery from the disease causing it. If hoarseness is due to a growth of any kind it will never disappear until the growth has been in some way removed, either by direct or indirect laryngoscopy, or in the case of malignant diseases, it may be removed by an external operation.

We hardly need emphasize the vital importance of early removal of any malignant or partially malignant neoplasms. This is particularly true in the case of the larynx, because of the very distressful nature of the latter stages of malignancy in this situation.

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MEDICAL TREATMENT OF IDIOPATHIC EPILEPSY

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INTRODUCTION

Epilepsy is a symptom-complex which is characterized by 'periodic or irregularly recurring attacks of loss of consciousness, with or without convulsive seizures, the typical attacks being occasionally replaced by other manifestations of a nervous or physical nature, and though often associated with organic disease of the brain is not itself dependent on any recognisable specific structural changes in it' (Holmes, 1924). When epilepsy is not associated with any demonstrable disease it is termed *idiopathic*; and the treatment of this condition is dealt with in this paper.

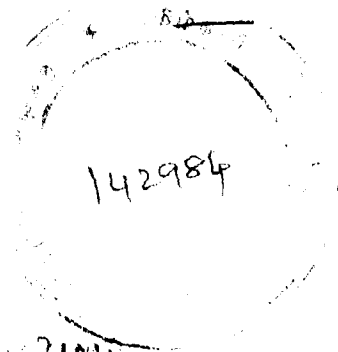
GENERAL MANAGEMENT

The General health of the epileptic patient should be kept as best as possible. Light and well regulated physical exercise and light study are beneficial. Constipation should be avoided; when present, small doses of cascara sagrada should be added to the bromide mixture. The patient's mode of life will require revision. The risk of deleterious effects of overexertion in exciting games and recreation should be remembered. Swimming, bathing, bicycling, riding, motoring, boating and working near a machinery are particularly hazardous and should be forbidden. Light and well regulated indoor exercises or walking in the morning and evening are permitted. Fire and water provide the main dangers to the epileptic.

All sources of irritation such as carious teeth, impacted wisdom teeth, septic tonsils, refractive errors, phimosis, intestinal worms, nasal polypi, etc. should be sought for and removed. Sometimes fits disappear temporarily in the course of exanthems and other fevers, e.g. measles, diphtheria, pneumonia, typhoid fever and scarlatina.

It is desirable that the sensitive mind of the epileptic should not harbour an inferiority complex. He should be made to feel as a normal individual. It is essential at the beginning to find out (1) any abnormality of mind or body which is acting as a precipitant, and (2) any combination of circumstances which seems reasonable for individual fits. The immediate excitant, which seems to bring the fits on, may be injudicious feeding, violent exertion, fatigue, constipation the keeping of unusual hours, recurring anxiety, emotional strain, etc. The above may suggest the proper lines of future treatment.

Regarding the question of education and vocation of epileptics, it should be remembered that some of these patients are hopelessly 'ineducable and unemployable'. Many are suitable for graded occupations. The great majority are suitable for light mental exercise



and less onerous employment. There should be no 'bossing' over an epileptic employee.

Regarding the question of *marriage* of epileptics, it is well known that the risk of epilepsy in the offspring of normal (i. e. free from all taint or evidence of epilepsy) parents is from one to three per thousand. But where one of the parents is an epileptic, the chances of development of the disease in the child is ten times as great (Wilson). Therefore, marriage or rather parenthood in such subjects is inadvisable. At the same time it should be remembered that 'acquired characters do not reappear in a succeeding generation.' Sexual excess is to be avoided.

THE DIET

The diet should be bland, simple, easily digestible and nourishing. The quantity of any meal should not be excessive, nor should the meal be eaten hurriedly. Although there is no clinical or experimental evidence to prove the deleterious effect of proteins in epileptics, meat is empirically excluded from the dietary. Common salt is thought to augment nerve-cell excitability. A salt-free diet is, therefore, recommended (Richet and Toulouse). Such a diet lowers the chloride concentration in the blood and helps in diminishing the frequency of fits. Alcohol is forbidden. Light smoking may be allowed.

Periodic *fasting* is said to have a striking though temporary value. How this is brought about is not definitely known. *Ketogenic* or acid-producing diet with restricted drinks is on the whole disappointing, 'though occasionally worthy of trial in case of children and in minor epilepsy'. In children ketosis can be readily induced on a low ratio between ketogenic (fat) and anti-ketogenic carbohydrate and protein food stuffs, a 3 to 1, or 4 to 1 ratio being sufficient. In adults it is more difficult (Wilson). *Dry diet*, which has been recently introduced, is not encouraging.

The principle of ketogenic *diet* is summarised by Paterson (1938) as follows: Try to give as much fatty food as possible, such as cream, butter, margarine, eggs, dripping, fat, meat and oils. Avoid giving much sugar or sweet jams, and reduce greatly all starchy foods such as porridge and other cereals, potatoes, bread and rice pudding: suet pudding may be given. Restrict *drinks* to not more than three half glasses of fluid in the day. This includes soup, milk, tea, water, coffee and cocoa. The proportion of *fats* must be adjusted according to the presence or absence of acetone in the urine. If fats are pressed too far vomiting occurs and the diet must then be stopped, very little fat given and carbohydrate offered freely.

MEDICAMENTS

The welfare of epileptic patients depends more on empirical treatment by anti-convulsant drugs than anything else. An effective

sedative is, therefore, the sheet anchor of treatment. Sedative treatment by drugs aims at symptomatic control of the fits. The bromides and phenobarbitone are the chief depressants used for the purpose. Recently a new drug, sodiumdiphenyl hydantionate (*synonym*: dilantin sodium, epanutin), has been introduced which has been acclaimed as the treatment of choice in epilepsy. It is essential that the depressant selected should be taken regularly and continuously for a period which may extend to several years. For, if even a single dose is omitted, a fit will often quickly follow. Sedatives so employed do not produce mental deterioration. The latter appears as a part of the disease. Even when the fits have been controlled, the particular drug should be continued steadily for as long as two years after the last attack. Thereafter, it should be gradually reduced. If the sedative treatment (with the anti-depressant drugs used in turn) fails to reduce the number of fits, the treatment should better be discontinued.

Epileptic fits may tend to occur at certain fixed hours of the day or night. In these cases a larger dose of the sedative selected should be given an hour or two before the attack is liable to occur. Epileptic fits may, in certain cases, occur near the time of menstruation. In these cases continuous administration of large doses of sedatives is not necessary; it should be given in large doses only during and for a few days preceding the menstruation.

1. THE BROMIDE

Bromide is the time honoured remedy for reducing or abolishing the tendency to fits. Clinical experience points to sodium or potassium bromide having superior merit over lithium or ammonium bromide. There is no advantage in combining the different bromides in a prescription. The *dosage* of the bromide varies with frequency and severity of fits and the age of the patient. The total daily dose rarely exceeds 60 grains. Good results generally follow a morning and evening dose of 25 grains. This dose is well borne. Inadequate single doses only dissipate the bromide effect; for example, 15 grains twice daily is preferable to 10 grains thrice daily. Commencing dose for children would be 5 to 10 grains, morning and evening.

It should be the aim of the practitioner to effect the maximum concentration of the bromide on that period (when known) of the twenty-four hours which is most likely to witness the attacks. For example, in nocturnal epilepsy a single dose at bedtime may be sufficient; for morning cases a single dose at night and another on waking (but before rising) should be recommended. Those cases respond best to bromide that best tolerate its long-continued use.

The bromide has cumulative effect, and when the total amount is too high, symptoms of bromism may develop. The initial symptoms of bromide intoxication are excitement or delirium, which

should not be mistaken as indication for increasing the dose of bromide. Cutaneous eruptions are obviated by prescribing arsenic in the bromide mixture as in the following prescription:

R/ Sodium or potassium bromide ... gr. 15
 Liq. arsenicalis ... m. 3
 Aqua chloroform to ... dr. 2 to 4
 Fiat mist.

One, thrice daily after food.

some patients tolerate bromide badly. Digitalis, when given with bromide, is sometimes attended with benefit distinctly in excess of that obtained from the latter salts alone, especially when there is any cardiac complication (Bramwell).

Regular and persistent administration of bromide is imperative and neglect is fatal. Wilson advises that the graded withdrawal should not be begun until two years have been passed in security. Once a good result is secured, the amount can be lessened by degrees. The total daily (and not the separate) dose should first of all be gradually reduced. For example, the bromide can be taken twice daily instead of three times: then once; then on alternate days and so on. It is better to err on the side of delayed reduction.

Many proprietary preparations containing bromide have been introduced in the market. Some of them are listed below: bromocarpine (potassium bromide and pilocarpine), brompin or brominol (bromide and sesame oil), bromalin (hexamine ethyl bromide), *Gelineau's dragees* (potassium bromide, antimony arsenate and picrotoxin), sedobrol (sodium bromide and sodium chloride), *Trench's remedy* (potassium and ammonium bromide), etc.

2. THE PHENOBARBITONE

(a) *Luminal* (phenyl-ethyl malonyl-urea) is an anti-epileptic with powerful hypnotic effect. Major epilepsy responds better to luminal, as also bromide, than minor form. Luminal, given alone, acts best in mild and moderate epilepsies. In more severe or chronic cases best results are obtained when it is combined with bromide (luminal sodium is, however, incompatible with ammonium bromide) or borax or other sedatives (Holmes, 1924). The drug has least value in cases recurring at long intervals. It does not cause mental depression, but tolerance with the drug is readily established. The effective dose of the drug is not far removed from the toxic dose. Rashes may appear readily. Vertigo, ataxia and restlessness or torpor and stupor may develop if the drug is pushed too far. Luminal is generally prescribed in tablet form. The maximum daily dose should not exceed 4 grains. Treatment is begun with $\frac{1}{2}$ grain of luminal sodium, morning and evening; increasing the dose, where necessary, from $\frac{3}{4}$ to 1, or $1\frac{1}{2}$ grain, one to three times daily. The commencing dose for children would be $\frac{1}{2}$ grain, morning and evening. Luminal sodium is a soluble salt.

(b) *Prominal* (N-methyl-ethylphenyl malonyl-urea) is preferred by some workers to luminal in the continuous treatment of epilepsy, even in severe cases with psychic change. It is an effective anti-epileptic without hypnotic effect. Even with continuous administration no by-effects appear. According to the severity of the case the daily dose varies from $1\frac{1}{2}$ to 9 grains. Usually 3 to 6 grains (one to two tablets) twice daily for an adult are sufficient.

3. SODIUM-DIPHENYL HYDANTIONATE

Sodium-diphenyl hydantionate (*synonym*: dilantin sodium, epanutin) has been introduced as dilantin sodium in America and as epanutin in Britain for the treatment of epileptics not responsive to other medications. It is not recommended for the treatment of epilepsy in which the seizures occur only at long intervals unless moderate doses of luminal or bromide are ineffective, or induce disagreeable side actions. It is particularly effective in grand mal seizures than those of petit mal type. The mode of action of the drug is uncertain. It is believed to be of the nature of an increased resistance to the convulsive discharge. The action of the drug is rapid and does not appear to be cumulative. The drug has relatively the greatest anti-convulsant and least sedative effects: and in this latter respect it is much superior to luminal (phenobarbitone). Its anti-convulsant effects are striking. There is practically complete absence of sedative effects with dilantin sodium (c.f. luminal, which while controlling the fits, has a soporific and almost stupifying effect). The great disadvantage of the drug is its toxicity.

Dosage: Dilantin sodium is issued by the manufacturers in the form of 0.1 gm. ($1\frac{1}{2}$ gr.) capsules. In all adults a minimum threshold dose of 0.3 gm. (or three capsules) per day seems to be necessary, regardless of how small a dose of sedative the patient was on before. Neglect to follow this direction will almost certainly lead to fits. In subjects who have nocturnal fits, one of the capsules should be given just before going to bed.

Merritt and Putnam (1938) recommend a starting dose in adults of 0.1 gm. (one capsule) three times a day. The dosage may be increased gradually to a total of 0.6 gm. (six capsules) daily, if necessary. The starting dose in children is 0.1 gm. (one capsule) twice daily. The subsequent dosage is adjusted according to the therapeutic response. For very young children (under four years of age) dosage can be conveniently started at low levels by giving 0.03 gm., twice daily. The bitter taste of the medicine can be partially covered by mixing it with cream. In all cases the optimum dosage of the drug must be determined by trial. According to these authors children above the age of six, and adults, be started on 0.1 gm. (one capsule) three times a day. Where the above dosage prove ineffective after one week's trial, a fourth dose of the drug (0.1 gm. or one capsule) should be given at bedtime, making the total

daily dose of 0.4 gm. (four capsules). Further increase up to a total daily dosage of 0.6 gm. (six capsules) may be made if necessary. All increase in dosage should be made at intervals of approximately one week. The *minimum effective* daily dose of the drug should be continued.

Dilantin sodium is more rapidly effective when given *before* meals, and it is advisable to give at least the first of the daily doses immediately on arising. The drug is strongly alkaline and should be taken with at least half a glassful of water to minimize gastric discomfort complained of by some patients. Where there is a tendency to gastric intolerance to the medication, it may be given with or immediately following meals.

The change over from previous sedatives to dilantin sodium should be effected by the *method of substitution* (Blair): If the patient is previously on three doses of sedatives, replace one dose (regardless of its quantity) by one capsule of dilantin sodium every alternate day. By the end of the sixth day, the patient will be on dilantin sodium alone. If the previous sedative is being given four times a day, withdraw the fourth dose on the eighth day without replacing it by a dilantin sodium capsule. The drug is not increased beyond one capsule, three times a day until after the fourteenth day, unless there is a marked increase in fits.

Toxic symptoms: Patients receiving the drug should be closely watched for toxic symptoms. The liability of toxic symptoms increase with dosage. Donald Blair (1939) has summarised the toxic symptoms of the drug as follows: The *nervous* symptoms may be dizziness, giddiness, blurring of vision, diplopia, clonic spasms, tremors, general muscular irritability, ataxia and nystagmus: The *mental* symptoms may be euphoria; excitement, agitation, irritability, confusion, delusions and hallucinations, dullness, depression, and reactivation of suicidal tendencies. *Skin* eruptions may be of a mild erythematous, scarlatiniform or morbiliform type. Sometimes these may be severe and accompanied with fever and very occasionally an exfoliative dermatitis. *Gastro-intestinal* symptoms may be nausea, anorexia, abdominal discomfort, constipation etc. There may be sponginess and tenderness of the gums. There may also be epistaxis. There may be slight *blood* changes, such as leucopenia, lymphocytosis, eosinophilia and an increase of hemoglobin percentage.

Treatment of toxic symptoms is very simple, namely, the immediate and complete withdrawal of the drug, without replacement by other anti-convulsants. If the toxic symptoms disappear within a few days, the treatment with the drug may be started again. The starting dose should preferably not exceed three capsules a day. If the patient was previously receiving more than three capsules a day re-introduction should always commence with *one less* capsule a day.

If excessive number of fits occur on the cessation of the drug, immediate resort should be made to intramuscular injection of luminal (phenobarbitone), followed by a return to the sedative used before dilantin sodium was started. As mentioned elsewhere, the gastro-intestinal symptoms are due to the alkalinity of the drug and are relieved by giving the drug with a drink of water during or just after a meal.

Blair mentions the following contra-indications against the return to dilantin sodium (after toxic symptoms) (1) those cases with exceptional severe toxic symptoms, and (2) those who, after being on the drug for a reasonable length of time, have fared no better regarding the number of fits than they did on previous medications.

4. OTHER DRUGS.

Borax and its compounds sometimes prove effective. Sodium baborate or *borax*, gr. 5 to 15, two or three times a day, is often prescribed, either alone or combined with the bromide mixture, or with luminal, for both major and minor epilepsy. Borates are valuable in major nocturnal epilepsy. Tincture *belladonna*, min. 5 to 15, two or three times a day, is prescribed with the bromide mixture for petit mal. *Zinc oxide*, gr. 3 to 5 in capsules, is given twice daily in petit mal. *Calcium lactate*, gr. 7 to 10, combined with bromide sometimes helps in checking seizures in children. *Dialacetin* has been given in nocturnal epilepsy. *Liquor trinitrini* (nitroglycerin) sometimes prove useful in petit mal and visceral fits.

TREATMENT OF AN ATTACK.

During a major seizure the patient should be protected from injuring himself. The clothing about the neck and chest should be loosened in order to prevent obstruction to respiration. The patient should be placed on his back. Any accident should be prevented as far as possible. Biting of the tongue should be prevented by inserting any suitable object, such as the handle of a spoon, between the teeth. Respiratory embarrassment from falling back of the tongue may prove fatal during the status epilepticus. This should be prevented. Should the tonic phase be unduly prolonged or should a second attack follow immediately upon the first, a few whiffs of chloroform, or an inhalation of amyl nitrite, may be administered. If vomiting follows a fit, the patient should be turned on his side, to prevent the risk of regurgitation of vomited matter into the respiratory passages. No stimulant is necessary. The patient should be allowed to go to sleep after a seizure. The severe headache which follows a fit is relieved by phenacetin (gr. 10). When fits are preceded by aura suitable measures should be undertaken to inhibit an attack. In Jacksonian epilepsy the limb may be firmly seized or ligature applied. When the aura and convulsions are of a general nature, smelling salts, or amyl nitrite inhalation may inhibit the

attack. Although alcohol is generally forbidden. Wilson has found it sometimes effective in warding off a fit.

STATUS EPILEPTICUS

The frequent recurrence of fits leading to status epilepticus is a grave but rare complications which may cause death of the patient. If this condition has followed omission of bromide or other sedatives, such sedatives are at once indicated. But if it has developed in spite of such medication, no benefit can be expected from massive increase. Inhalation of chloroform, or amyl nitrite may check the attack temporarily. Sometimes it can be averted by prescribing bromide and opiates together (Clark and Prout) as in the following prescription :

R/			
Tincture opii	...	...	m. 5
Potassium bromide	...	...	gr. 25
Chloral hydras	...	...	gr. 20
Liq. morphine sulph	...	...	m. 60

This is given by mouth or rectum, and repeated in two hours

Whitla (1938) recommends the following treatment for status epilepticus :

1. Examine the rectum with the finger and if found loaded with feces a large soap-and-water enema should be given. At the same time place the patient under chloroform or ether ; the heart and respirations being carefully watched for signs of failure. On regaining consciousness a mixture containing 30 gr. potassium bromide and 10 gr. chloral hydras or 10 min. tincture opium should be given by the mouth and the patient allowed to drink copious fluids.

2. If no improvement is observed and blood pressure is much raised, venesection should be performed. Lumbar puncture with free drainage may be tried.

3. If still no effect is obtained, give an injection into the rectum through a rubber catheter and funnel, of 4 to 6 drachms of paraldehyde made up in an equal quantity of olive oil or shaken up with 10 parts of water before injection. Avertin is more certain in its action and if obtainable is to be preferred, the dose being 0.06 to 0.1 gm. per kilo. of body weight.

4. If treatment advocated in para No. 3 fails or is not available, morphia and hyoscine should be resorted to in doses of gr. $\frac{1}{4}$ gr. 1/100 respectively, injected hypodermically and repeated if necessary within an hour or two.

Paraldehyde seldom fails to stop the fits. It is a very safe and effective way of treating the condition. Paraldehyde (six drachms) is mixed with an equal quantity of olive oil and given per rectum. Some clinicians dissolve each dose of paraldehyde in 6 oz. of normal saline. One ounce of paraldehyde can be given by the rectum in this way for the first dose, and thereafter 2 drachms of paraldehyde

in saline per rectum every half hour until the fits cease (Russell, 1939). Another method of treating the condition is to inject slowly into a vein a solution of 3 gr. of *luminal sodium*.

The most efficacious remedy in this connection, according to Wilson, is *somnifain* (2 c. c. by hypodermic injection). *Chloral hydras*, gr. 30, may be combined with *potassium bromide*, gr. 60, for rectal administration every four to six hours. Hypodermic injection of freshly prepared *luminal sodium* solution (gr. 1 to 3) and bromide (10 per cent) is sometimes recommended. Hypodermic injection of *hyoscine hydrobromide* (gr. 1/75 to 1/50), repeated within an hour or two, are also useful.

Oxygen inhalation is recommended for these patients. Venesection and injection of saline are recommended in robust individuals. Withdrawal of 10 c. c. of cerebrospinal fluid by lumbar puncture sometimes prove efficacious. Support to the heart may be obtained from strychnine, digitalis, alcohol etc. Diuretics and cathartics should also be prescribed. Plain nourishing liquids should be given by nasal tube from the beginning. Where possible, 25 per cent glucose solution should also be given intravenously.

(*Calcutta Medical Review*, Sept. 1940.)

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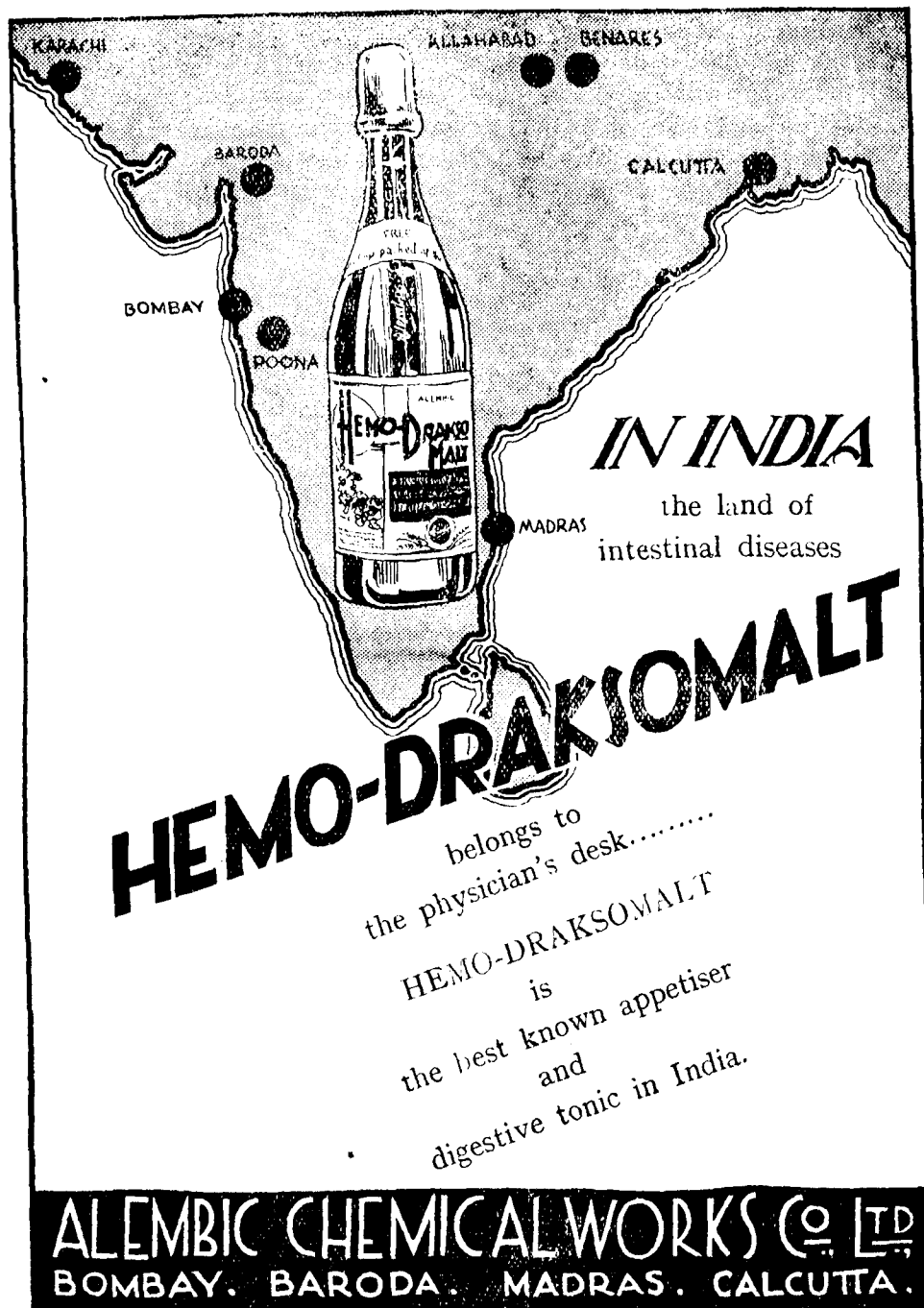
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MEDICAL ASSOCIATIONS IN INDIA.

It was but a few months ago that Dr. A. Viswanathan, the talented editor of *The Indian Medical Journal* made a brilliant analysis of the position of the various medical associations in this country, in the pages of his Journal and also in the *Journal of the Indian Medical Association*. If there is any country where a strong medical organisation is badly wanted and is inadequately organised, it is India.

The two organisations of an all-India character and having the most popular appeal are the All-India Medical Licentiates' Association and the Indian Medical Association. The former is of a longer standing, has a great deal of pioneering work to its credit, and has a very large membership. It holds its provincial and all-India conferences regularly and works for the betterment of one class of medical practitioners, namely, the medical licentiates. There is no gainsaying the fact that the medical licentiates are proud of their organisation, and are striving to improve themselves.

The Indian Medical Association is of a more recent origin and caters for all classes of medical practitioners—licentiates and graduates. It is an all-embracing organisation and works for the improvement of the medical profession as a whole. It does not distinguish one medical practitioner from another. It works for and speaks in the name of the entire medical profession in the country. It tries to attain a position in this country similar to that attained by the British Medical Association in Great Britain and the American Medical Association in the United States of America—associations whose opinions carry an enormous weight and who are frequently consulted by the state regarding medical affairs. The Indian Medical Association also works through local branch associations, a number of these local associations grouping together to form a provincial branch, and several provincial branches working under and directed by the central body of the Indian Medical Association. The provincial bodies as well as the All-India body meet annually in conference and discuss their problems, medical and medico-political. The Punjab, the United Provinces, Bihar and Bengal have each of them a provincial branch. Sind, N. W. Frontier Province, Assam,

Orissa, and Central Provinces have a few branches in some of the bigger towns. Hyderabad state has one branch. In the Madras Presidency. The Andhra districts have recently formed an Andhra provincial branch, and the other districts have recently followed suit and formed a South Indian Provincial branch. In the Bombay Presidency there is the Maharashtra and Karnatak provincial branch. One would like to see the entire Bombay Presidency moulded into one provincial branch so as to form a Gujarat—Maharashtra-Karnatak provincial branch, with probably the Marathi speaking areas of Berar and of the Central Provinces added on. We hope this suggestion will ere long be acted upon in the interests of better organisation of the medical profession in our sister Presidency. The function of these provincial branches is mainly to organise local branches within their area, to discuss and formulate medical policies, and to speak with one authoritative voice on behalf of the medical profession in the province. The time is not far distant when the Indian Medical Association will blossom out into the supreme body in India which counts in medical matters.

The British Medical Association has its branches in the big cities in this country and its membership is mostly taken up by the I. M. S. men and a few of their camp followers. This body is usually a bulwark of vested interests and usually supports all the extravagant claims and demands of the Indian Medical service. The demands made by the spokesmen of this service before the Lee commission several years ago and the reservation of the "cushy" jobs and the lucrative posts in this country to the I. M. S.—particularly to those recruited from outside India—had the support and backing of the British Medical Association.

There are some well-established and long-established medical associations in this country, some of which are of longer standing than the two all-India associations mentioned above. To name a few of them, they are the Bombay Medical Union, the Calcutta Medical Club, the Delhi Medical Association, and the South Indian Medical Union of Madras. These associations concentrate their activities more or less to the cities and have a splendid record of useful work to their credit. They have been slowly and carefully nurtured from small beginnings with all the tender care of a mother to her infant, by a handful of enthusiasts who gradually attracted increasing numbers of medical men and women into their midst, until they reached the pre-eminent position which some of them hold in the Indian medical world to day. Of late the spokesmen of the Indian Medical Association have been trying to get these bodies to merge themselves in their Association so that one powerful all-India organisation could spread its sheltering wings over smaller organisations in order that the medical profession all over the country could become a strong, well organised and well knit body. It is

very proper for every medical practitioner to join the Indian Medical Association, but it is rather hard for a long standing association working in a large city to lose its identity over night and merge itself in the larger all-India body. It is perfectly legitimate to enlist all the members of these local associations into the Indian Medical Association, while leaving their associations to maintain their separate individuality for their local purposes. It is worth mentioning in this connection that almost all the members of the Calcutta Medical Club are also members of the Calcutta branch of the Indian Medical Association.

The signs of the times indicate that the old adage "union is strength" holds good in medical associations, and small associations should unite and join a large association like the Indian Medical Association, and small groups of medical men should also unite under the aegis of the Indian Medical Association, which is, at present, the only organisation which could claim to represent the medical profession in this country.

It would indeed be a happy event and a red letter day in the medical history of India when these two all-India associations coalesce and work together as one joint association. The first steps towards this end would be to hold their annual conferences and scientific sessions together in the same city and under the same roof. The branches of these two associations could conduct their regular clinical meetings at least conjointly. These and other similar measures should precede an ultimate merger and amalgamation of these two associations into one powerful and united body.

Other medical associations for special purposes and with special functions are also flourishing in our country. The Indian Research Fund Association is an official body which finances medical research. The Medical Research section of the Indian science congress, is a quasi-official body. The Tuberculosis Association of India hopes to tackle the problem of the control of tuberculosis in this country. The All-India Obstetric and Gynecological conference brings together all those interested in this speciality. The Indian Radiological Association works in a silent way. The All-India Ophthalmological conference also meets regularly. The association of Surgeons of India is of recent origin and bids fair to grow big societies for physiology, biochemistry, anatomy, pathology, oto-rhino-laryngology and dentistry are working in some of the provinces but they have not yet attained an all-India status and reputation. All these specialist associations are indicative of a progressive tread and serve a very useful purpose. The entire medical profession in India, if united and organised, will undoubtedly be a mighty force for good, both for itself and for the general public.

Extracts.

TUBERCULOSIS AND SYPHILIS.

H. C. G. SEMON. BRIT. J. VEN. DIS., LONDON. July 1939, 15: 159.

It is self-evident that a man who has syphilis cannot contract syphilitic primary chancre, and an identical stipulation applies to tuberculosis which explains the extreme rarity of the primary tuberculous "chancre." The tuberculous lesion differs from that in syphilis in several particulars; the infiltration is softer and the edges are flat and somewhat overhanging, while the base has a rather granular appearance and tends to bleed more easily.

The histologic differentiation of the tuberculous from the tertiary syphilitic lesions is based on the fact that tubercles due to Koch's bacillus are more frequently multiple, while the gumma is usually a solitary infiltration with a strong tendency to necrose in its center. The author treats of the clinical differentiation of the cutaneous manifestations of the two infections and he discusses these manifestations according to the various regional areas which may be involved. On the scalp, the moth-eaten appearance of the syphilitic secondary stage, with or without roseola, is never seen in the diffuse fall of hair due to tuberculosis. Interesting problems of diagnosis occur on the area including the face, the ears, and nose. Lupus vulgaris is the commonest example of tuberculous infection. Cutaneous gummas can be mistaken for scrofuloderma which is more apt to occur on the neck than on the face. A history of cervical tuberculous adenitis may afford the diagnostic clue in nearly every case of so-called scrofuloderma. When something resembling it occurs for the first time in the adult, a Wassermann test should always antedate any surgical procedure. Tuberculous ulcers of the lips, gums and buccal mucosa are usually painful and have a more eroded and frayed appearance than syphilitic lesions.

Verrucose tuberculid is the commonest tuberculous manifestation of the forearms and hands. When the warty character prevails on palms and soles it is much more likely to be syphilitic than tuberculous. Difficulty may sometimes arise in diagnosis between tuberculids and papular syphilids occurring on the trunk, and it must be remembered that the former may occur in children with a positive Wassermann reaction. The syphilids are usually universal and may be associated with lesions of the mucous membranes. In the anogenital region the difficulty in diagnosis is not so much the differentiation between tuberculosis and syphilis as between syphilitic and other ulcers and nonulcerative infiltrations. Perianal sinuses are more likely to be tuberculous than syphilitic. A practically diagnostic characteristic of tuberculosis is the tendency to recurrences in scar tissue. Cicatrization in tuberculous cases is more likely to be followed by deformities than in parallel syphilitic involvement.

PULMONARY TUBERCULOSIS AND SYPHILIS.

R. R. TRAIL. BRIT. J. VEN. DIS., LONDON July 1939, 15: 171.

Medical opinion is strongly divided in regard to the incidence and extent of pulmonary syphilis. Since the final proof must always be demonstration of the organism in the lesions of their products, and in lung cases such proof is almost always missing, definite diagnosis of pulmonary syphilis cannot be established as it can be in tuberculosis. Radiologists contend that there are no essential characteristics and that diagnosis in such cases rests on elimination of the other possible causes. Even the proof of improvement by treatment is not completely satisfactory, for other conditions, such as amebic diseases, are known to be amenable to certain anti-syphilitic measures. There is, however, a fairly definite radiologic finding in lung films of certain cases of syphilis, and when it is present a positive Wassermann reaction is not unusual. This consists of a marked central fibrosis extending from the hilum outward to the periphery in fan-shaped distribution and nearly always equally marked on both sides. There is danger of missing the presence of pulmonary tuberculosis in syphilitic persons unless full investigation of the bronchitis in the secondary stage is undertaken, and an equal danger of missing the diagnosis of coincident syphilis in persons with pulmonary tuberculosis unless the extent of fibrosis is studied.

Since tuberculosis and syphilis are both comparatively common it is inevitable that a certain number of patients will have both conditions. Various authorities show that from 3 per cent. to 17 per cent. of all patients with one or the other condition have both conditions. An investigation carried out in England from 1931 to 1934 showed that 1.42 per cent. of 774 patients with positive sputum had a positive Wassermann reaction confirmed by the patient's history.

It has been almost impossible to come to a definite conclusion as to whether syphilis aggravates tuberculosis and makes it caseous or not; for it would be necessary to know the time at which each of the diseases was contracted. Trail believes that the conclusions of Dujardin and Duprez that secondary syphilis always aggravates tuberculosis while tertiary syphilis exercises a favorable action and produces fibrosis are true. From a study of other figures it would seem that there is no definite predisposition to tuberculosis among syphilitics.

Literature on the interplay of the two diseases is very contradictory. Figures are quoted which seem to prove that syphilis coincident with tuberculosis has a very favorable influence on the type and development of the tuberculosis and that treatment of the syphilis produces a marked improvement in the tuberculous lesion. Observations by the author on 14 cases diagnosed as having both diseases between 1927 and 1934 and followed up to date support these views.

**MAPHARSEN IN THE TREATMENT OF EARLY SYPHILIS.
COMPARISON OF RESULTS IN 188 CASES WITH THOSE OF THE
CO-OPERATIVE CLINICAL GROUP.**

LOUIS CHARGIN, WILLIAM LEIFER AND THEODORE ROSENTHAL.
ARCH. DERMAT. & SYPH., CHICAGO. Aug. 1939, 40: 208.

The need for a drug of high therapeutic efficiency and minimal toxicity and with facility of preparation and administration, is of vital importance for the ultimate eradication of syphilis. Among the drugs recently introduced, mapharsen seems most nearly to meet these requirements. Its manufacturers claim that it is a pure and stable chemical; it has low arsenic content in therapeutic doses; it has a high therapeutic efficiency in human beings as well as in animals; it is readily soluble, easy to administer, and has relatively low toxicity.

The authors undertook a study, the purpose of which was to determine the efficacy of mapharsen against early syphilis and to compare the results with those obtained by the most effective method of therapy now in use. The total series comprised 215 cases, of which only 188 were followed long enough to allow a fair evaluation of the therapeutic efficiency of the drug. The period of observation varied from 12 to 42 months, 135 (71.8 per cent.) having been observed for 18 months or longer.

The continuous method of treatment advocated by the Co-operative Clinical Group was followed in all cases. For most of the patients the maximal recommended dose was used—for males 0.06 gm. and for females 0.04 gm. In all cases 10 per cent. bismuth subsalicylate in oil was given in doses averaging 1.5 cc. per injection.

A comparison of results is made with those obtained by the Co-operative Clinical Group in their series of 169 cases of early syphilis treated similarly but with arsphenamine or neoarsphenamine and observed for 2 years or longer. In the author's series satisfactory results were obtained in 158 cases (84 per cent.), and unsatisfactory results in 30 (16 per cent. as compared with 134 (79.3 per cent.) satisfactory results and 35 (20.7 per cent.) unsatisfactory in the other group. The difference between the percentages for the two groups is so small that the authors consider it insignificant. Since the authors' patients were observed for a shorter period than those in the other group, it is possible that a small percentage of their cases will be found, on further observation, to be unsatisfactory, but the authors do not think it likely that the number of such cases would be large enough to alter the results materially.

The toxicity in this series was minimal, consisting chiefly of immediate reactions (pain in the arm, dizziness, nausea), and in practically every case the treatment with mapharsen could be continued. The rarity of cutaneous eruptions resulting from mapharsen was remarkable. No exfoliative dermatitis occurred.

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There was noteworthy parallelism in the results with the two groups; the best results were obtained with the disease in the seronegative primary stage, the next best with the secondary stage and the least satisfactory with the seropositive primary stage.

The authors are of the opinion that the results obtained with mapharsen in the treatment of early syphilis appear to be as satisfactory as those obtained with other effective arsenicals when the same method of therapy is used and that the favorable results justify further trial on a more extensive scale with careful, prolonged observation.

TREATMENT OF GONORRHEA IN PRIVATE PRACTICE.

ROBERT FORGAN AND H. P. NEWSHOLME. BRIT. M. J., LONDON.
July 15, 1939, 2: 138.

In this letter to the editor, Forgan states that specialists have been able to lay down certain broad rules for the use of the sulfonamide drugs in cases of gonorrhea but that they have not yet decided questions of optimum dosage and reliable tests of cure. It is painfully evident to these specialists that the rules are unknown or unobserved by practitioners who have neither the facilities nor the training for making accurate diagnoses or tests of cure.

Newsholme states that in the majority of cases of gonorrhea the symptoms of the disease will quickly disappear with sulfonamide therapy, but this does not mean that the patient is cured. Frequently such a patient is a "carrier" of the disease. Practitioners are urged not to discharge a patient as cured until proper tests of cure have been made and the patient has been under observation for at least 3 months after cessation of treatment. Tests of cure should include the examination of at least 3 smears and 3 cultures. Semears alone are not reliable. A blood examination, Wassermann test, and gonococcal complement fixation test should be made in each case. If the practitioner cannot make the necessary cultures, the patient should be referred to a venereal disease clinic, where the tests may be made and reported to the practitioner. Patients taking any of these drugs should be watched carefully for gastric symptoms, dermatitis, leukopenia, or any other signs of toxemia resulting from the treatment.

Forgan states that the percentage of defaulting patients with gonorrhea has risen sharply since the introduction of the sulfonamide drugs. However, if these drugs are wisely used early in the course of the disease, and if every patient is closely observed until cure is certain, the result should be a fall in the incidence of gonorrhea vastly greater than that of syphilis (in England) during the last 20 years. Some measure of legal compulsion will probably be required before gonorrhea is finally conquered.

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