The Purvis Oration

ON

RESPIRATORY PHENOMENA IN NERVOUS DISEASE.

DELIVERED BEFORE THE WEST KENT MEDICO-CHIRURGICAL SOCIETY ON DECEMBER 9TH, 1921,

BY

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APART from the large proportion of our patients who suffer from primary respiratory affections, there are a considerable number who exhibit respiratory symptoms which are a consequence or complication of other maladies. I wish here to draw attention to that important group which we find occurring secondary to various diseases of the nervous system.

Respiratory Centres.

Let me first of all recall the nervous mechanism of respiration. The primary or lowest reflex respiratory centre is located in the lower part of the medulla oblongata, probably in the region of the visceral sensory nucleus of the fasciculus solitarius. This vital centre maintains ordinary breathing with its regular rhythm. During ordinary quiet breathing the inspiratory phase is an active muscular process of sucking air into the lungs; expiration, on the other hand, is a mere passive recoil of the lungs and thoracic cage, due to elasticity of their tissues.

The muscles involved in ordinary inspiration comprise, first, the diaphragm (innervated by the phrenic nerves whose motor nucleus in the spinal cord is at the level of the third, fourth, and fifth cervical segments); and second, the intercostal muscles (innervated by the series of anterior spinal roots from the first to the twelfth thoracic inclusive).

During forced or laboured respiration, and also in coughing and sneezing, various supplementary or accessory muscles, both inspiratory and expiratory, are called in, so as to reinforce the ordinary mechanism of inspiration and recoil.

Ordinarily the respiratory centre in the medulla is excited to activity by variations in the oxygen and carbon dioxide content of the blood. But it is also influenced by nervous stimuli. These stimuli reach it through various sensory nerves, chiefly along the vagus, which is the visceral sensory nerve of the respiratory tract, but also from almost any sensory nerve of the body; thus coughing and other respiratory reflexes can be induced by widely varying stimuli.

Perched above the vital respiratory centre in the medulla there is a higher reflex centre in the mid-brain, situated bilaterally in the mesial part of the optic thalami. This centre is concerned with emotional reflexes, such as laughing and sobbing, actions which involve modifications of the respiratory movements, accompanied at times by secretory phenomena, such as lacrymation, and even by visceral phenomena—for example, precipitate contractions of the bladder or bowel. Lastly, higher still, there are motor centres in the cerebral cortex, by means of which we can exercise a partial voluntary control over the lower respiratory mechanisms.

It is not surprising that these complex respiratory centres, situated as they are deep within the brain, medulla, and spinal cord, are liable to become disordered in diseases and injuries of the nervous system, whether the lesion implicates the central nervous system itself or the sensory or motor elements of the reflex respiratory arc.

Respiratory phenomena are commonly an accessory element in nervous diseases or injuries; sometimes, however, they may dominate, for a time, the whole clinical picture.

Respiratory Symptoms in Cerebral Lesions.

In *cerebral concussion*, say from a head injury, the morbid process consists in a sudden violent squeezing of the brain. This produces widespread cerebral anaemia and is associated with minute perivascular haemorrhages in various parts, notably in the grey matter of the cortex, the mid-brain, and the spinal cord. Besides the characteristic feeble frequent pulse, the subnormal temperature, and the low blood pressure of the concussed patient, we observe that his breathing is curiously slow, shallow, and irregular. Later on, when reactionary cerebral oedema supervenes, consciousness returns, the temperature rises, the pulse becomes full and bounding, and the respirations are now deeper than normal.

In cases of *apoplexy* from destructive cerebral lesions, whether due to haemorrhage, thrombosis, or embolism, in addition to the unilateral paralytic signs and symptoms, varying in distribution according to the location of the lesion, we often find those of coma. The probable explanation of this is that the brain as a whole is compressed by diffuse oedema surrounding the focal lesion, whilst beyond the oedematous zone there is an outlying zone of venous obstruction extending down towards the medulla, where it produces irritative symptoms. In such cases of cerebral compression, in which the patient is comatose with slow pulse and raised blood pressure, we note that his breathing is loud and stertorous in character. The snoring noise is due partly to flapping of the soft palate and partly to falling backwards of the root of the tongue. The noise of such stertorous breathing can often be alleviated by gently turning the patient on one side and propping up one shoulder, so that his tongue falls to one side instead of backwards. The patient, of course, remains unconscious as before, but his friends at the bedside are comforted by the quietening in his breathing. Sometimes in comatose patients of this sort we observe the highly ominous phenomenon of rhythmic breathing—the so-called Cheyne-Stokes type of respiration. In this there is a regular alternation of periods of activity and inactivity of the bulbar respiratory centre. During the inactive phase there is total cessation of breathing, which may last several seconds; then the respiratory movements gradually reappear, steadily becoming stronger and deeper. After persisting at a maximum for a few breaths the movements again subside, gradually declining in amplitude, until temporary complete apnoea is again reached. This process is repeated again and again until, in fatal cases, it is replaced by the true terminal breathing, popularly known as the "death-rattle." This is irregular and gasping in character, and to it coarse bubbling tracheal sounds are commonly superadded.

There is another less common form of cerebral haemorrhage in which the respiratory symptoms are peculiar and characteristic. This is what is known as *chronic subdural haemorrhage* or *delayed traumatic apoplexy*, a condition in which a collection of blood, often very large, gradually accumulates for days and weeks between the dura mater and the cerebral hemisphere. The blood is enclosed within a distinct fibrinous membrane, derived from the coagulated blood itself, which restricts the rate at which the haemorrhage spreads. The condition is sometimes bilateral. The bleeding is venous in origin, being a slow ooze from the short cerebral veins which enter the superior longitudinal sinus. Subdural haemorrhage of this sort is traumatic in origin, but the injury is often so trivial as to be overlooked. Following some slight blow on the head, after an interval, several days at least, the venous haemorrhage gradually becomes large enough to produce symptoms of intracranial pressure. Then headache supervenes, and gradually, a week or two later, the patient becomes dull and drowsy. After several weeks more the limits of mechanical compensation are reached and the symptoms change, rather suddenly, so that the drowsiness deepens into coma, perhaps ushered in by sharp pain in the head and by vomiting. This coma comes and goes, varying in intensity in a curious way, so that within a period of twenty-four hours the patient may pass from consciousness to coma and back again, being perhaps only a little dull mentally in the intervals. In this condition the respiration is very curious, for the patient, even when awake, breathes as if he were sound asleep—that is, his inspirations are deeper than normal and expiration is also slightly emphasized.

In the terminal coma of *cerebral meningitis*, *cerebral abscesses* or intracranial *tumours* the onset of stertorous breathing or of Cheyne-Stokes breathing is of grave omen and calls for prompt relief of intracranial pressure if this be still possible. In meningitis, especially in tuberculous basal meningitis, quite apart from threatened coma, it is not uncommon to observe a curious irregular type of respiration consisting in a want of harmony between the

diaphragm and the intercostal muscles, so that they no longer act synchronously.

In other varieties of coma—for example, post-epileptic coma, toxic coma (whether from outside poisons such as opium and alcohol, or from endogenous poisons as in uraemic or diabetic coma), in the coma of cerebral malaria and of sunstroke, and so on, stertorous breathing is one of the cardinal symptoms. In the coma of opium poisoning there is excessive slowness of respiration. Diabetic coma is often preceded for a day or two by air-hunger with slow, deep, sighing respirations and by uncontrollable drowsiness, gradually merging into coma with deep stertorous breathing.

Many years ago Hughlings Jackson made the interesting observation that in some cases of ordinary hemiplegia the chest wall during quiet respiration moves more freely on the paralysed side, corresponding as it were to the exaggeration of the deep reflexes in the paralysed limbs, whereas during voluntary deep inspiration the respiratory excursion, like other voluntary movements on the hemiplegic side, is distinctly less than normal.

Respiratory Symptoms in Bulbar Lesions.

In chronic nuclear diseases of the medulla—for example, in *chronic bulbar palsy*, whether this commences as a primary affair in the bulb or occurs as the upward extension of an amyotrophic lateral sclerosis of the spinal cord, or is due to syringobulbia or to tumours of the medulla—we observe that, if the vagal nuclei or nerves are implicated, the patient has a continuous dyspnoea, more or less severe, to which from time to time are superadded paroxysms of more intense dyspnoea. In such cases as the various bulbar nuclei, supplying the tongue, lips, palate, and larynx, become involved, the voice becomes feeble and monotonous. If the laryngeal abductors are paralysed, the glottis becomes narrowed to a mere slit, talking is an effort, respiration is laboured, even at rest, and the superaddition of the mildest bronchitis is liable to be fatal owing to difficulty in coughing up the bronchial secretions. If the adductors of the glottis are paralysed, phonation is lost and the patient may be unable to speak above a whisper; moreover, imperfect closure of the glottis favours the accidental inhalation of food particles with the possibility of a resulting bronchopneumonia. Curiously enough, syringomyelia with severe implication of the bulbar nuclei is sometimes compatible with prolonged survival.

Thus, I have in mind a lady who developed typical phenomena of syringomyelia in 1907, including dissociated anaesthesia of the upper limbs and neck. In 1909 she developed well-marked laryngeal stridor with bilateral, asymmetrical abductor paralysis, and became liable to attacks of acute dyspnoea. Nevertheless, she is still alive and able to get about in 1921, twelve years from the onset of her bulbar symptoms.

Acute or subacute bulbar palsy from affection of the bulbar nuclei or nerves is a much more serious affair. These nuclei may be implicated by the upward spread of an acute anterior poliomyelitis, or by the downward extension from the mid-brain of an epidemic encephalitis; or an acute ascending neuritis of toxic origin may spread up to the bulbar nerves as in Landry's paralysis, in which case respiratory paralysis begins with weakness of the abdominal and intercostal muscles, spreads up to the phrenic nerves innervating the diaphragm, and culminates in implication of the laryngeal and palatal nerves.

Respiratory Symptoms in Spinal Cord Lesions.

Progressive ascending lesions of the spinal cord in the dorso-cervical region are often accompanied by gradually increasing respiratory embarrassment as the muscles of inspiration become successively involved. Paralysis of the abdominal muscles favours the occurrence of meteorism or intestinal distension, pressing the diaphragm upwards, and as the intercostals cease from activity, more and more strain falls upon the diaphragm and upon the auxiliary muscles of inspiration which are called in to supplement its activity. Respiration becomes slower and more difficult, whilst expiration is shortened and followed by a long pause before the next inspiration sets in. Finally, when the upper level of the phrenic nucleus, the third cervical segment, is reached, the diaphragm is paralysed, as well as the intercostals, and the patient dies from asphyxia, since the supplementary muscles of respiration above that level (chiefly the sterno-mastoids, sterno-hyoids, sterno-thyroids, and the uppermost fibres of the trapezii) are rarely able by themselves to carry on adequate respiratory movements for more than a short time.

Symptoms such as we have just described may occur suddenly in fracture dislocations of the cervical vertebrae, and in wounds or haemorrhages within or around the *spinal cord;* they may come on acutely in acute poliomyelitis, in softening from arterial thrombosis (formerly misnamed "myelitis"), more gradually in abscesses or meningeal inflammatory deposits compressing the cord, and still more insidiously in tumours of the spinal cord and in primary degenerative diseases of the anterior cornual cells, as in amyotrophic lateral sclerosis.

An important group of respiratory troubles occurs as a complication of *tabes dorsalis* in the form of laryngeal crises. Sometimes the tabetic patient, even in the early, pre-ataxic stage of his malady, may have paroxysms of violent, causeless coughing coming on suddenly day or night, so severe that the patient becomes cyanosed for a few moments, has acute laryngeal spasm and may even become unconscious for a few moments. Later in the disease we meet with laryngeal palsies of various types, the commonest being a bilateral abductor paralysis. In such cases, even during quiet respiration, if we examine with the laryngoscope we can see distinct ataxia of the vocal cords, sometimes when they are moving outwards during phonation, sometimes as they come together for phonation, thereby rendering the voice tremulous. But it is especially in bilateral abductor palsy that laryngeal crises are liable to supervene in which, from some trivial catarrh or even without apparent exciting cause, the patient has a superadded laryngeal spasm producing cough and urgent dyspnoea, so urgent perhaps as to demand tracheotomy or intubation. A much rarer form of respiratory affection in tabes is a widespread apnoeic crisis, of which I have only seen one example.

This occurred in a tabetic patient of 42 who had suffered from unsteadiness of the legs for eight years. During the last three or four years he had been subject to occasional bladder crises and also to typical laryngeal crises. About a year before I saw him he developed occasional crises of a totally different sort, in which he became unconscious with cyanosis and stertorous breathing; but had been free from them for two months until his terminal attack in which I had the opportunity of witnessing several paroxysms.

The paroxysms occurred at intervals of about ten minutes. Each attack began with sudden total cessation of respiration, both diaphragmatic and intercostal. After a couple of minutes of this apnoea the patient gradually became cyanosed and then developed asphyxial convulsions, with clonic twitching of the face, trunk, and limbs. These passed off in a few seconds and were succeeded by inspiratory stridor, after which he gradually recovered consciousness with normal quiet respiration. The whole affair lasted about two minutes in all. The pulse persisted regularly but was rapid, 136 per minute, both during and between the successive attacks. These apnoeic crises recurred for two days, at intervals of ten to twenty minutes, in spite of assiduous treatment by full doses of atropine and strychnine, oxygen inhalations and occasional morphine. The individual attacks became shorter in duration, lasting only a minute in all, and the intervals between the paroxysms became longer, as much as twenty minutes, but the patient became stuporose and died from exhaustion on the fifth day.

Crises of sneezing are occasionally met with in tabes, accompanied by secretory phenomena in the form of rhinorrhoea and lacrymation, but, although these are exhausting to the patient, they do not cause danger to life such as occurs in the laryngeal and still more in the apnoeic crisis.

Laryngeal palsies occur in other degenerative nervous diseases, notably in disseminated sclerosis, and even in Friedreich's ataxia, but unlike tabetic cases, they are rarely accompanied by respiratory difficulties.

Respiratory Symptoms in Peripheral Nerve Lesions.

Respiratory troubles are sometimes met with in *peripheral neuritis* from toxic degeneration, whatever be the cause of such neuritis—for example, alcohol, diphtheria, arsenic, beri-beri, etc. Diphtheritic neuritis has a special tendency to select the nerves of the soft palate, giving a nasal twang to the voice and also causing difficulty in swallowing, with a special inclination to nasal regurgitation of fluids. Post-diphtheritic laryngeal palsies are less common, but we sometimes meet with them.

Diaphragmatic paralysis from peripheral neuritis is often overlooked, since the patient has little or no discomfort when at rest and shows merely a certain degree of acceleration in breathing. But as soon as he makes the

slightest physical effort he becomes markedly dyspnoeic. Once it is looked for, diaphragmatic palsy is easy of recognition, for we note that instead of the normal pushing forward of the epigastrium during inspiration, the abdominal wall is sucked inwards at each inspiration; meanwhile, there is marked over-action of the intercostals.

Paralysis of the intercostal muscles is rare in peripheral neuritis. In spinal cord lesions, however, it occurs fairly commonly, and the level of the lesion in the cord can often be accurately located by noting up to what level the intercostals are thrown out of action. In patients who are not too fat the healthy intercostal muscles can normally be felt to harden during deep inspiration. Absence of intercostal contraction, up to a certain level in the chest, is an important confirmatory sign in localizing lesions in the thoracic region of the spinal cord. When all the intercostals are paralysed, there is absence of expansion of the thoracic cage during inspiration.

Unilateral injuries of the *vagus*, and especially of its recurrent laryngeal fibres, are not uncommon in war time. The following is an example:

After a hand-to-hand encounter on the Salonica front in 1917 a Bulgarian prisoner was brought in with a bayonet wound in the back of his neck. The entry wound was immediately above the middle of the spine of the left scapula, three inches internal to the tip of the acromion process. There was no exit wound. Within a week a small pulsatile swelling became palpable at the outer side of the left sterno-mastoid, at the level of the cricoid cartilage; no thrill or bruit was made out over this little swelling, but it was evidently a small aneurysm in the neighbourhood of the common carotid artery. When admitted to hospital, a few hours after being wounded, the patient had surgical emphysema of the left upper chest and some haemoptysis from wounding of the apex of the lung. His voice was hoarse and he had paralysis of the left deltoid, biceps and supinator longus muscles, all supplied by the fifth cervical root. These muscles ultimately developed electrical reactions of degeneration, and there was also an area of cutaneous anaesthesia along the outer side of the left upper limb, from the middle of the deltoid to the thumb, corresponding to the fifth cervical root, together with another area of cutaneous hyperaesthesia above this, over the tip of the shoulder and side of the neck, corresponding to the fourth cervical root. The biceps jerk and the supinator jerk were absent on the left side, whilst the other reflexes, including the pronator jerk and triceps jerk, beyond the territory of the fifth cervical segment, were unimpaired. In addition, the left vocal cord was fixed in the cadaveric position, evidently from damage to the vagus or to its recurrent laryngeal branch within the left carotid sheath. The curious area of cutaneous hyperaesthesia might be accounted for as due to irritation of the sensory fibres of the phrenic nerve in the scar, but no motor impairment of the diaphragm could be detected.

Even in civilian practice, as the result of aortic aneurysm and other intrathoracic abnormalities, the *recurrent laryngeal nerve*, generally on the left side, is not uncommonly affected. The vagus nerve, including its recurrent laryngeal branch, may also be accidentally damaged in operations in the thyroid region.

Unilateral traumatic lesions of the *phrenic nerve* are much less frequent, but the inclusion of this nerve in a ligature has been known to produce inveterate cough from irritation of its sensory fibres. This nerve may also be implicated by bullet and bayonet wounds, causing unilateral paralysis of the diaphragm, easily verified by radioscopy.

Respiratory Symptoms from Muscular Affections.

In many of the primary myopathies the progressive general muscular enfeeblement may in time implicate the respiratory muscles and thereby diminish the patient's resistance to pulmonary affections, but localized respiratory paralysis does not occur. In *myasthenia*, on the other hand, all the features of bulbar palsy may be reproduced, including laryngeal palsies and respiratory failure. In this disease the paralytic symptoms vary in intensity from time to time; they are specially aggravated by fatigue and tend to clear up again after a period of rest. Unfortunately, with the respiratory muscles prolonged rest is unattainable, for whilst the patient's tired-out respiratory muscles are resting he may die of asphyxia, and as a matter of fact this is the common termination of myasthenia gravis.

Family periodic paralysis is another rare affection, sometimes appearing in successive generations of the same family, in which the patient, otherwise healthy, has attacks of profound flaccid paralysis of the limbs and trunk, lasting from a few hours up to a couple of days at a time. The affected muscles are not only paralysed, but for the time they become inexcitable to electrical stimulation—the so-called cadaveric reaction. In such attacks the intercostal muscles may be implicated, but, so far as I know, the diaphragm has never been affected, and therefore respiration can still be carried on.

Spasmodic Respiratory Affections.

The foregoing respiratory troubles are mainly paralytic. Let us now turn to the group of spasmodic respiratory affections. These may be due to disease either of the vegetative or the cerebro-spinal nervous system.

With regard to the vegetative nervous system I need only mention in passing the important spasmodic respiratory affection known as *asthma*, whose phenomena of laboured breathing consist essentially in spasm of the non-striped bronchial muscles, innervated as we now know by the dorsal motor nucleus of the vagus. In asthma the bronchial nerve centres react with abnormal violence to various blood-borne irritants, whether these be foreign proteins or bacterial toxins, also to peripheral irritants, nasal or otherwise, or even to psychical stimuli. The result is the asthmatic paroxysm, whose features are so familiar that I need not weary you by recapitulating them.

Coughing, yawning, sneezing, and hiccup are all of them reflex phenomena which may be induced in healthy individuals by appropriate stimuli. Of these, *hiccup* calls for special mention. Hiccup is a spasmodic myoclonic contraction of the diaphragm, occurring so suddenly that the glottis is momentarily closed by suction, producing the characteristic inspiratory noise or "hic." In slight cases of hiccup the affair is transient and produces only trivial discomfort. Sometimes, however, it may become so severe and prolonged as to induce profound and even fatal exhaustion. The reflex phenomenon of hiccup is sometimes excited by transient gastric or other abdominal irritation. It may also be due to disease of the peritoneum in the region of the diaphragm (the phrenic nerve being sensory as well as motor in function). Other cases of hiccup are due to central disease of the respiratory centres in the medulla. Thus, for example, it may be produced by the toxin of epidemic encephalitis. In encephalitic hiccup the myoclonic spasms may implicate not only the diaphragm and the constrictors of the glottis, but may even extend to the muscles of the abdominal wall and trunk. Hiccup of this encephalitic type may continue for days at a time, at the rate of twelve to fifteen hiccups per minute. Most cases clear up in three or four days, but others go on for weeks. Hiccup may also occur as a terminal symptom in tumours of the posterior fossa of the skull. Lastly, we sometimes meet with cases of hysterical hiccup. These are recognized by their history and concomitant phenomena, and the symptom promptly subsides under appropriate suggestive treatment.

Before dismissing the subject of cough we ought to recall whooping-cough or pertussis (an infective disease causing irritation of the coughing centres in the medulla) with its initial and terminal stages of catarrh and its paroxysms of rapidly succeeding expiratory coughs, followed by a single long-drawn inspiratory laryngeal "whoop." These paroxysms of alternate coughs and whoop follow one another in succession until the attack culminates either in temporary apnoea with cyanosis, or in vomiting.

Laryngismus stridulus, especially occurring in rickety children, consists in laryngeal inspiratory stridor and is often associated with tetany or painful tonic spasms in the hands and feet. Laryngismus itself may be regarded as a tetany of the adductor muscles of the larynx.

In the classical accounts of *tetanus* the respiratory muscles are described as becoming implicated in the general muscular spasm so that during a severe tetanic paroxysm not only is there opisthotonos, but also tonic spasm of the respiratory and laryngeal muscles, causing asphyxia which may be fatal. I had considerable personal experience of tetanus during the Gallipoli campaign and had the opportunity of watching a number of severe cases of tetanus. Seven of my cases were fatal, but in none of them did death happen to be due to respiratory spasm or to asphyxia; what the patient died of was progressive cardiac feebleness, sometimes several days after the tetanic spasms had subsided completely.

Personally, I have never seen a case of *rabies* (or so-called hydrophobia), but the symptoms are described as being of two varieties, sometimes a spasm of the inspiratory muscles, as well as of the pharynx and oesophagus, at other times a paralytic inertia of the whole respiratory apparatus, occurring either primarily or following the spasmodic attacks. The patient may die either from respiratory spasm, or from syncope. On the other hand, *hysterical pseudo-hydrophobia* is not uncommon, and I have seen several instances of this in which the patient erroneously thought that he had been bitten by a rabid dog. Here we observe globus hystericus with rapid breathing and various dramatic movements of the trunk and limbs, sometimes accompanied by a loud hysterical laryngeal noise intended to resemble a dog's bark. But there is neither true respiratory spasm nor paralysis, and to the careful observer the diagnosis presents no difficulties.

There are also other nervous diseases in which the respiratory mechanism may be upset. For example, in a *major epileptic fit* the tonic phase of the fit is often ushered in by a curious laryngeal wail, the so-called epileptic cry, produced by tonic spasm of the expiratory muscles. Throughout the tonic stage of the epileptic fit the respiratory muscles are fixed, respiratory movements cease and the patient becomes increasingly blue and cyanosed. Then, when the clonic stage sets in, the cyanosis passes off, the respiratory muscles take part in the jerking movements and saliva may be jerked from between the lips. I once had the opportunity of doing a laryngoscopy during the clonic stage of an epileptic fit and was able to see the glottis alternately opening and shutting, synchronously with the clonic jerks of the trunk and limbs.

Both in *athetosis* and in *chorea* the irregular involuntary movements may implicate not only the limbs and face but also the respiratory muscles, causing the breathing to be irregular and clumsy, so that the diaphragm and intercostals no longer contract synchronously and the smooth rhythm of respiration is interrupted. In consequence, even the patient's articulation may be jerky. In so-called "paralytic" chorea the superadded factor of muscular feebleness may sometimes be so severe that the patient's voice is reduced to a faint whisper.

The fine muscular tremors of the limbs in *exophthalmic goitre* may sometimes spread to the trunk and to the respiratory muscles. This tremulous respiration, as Minor pointed out, is best marked during a long expiration.

In advanced *paralysis agitans*, if we examine the patient laryngoscopically, we may observe a rhythmic tremor of his vocal cords, synchronous with the tremor of the limbs. This laryngeal tremor usually ceases during phonation, but not always. The patient's voice becomes thin, monotonous, and feeble. His articulation, like his gait, becomes festinant, so that he begins his sentences slowly but tends to hurry towards the ends of sentences or of long words, pronouncing the final syllables hurriedly.

Various respiratory phenomena may be met with in *hysteria*. Of these, hysterical cough is the commonest, but hysterical tachypnoea or rapidity of respiration, hysterical sobbing, also all sorts of curious laryngeal noises, may be met with in different cases. But, whatever their variety, all these hysterical respiratory affections cease spontaneously during sleep. Hysterical cough occurs almost always in bouts or paroxysms, often at the same special time of day, and most frequently when the patient knows herself to be under observation. It may last for hours at a time. Hysterical cough is unaccompanied by true dyspnoea, whilst abnormal physical signs in the respiratory organs are conspicuous by their absence. Hysterical cough often has a peculiar metallic musical sort of noise. Hysterical laughing or sobbing are frequently part of a hysterical paroxysm, whether occurring before, during, or after the hysterical attack. Hysterical tachypnoea is another fairly common manifestation, consisting in paroxysms of extremely rapid breathing (80, 100, or even 150 per minute), but without cyanosis or other respiratory distress. The pulse remains quiet and steady throughout the attack, there are no abnormal physical signs in the chest, and the temperature remains normal throughout. Hysterical aphonia, another common type, is easily recognized, not only by its concurrent phenomena, but by its characteristic laryngoscopic appearances, showing deficiency of adduction of the vocal cords.

Finally, we have the interesting group of *respiratory tics or habit-spasms*, which occur in highly strung, often highly intelligent individuals of psychasthenic nervous constitution. Such patients are not necessarily mentally subnormal; on the contrary, some of them are far above the average of intelligence. Respiratory tics are of many varieties. I recall one individual—a distinguished university professor—whose habit-spasm consists in an occasional sudden sniffing inspiration accompanied by wrinkling up of the nose; another individual—a surgeon of world-wide reputation—who makes an occasional sudden inspiratory laryngeal noise from time to time when talking. In another highly strung young man the breathing is interrupted at irregular intervals by paroxysms of short little expiratory puffs at the rate of about two per second, associated with grimacing and with jerky movements of the arms; another individual gives an occasional little nervous cough or grunt, and so on. Then again, one variety of stammering, in which the patient sticks in the middle of a vowel (less common than the type where he sticks over a consonant) is simply a respiratory tic affecting the muscles of the false vocal cords. Examples of tics or habit-spasms of this sort might easily be multiplied, and many of you can recall for yourselves friends or patients who perform peculiar respiratory actions of this sort. A tic or habit-spasm, it should be remembered, is a psychomotor affair of cortical origin, consisting in the frequent explosive repetition of the same motor act. It never interferes with voluntary movements. Sometimes the tic takes place automatically and without the patient's conscious attention; in more severe cases the tic absorbs the patient's attention during its performance, and is accompanied by a feeling of irresistible compulsion. The greater the psychical abnormality the more violent does the tic tend to be.

An Address
ON
SOME OF THE CAUSES OF OUR C3 POPULATION.*

BY

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WE as a profession had a more or less general acquaintance with the existence and growth of that section of the population which has come to be known as C 3, but it required a great war, necessitating the passing of a limited age group of the manhood of the nation through the sieve of physical and mental fitness, for it to become general knowledge. The examination of recruits was conducted by medical men who, generally it can be said, had no expert knowledge of mental diseases, and consequently the sieve had a very open mesh as far as mental disability was concerned—sufficiently open to allow the passage through of all except the most obvious cases of mental disease, and the rapidity with which the examinations had to be made, owing to the urgent demands of the situation, caused, in many instances, even men suffering from gross forms of insanity and mental defect to be passed into the service. Early in 1917, owing to the large number of soldiers being certified as insane, there was an outcry that the army was creating lunatics, and calling for special treatment of these men as victims of the war. In reply to this I pointed out that down to the end of 1916, estimating from the statistics then available, 5,000 men had been enrolled in the forces in England, Scotland, and Wales alone who, had the war not taken place, would have been certified as insane and placed under treatment as pauper patients. If this estimate had been corrected up to the end of 1919 the figures would have been approximately 10,000.

In this connexion it is most interesting to note the statement made in the House of Commons by the Minister of Pensions that on December 9th, 1920, over two years

* Delivered at the annual meeting of the Kent Branch, held at the London County Mental Hospital, Bexley.