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Contents February, 1947

The Sequelae of Closed Head Injuries*

Evaluation, Treatment and Prognostic Considerations

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IT has long been recognized that the great majority of patients subjected to acute craniocerebral trauma eventually make a complete recovery, but nevertheless, many suffer from debilitating symptom-complexes, which vary in severity and duration, before this ideal of complete recovery is attained. It is because of the fact that these post-traumatic sequelae can be appreciably alleviated in both degree and duration by the correct evaluation of the causative factors involved, and by the application of modern concepts in treatment, that this paper is presented.

The important sequelae will be enumerated below followed by a discussion of the important features associated with those most commonly encountered, and a final consideration of the different methods of treatment.

The sequelae may be most conveniently considered under two general headings—(1) those symptoms commonly associated with the "post-traumatic syndrome," and (2) those signs most commonly associated with "residual organic lesions."

1. "*Post-traumatic syndrome.*"
   - (1) Headaches and dizziness.
   - (2) Post-traumatic confusion.
   - (3) Fatiguability.
   - (4) Apprehension and anxiety.
   - (5) Emotional instability.
   - (6) Defects in concentration (and memory).
   - (7) Intellectual impairment.
   - (8) Changes in personality (late).

2. *Residual organic signs.*
   - (1) Cranial nerve palsies.
     - (a) Blindness.
     - (b) Diplopia.
     - (c) Anosmia (with impairment of the sense of taste).
     - (d) Facial paralysis;—immediate or delayed.
   - (2) Organic cerebral damage.
     - (a) Spastic paralysis.
     - (b) Discriminative sensory loss.
     - (c) Aphasia.
     - (d) Hemianopsia.

* Presented at the Dalhousie Refresher Course, October, 1946.

(3) Signs due to involvement of the auditory apparatus.
   (a) Dizziness.
   (b) Deafness—middle ear or inner ear types.

(4) Intellectual impairment (due to bilateral frontal lobe contusion or laceration).

(5) Symptoms related to skull defects.

(6) Post-traumatic epilepsy.

Commonly Recognized Sequelae

1. *Generalised Headache.*

The complaint of persistent headache commonly associated with dizziness is undoubtedly the most frequent symptom occurring as a result of head injury. In general, the outstanding feature of this headache is its aggravation by changes in the position of the head, as for example when the erect position is suddenly assumed, or if the patient is forced to stoop, and particularly if the stooping is associated with the added effort entailed in picking up some object of considerable weight. This type of headache is commonly associated with dizziness, light-headedness, "black-out," and a sense of mental and physical fatigue, and the patient may become completely debilitated as a result of the severity of this symptom-complex. The nature of the headache suggests immediately that it is related to changes in the cerebral circulation, and indirect support for this view is supplied by the fact that sensitive tissues within the skull are largely related to the major vessels such as the meningeal vessels, the large vessels at the base of the brain, and the dura in relation to the large venous sinuses. It is generally believed that these headaches are dependent upon an unstable cerebral circulation, and probably the result, as McKenzie[1] points out, of instability of vasomotor centres that have been subjected to trauma and are subsequently incapable of quickly supplying an adequate circulation to the brain when the position of the head is suddenly changed. McKenzie has further observed that in severe cases the blood pressure may fall to zero when the erect position is suddenly assumed, and this would suggest that the headache may be caused by the resultant traction on sensitive areas by the sudden reduction in the circulating intracranial blood volume. When stooping the same chain of events occurs if the vasomotor tone is not quickly altered, for congestion of the intracranial vessels occurs suddenly, causing direct stimulation, by pressure, of these sensitive areas. The treatment of this generalized form of headache,of course, primarily consists in the prevention of its occurrence by active management of the patient initially, with a view to restoring a normal function to the damaged vasomotor centres as soon as possible. The prevention of headaches and dizziness is extremely important for, if they persist, they may be accompanied by an extreme functional overlay, particularly in patients with a pretraumatic unstable personality.

2. *Migrainous Headaches.*

Although not as common as the preceding type of headache, a migrainous form occurs quite frequently and Rowbotham[2] believes the pain in this form of headache is due to extreme spasm or dilatation of the arteries of the scalp, the dura, and the Circle of Willis. The hypothalamus is normally the highest

controlling centre of the vasomotor system, efferent impulses traversing the brain stem and spinal cord to reach the peripheral autonomic nervous system, and if injury occurs in the region of this centre, its function may be so changed that it reacts in an abnormal manner to the many stimuli, either emotional or physico-chemical, which affect it and consequently abnormal impulses are given off causing painful spasm of blood vessels within the head and the resultant peculiar sensation characteristic of migraine.

3. *Localized Headaches.*

Penfield and Norcross[3] believe that this type of headache is due to the presence of subdural adhesions, pain resulting when adhesions of this type are attached to a sensitive area of the dura. They postulate that, at the time of injury, displacement of the brain occurs at the arachno-dural interface and for some physical reason, such as haemorrhage, does not immediately regain its normal position after the violence has ceased to act. In this position, adhesions form between the arachnoid and the dura and are later stretched as the brain tends to take up its normal position, resulting in the production of a painful stimulus if a sensitive area of the dura is involved. It is in this type of headache that the sub-dural insufflation of air has been suggested in an attempt to break down the adhesions, but in our own experience a definitely localized form of headache has been extremely rare and we have no experience therefore with the use of sub-dural insufflation or the introduction of air by means of an encephalogram, in its treatment.

4. *Dizziness.*

It is important to realize that when the patient complains of dizziness or giddiness he usually means that things go black before his eyes and he feels faint whenever he makes a sudden movement, such as stooping, and the symptom is not one of true vertigo in any way. These complaints are probably related to the headaches already described and are due to momentary ischemia of the brain as the result of a labile cerebral circulation dependent upon vasomotor instability. Occasionally the dizziness presents the attributes of vertigo, and in these cases the underlying pathology may be a contusion of the labyrinth or involvement of the vestibular portion of the eighth cranial nerve, with stimulation of these affected structures, leading to disabling symptoms.

5. *Post-traumatic confusion.*

The picture presented by the patient is usually one suggestive of a "Korsakow's Syndrome" with marked temporal and spatial disorientation and an attempt by the patient to rationalize these states by confabulation. Recently Lehmann[4] has suggested that this syndrome may have an organic basis in changes in the cerebral circulation, and has reported an interesting result from the use of massive doses of nicotinic acid in its therapy. In our own series, two cases were treated in this manner and their improvement was thought to have been accelerated although there is no definite way of assessing the results. Presumably the drug exerts any beneficial action it may have due to the resultant increase in cerebral blood flow accompanying the vasodilatation of cerebral vessels.

6. *Intellectual Impairment.*

Permanent total incapacity from intellectual impairment after closed head injuries is fortunately very rare and if the impairment is reversible, its duration is usually less than three months, the condition becoming less marked with increasing remoteness from the time of injury. Although mental dulling as compared to specific impairment of intellect may be functional in nature representing an unsatisfactory adjustment of the patient to the psychological trauma of the accident, this impairment probably has an organic basis in most cases and it is most marked in the cases where an encephalogram reveals dilatation of the frontal horns of both ventricles as the result of a bilateral frontal lobe contusion or laceration. This ventricular dilatation, termed "hydrocephalus ex vacuo," results either from traction on its walls by contracting glial scars or by expansile enlargement into an area of softening as the result of normal rhythmic ventricular pressure changes coincidental with intracranial pulsation (O'Connell)[5]. The evaluation of the degree of impairment is confused by neuro-psychiatric overlay, but Reynell[6] points out that vocabulary, general information, and comprehension are less affected by organic cerebral disease than are arithmetical reasoning, memory and rational thinking, and Ruesch[7] emphasizes the fact that the mental functions chiefly affected are speed, judgment, and the ability to maintain effort. He states that the most reliable tests in evaluating the impairment are the "100-7" test, pictorial absurdities, pictorial discrimination, the naming of colours, and the ability to read intelligently, but it is true that in minor variations the testing must be performed by a skilled neuro-psychiatrist before a definite opinion can be offered as to the presence or absence of an organic basis for the findings.

As Ross and McNaughton[8] point out, in referring to psychological tests in common usage, standardised psychometric procedures may be applied to determine whether there is any discrepancy between the individual's previous intellectual level as judged by vocabulary or other verbal tests, and his present level as indicated by performance at new or abstract problems.

7. *Post-traumatic epilepsy.*

Epilepsy may occur immediately after the injury as a result of the irritative effect of the cerebral contusion or laceration, the presence of a depressed fracture, or the occurrence of a space-occupying complication. In the later stages, it results commonly from the healing processes, particularly when scar formation has occurred, or occasionally from the effects of a chronic sub-dural haematoma compressing the underlying brain.

In considering post-traumatic epilepsy, we are most interested in the demonstration of these irritative phenomena as the result of the presence either of cerebral or meningo-cerebral scars. As Penfield[9] has pointed out, the meningo-cerebral scar is composed of fibrous tissue (rather than glial "felting") resulting from mesothelial invasion of the damaged brain, largely from connective tissue elements in the surface vessels in the regions adjacent to the injured area, and consequently this type of scar is most liable to produce an epileptogenic focus. This is particularly true in the presence of a bone defect, for, when the dura has been lacerated, the underlying cortex may then be adherent to the skin and the continual traction resulting from cerebral pulsation may cause headaches, dizziness, and in the later stages, epileptic manifestations.

Williams[10] points out that the gradual development of the epileptic state can be observed by recording serial electro-encephalograms over a period of months or years after the injury, and the changes exhibited in these cases will be briefly summarized. Immediately after injury, the normal alpha rhythm, consisting of sinusoidal waves with a frequency of about ten per second and a potential varying between five and fifty micro-volts, is suppressed and replaced by slow waves of relatively high voltage dominating the whole record. Interestingly enough, these changes in general are the same as those produced by other forms of cerebral insult such as tumours and abscesses. As recovery proceeds, there is a transition from this slow-wave form to increasing frequency of slow waves with a diminution in their size, and they become progressively less persistent. Finally normal frequencies make their appearance and if recovery is complete, a normal electro-encephalogram results.

In the stage of concussion these abnormally slow waves may be recorded from all points on the hemisphere, but later they become more evident at the site of injury and contre-coup effects are easily recognized, occasionally the electro-encephalogram unmasking most unexpected areas of damage. Where the trauma has been slight, areas of suppression of normal waves around local injuries may be the only abnormality present, and this is particularly common in sub-dural haematomas. If symptoms persist after the injury, about 50% of the patients will show persisting abnormalities in the electro-encephalogram, and Heppenstall and Hill[11] suggest that when these persisting abnormal waves have a focus of origin, they have usually been caused by the injury, although when they are generalized, they may have been present before the injury, and indicate a constitutional defect in the subject. If post-traumatic epilepsy is to result, episodes of abnormal waves of a different kind may appear at a later date, gradually becoming more evident and more frequent, and finally a clinical fit may be observed occasionally in association with an episodic discharge of these abnormal waves. Interestingly enough, these episodic disturbances, which are the indirect result of the injury, arise sometimes in a zone of maximal brain injury, but more commonly, Williams[10] states, they are symmetrically placed in the frontal or parietal lobes without any special spatial relation to the injury. In the distinction of these traumatic epileptic manifestations from those of the idiopathic type, it is stated that foci of abnormal discharges are more frequent in the symptomatic without any special spatial relation to the injury. In the distinction of these traumatic epileptic manifestations from those of the idiopathic type, it is stated that foci of abnormal discharges are more frequent in the symptomatic or traumatic form and that generalized "wave and spike" complexes seem to be largely confined to patients who have had an epileptic tendency of very long standing, as in the idiopathic group, although Lennox[21] reports the occurrence of these slow patterns appearing in post-traumatic cases as 2 per second complexes of "spike and wave" type having a focal point of origin as compared to the generalized 3 per second "dart and dome" formation characteristic of idiopathic (genetic) petit-mal epileptic manifestations. Clinically, these late epileptic manifestations may occur at any time after three months from the time of injury and have been reported up to many years.

8. *Anosmia.*

This symptom is particularly likely to result in contre-coup injuries to the orbital surfaces of the frontal lobes for these lesions are commonly associ-

ated with rupture of the delicate olfactory filaments as they traverse the cribriform plate, with resultant loss of the sense of smell and a consequent impairment in the sense of taste. Recognition that this is a common sequel of blows in the occipital region may have considerable medico-legal importance.

TREATMENT OF SEQUELAE IN HEAD INJURIES

Primary evaluation of the relation of organic and psychic trauma

Evidences of a neuropsychiatric basis for the presenting symptoms	Evidences of an organic basis for the presenting complaints
Presence of symptoms typical of neuropsychiatric disorders	X-ray evidence of gross fracturing
Evaluation of:	Bone defects
(i) situational factors	Residual organic signs
(ii) personality predisposition	Increased intracranial pressure
	Ventricular dilatation
	Electroencephalographic changes
	Intellectual impairment

TREATMENT ALONG THREE LINES

Medical Treatment	Neuropsychiatric Treatment	Surgical Treatment
1. active management	1. optimistic outlook	1. rule out space-occupying lesions by L. P., craniotomy, ventriculogram (common example is a chronic subdural hematoma)
2. graduated exercises to restore tone and confidence	2. reassurance	2. repair of bone defects
3. early elevation of head and shoulders with tension exercises in bed	3. encouragement	3. subdural insufflation of air (or encephalogram) for meningeal or localized form of headache
4. early ambulation	4. explanation	4. late excision of cortical scars in post-traumatic epilepsy (provided symptoms not controlled by anti-convulsant therapy)
5. stooping and bending exercises	5. mild barbiturate sedation for extreme apprehension or anxiety	
6. stress on "occupational therapy merging into therapeutic occupation"		
7. re-education in organic deficits		
8. anti-convulsant therapy in post-traumatic epilepsy.		

In no other condition is there such difficulty encountered in evaluating the symptomatology in regard to its organic or functional basis, for, as we have already pointed out in our consideration of headaches and dizziness, these symptoms, if allowed to persist, are rapidly clouded by a gross functional overlay, particularly in patients who have a neurotic predisposition. Consequently the first step in the treatment is the studied evaluation of the relative effects of organic and psychic trauma in producing the symptoms, and this involves searching for evidences of organic lesions, and evidences to indicate a neuropsychiatric basis for the presenting symptoms.

As outlined in the accompanying table, evidences of organic lesions may be considered to consist of the following signs:

(1) X-ray evidence of gross fracturing of the skull.
(2) residual bone defects.
(3) residual organic signs.
(4) increased intracranial pressure.
(5) ventricular dilatation.
(6) electro-encephalographic changes.
(7) intellectual impairment.

On the other hand, evidences of neuropsychiatric symptomatology are found in the presence of symptoms typical of neuropsychiatric disorders and in a studied evaluation of the situational factors involved and the personality predisposition of the patient. In this connection, there is widespread agreement that a thorough examination of the family history and the pre-traumatic personality of the patient for evidences of liability to these neuropsychiatric disorders forms an indispensable basis for successful treatment, and if one finds that a patient with such a personality predisposition is faced with environmental conditions that require considerable effort on the part of the patient to make a satisfactory adjustment, the resultant conflict may readily precipitate functional disorders.

In assessing the personality predisposition, one is particularly interested in the following factors:

(1) A family history of nervous or mental disorders.
(2) Unhappy or unsettled family life during the formative years.
(3) History of maladjustment to school life (particularly truancy, and the avoidance of bodily contact in boys).
(4) Abnormal persistence of childhood fears and habits (particularly enuresis) occurring after the school age is reached.
(5) A history of being "high-strung" as a child, becoming readily excited and over-tired.
(6) A poor work history, prior to the time of injury.
(7) A history of hypochondriacal complaints.

Once the relative importance of these factors has been assessed, treatment can be carried out along three lines;—by medical, neuro-psychiatric, and surgical means. The relative stress placed on any one of these forms of therapy depends upon the opinion formed as to the causation of the symptoms.

I. Medical Treatment

Medical treatment forms the essence of therapy in all cases of acute head injuries and is aimed at the prevention of the development of a post-traumatic syndrome. Up until recent years, it had been considered that the best means of preventing the occurrence of headaches and dizziness after head injuries was the confinement of the patient to bed for many weeks after the injury, but recently it has been shown conclusively that this is not necessary and may in fact be harmful, in one whose pre-traumatic personality will respond poorly to the realization that he has suffered a serious injury. Consequently it is now the practice to institute a regimen of active management (Botterell and Wilson)[13] with graduated exercises to restore vasomotor tone and re-establish the patient's confidence in his ultimate complete recovery. These exercises are commenced as soon as the patient has no subjective complaints, and, generally speaking, the absence of headache is the most important and reliable guide on which to base the activity carried out by the patient.

The first stage consists of the early elevation of the head and shoulders, leaving the patient propped up on pillows for increasing lengths of time until he shows no tendency towards headaches, dizziness or black-out with these changes in position. He is then started on breathing and tension exercises in bed to regain normal tone and strength in postural muscles and prevent

pulmonary and vascular complications. When headaches and dizziness are no longer produced by these exercises in bed and have been absent for several days, the patient may be allowed up. This early ambulation is followed by a graduated set of exercises performed under supervision and again primarily directed at regaining normal vasomotor responses to changes in position, consisting largely of stooping and bending exercises as well as continuing the exercises designed at increasing the tone and strength of postural muscles.

At this stage it is important that the patient be not brought along too rapidly, and if at any phase of his increasing activity headaches or other symptoms recur, he should drop back one stage in the scheme of graduated exercises and continue for a sufficient length of time at that level until he can go on to the next stage without aggravation of the symptoms. Once he has become ambulant, stress is laid on occupational therapy designed ideally to suit the needs of the individual patient in an attempt to fit him for the occupation which he intends to pursue after complete recovery has occurred. Symonds and Lewis[14] have described this stage very neatly by the phrase "Occupational therapy merging into therapeutic occupation." At this time, specific re-education may be commenced for any organic neurological deficit which has resulted from local trauma to cortical tissue, and this may be the most trying stage for both the patient and the clinician, requiring as it does perseverance on the part of the patient and persuasion and firmness on the part of the physician. It is important to realize that persistent mental confusion, impaired memory, or other intellectual deficit, are not in themselves indications for restriction of the patient to bed, and, in fact, tend to improve more rapidly on an active regimen than when the patient is treated conservatively.

II. Neuropsychiatric Treatment

This form of therapy is an important adjunct to medical treatment and consists, as always, of the maintenance of an optimistic outlook at all times, active encouragement of the patient, and confident re-assurance when doubts arise as to the eventual outcome. The explanation as to the causation of symptoms when a functional overlay becomes apparent may in some cases result in dramatic improvement, the mere fact that the patient has an opportunity of discussing his fears and explaining his environmental conflicts to a sympathetic listener, often being sufficient to start him on the road to recovery.

If apprehension and marked anxiety are features of the post-traumatic picture in these patients, sedation with barbiturates accompanied by the therapy outlined above, offers the best means of promoting a normal emotional outlook.

III. Surgical Treatment

(1) *Space-occupying lesions.* Occasionally when post-traumatic amnesia or symptoms of headaches, dizziness or confusion persist for prolonged periods despite adequate medical and neuropsychiatric treatment, one must rule out the presence of a space-occupying lesion by lumbar puncture, and, if the intracranial pressure is elevated, by exploratory craniotomy and ventriculogram. The common example is a chronic sub-dural haematoma, which is often associated with localizing signs, and recently McConnell[16] has shown that collections other than blood are occasionally found in the sub-dural

space, and he has observed that, when these are drained a rapid and sustained improvement of the patient's mental state follows.

(2) *Bone Defects.* As we have already pointed out, defects in the skull are much more serious when the underlying dura has been torn and the brain is adherent to the scalp, as the continual pulsation of the brain results in traction on cerebral tissue in the region of a meningo-cerebral scar and may lead to the development of post-traumatic epilepsy, and consequently this type of defect should be repaired with a view to preventing the occurrence of epileptogenic foci. Occasionally the continual pulsation of the brain which is quite visible, particularly when the patient stoops or bends, may result in persistence of headaches and dizziness, presumably as a result of instability of the cerebral circulation, and in these cases symptoms are often dramatically alleviated by the closure of the defect.

Repair of the defect may also be indicated with a view to protection of the brain, particularly in young children or in adults in whom the head may be subjected to trauma by the nature of the work in which they engage. Undoubtedly a pulsating bone defect, of any size, in the frontal region is a distressing malformation and its repair is indicated both for cosmetic reasons and to prevent any psychological effects on the subject.

(3) *Localized Headaches.* If these headaches are thought to result from meningeal adhesions as Penfield and Norcross[3] have suggested, the sub-dural insufflation of air may be indicated in an attempt to break down the adhesions. Occasionally the performance of an encephalogram alone may be sufficient to cause improvement in this symptom although we have no experience in its use for this purpose as headache of this type has been extremely uncommon in our series.

(4) Rhinorrhoea. Rhinorrhoea may be immediate or delayed, and there has been considerable controversy about the treatment of this complication, some observers maintaining that the danger of meningitis is so great that surgical repair of the defects should be carried out as soon as possible, provided the patient has recovered from the initial shock state, whereas Verbruggen[16] on the other hand has recently stressed the value of postural drainage in an attempt to avoid the possibility of cerebro-spinal fluid that has escaped into the nasal passages becoming contaminated and then returned, in the presence of any blockage, to the clean cranial cavity with resultant meningitis. He points out that this complication is particularly apt to occur if there is any obstruction to drainage at this stage. He also suggests that defects on the posterior wall of the frontal sinus associated with rhinorrhoea may be closed by using the brain as a tampon, putting the patient with his head down and forward so that the brain covers up the slight tear in the dura and is made to adhere to the surface of the frontal sinus.

Our own experience with the efficiency of penicillin and sulfonamide therapy in combatting these post-traumatic meningeal infections has led us to adopt a reasonably conservative attitude, in that we believe an attempt should be made to obtain spontaneous closure of the defect before resorting to surgical treatment although it is readily admitted that occasionally the rhinorrhoea may recur after many months or years. If it persists after an adequate trial of conservative treatment and the patient has made an otherwise uneventful recovery, surgical treatment is usually carried out after three weeks or a month,

with closure of the defect using a patch of fascia lata, if it cannot be closed readily by ample suturing. The intradural approach is reserved for those cases involving the ethmoidal areas (usually posterior in position, and not easily accessible otherwise), this type of approach having the additional advantage of allowing inspection of both sides of the midline.

(5) *Post-traumatic Epilepsy.* Although general principles in the management of epilepsy may be outlined, it is important to realize that they must be individualized for each patient. Ideally, the treatment should be devised to prevent the whole seizure, or in other words, disturbances of consciousness and of the psyche as well as the involuntary muscular movement, and in addition should stop the convulsion without making the patient so stupid that he becomes a social outcast (Lennox)[12].

Effective medications are now available against all forms of post-traumatic epilepsy. Tridione has been used beneficially in the treatment of petit-mal attacks; and for psycho-motor episodes and grand-mal seizures, phenobarbitone or dilantin may be used; while in the cases more difficult to control, these anti-convulsant drugs may be used in synergistic combinations.

In the petit-mal attacks, treatment has until recently, been notoriously ineffective, phenobarbital being of only temporary or partial assistance in controlling the attacks and such adjuncts to therapy as a ketogenic diet and caffeine exerting a beneficial effect only in isolated cases. However, a new drug, tridione, (3, 5, 5—trimethyloxazolidine—2, 4 dione) has proven of value in controlling or substantially relieving two-thirds of the patients with epileptic symptoms characterized by alternate "spike and wave" forms in the electroencephalogram. In post-traumatic cases of petit-mal epilepsy, this change in the electroencephalogram usually appears as a petit-mal variant consisting of a two per second "spike and wave" pattern which is often focal in origin and represents the only pattern appearing in a routine testing which is distinctive of epilepsy. Lennox[12] advises a dosage of 15-30 gr. a day as required to control the symptoms and this is used regardless of the age of the patient. Certain toxic reactions have been observed including photophobia, skin rashes, symptomless leucopenia or eosinophilia (Greaves)[18] and aplastic anaemia with agranulocytosis (Harrison, Johnson and Ayer)[19]; and Mackay and Gottstein[20] but only one death has been recorded in 5000 patients under treatment and the drug would appear to be a valuable addition to our therapeutic armamentarium provided these manifestations of toxicity are kept in mind. Glutamic acid, in doses of 4-gr. three times daily, has also resulted in a gratifying increase in mental and physical alertness when used in conjunction with generally accepted forms of anti-convulsant therapy in these same types of petit-mal epilepsy (Price, Waelsch and Putnam[21] and Waelsch and Price[22].)

It has been the custom to begin the treatment of grand-mal attacks with phenobarbitone and only if this fails to control the seizures is sodium diphenylhydantoinate (dilantin) given. In beginning dilantin therapy, it is administered in the dose of 1½-grains three times daily, and then increased to a dosage sufficient to control the seizures. In difficult cases, dilantin, in doses varying between 3 to 10½-grains a day, may be given in combination with phenobarbitone in doses of 3/4 to 5 1/4-grains a day, and only in those cases which fail to respond to this form of therapy or show evidences of increasing severity of attacks, is surgical treatment indicated. This consists of an excision of the cortical and sub-cortical scar, care being taken not to remove adjacent

normal tissue particularly if the scar is adjacent to or involving a portion of the cortex known to subserve important functions.

Prognosis

The immediate prognosis as regards danger to life is based upon the classification of the patient as having suffered a major or a minor injury, depending upon the level of consciousness after the stage of concussion has passed. It is extremely grave in patients who are comatose or semi-comatose and particularly is this true if coma is associated with profound shock which fails to respond to resuscitation methods. If the patient survives the shock state but remains in coma or semi-coma for several days, the prognosos is also doubtful especially if the profound alteration of consciousness is associated with signs of failing circulation and irregularities in the respiratory rhythm. Signs at this stage indicative of brain stem involvement, such as absence of the corneal reflexes and a progressive hyperpyrexia, are also grave prognostic omens, particularly if they are associated, as Gray[23] has pointed out, with evidences of retinal haemorrhages to indicate either the severity of the acceleration injury or rapidly increasing intracranial pressure.

If these seriously-injured patients survive the first few days, the mortality is low, but they commonly show residual paralysis, dysphasia, or mental deterioration as pathological lesions involving functionally important structures have almost certainly occurred.

After the immediate acute stage, when the danger to life has been passed, the prognosis is based upon the factors to be enumerated below:

1. *Post-traumatic amnesia*:

It is generally agreed that the period of post-traumatic amnesia is the reliable guide to the severity of the head injury but it must be used with caution because a man with a brief period of post-traumatic amnesia may suffer prolonged disability from the effects of a local cerebral contusion resulting in an organic deficit requiring prolonged re-education. Guttman[24] states that the expectation of disability in post-traumatic amnesias of less than an hour is about four weeks. If the post-traumatic amnesia lasts up to 24-hours, the expectation of disability is about six weeks, and if it extends to seven days, the disability may last for eight to nine weeks, and this is in general agreement with the figures quoted by other observers. Russell[25] states that 80% of patients return to work within six months, and adds that in any large group severe enough to be admitted to hospital, symptoms may persist for a year or longer but are rarely disabling for longer than six months. This period of post-traumatic amnesia is measured from the moment when the patient becomes continuously aware of, and able to remember, his surroundings, and is of value in prognosis because it shows no alteration with a lapse of time, whereas the retrograde amnesia tends to shrink progressively as recovery occurs and consequently has little prognostic value..

2. *Personality Predisposition*:

Symonds[26] states that evidences of liability to psychological disorders is an indispensable guide to prognosis as the individual with a neurotic predisposition is more apt to suffer prolonged disability because:—

(1) the effects of the injury upon his brain are more apt to impair his capacity for psychological adjustment, and:—

(2) he is initially less well equipped for making adjustments to the psychological trauma of his accident, its effects upon his life situation, and whatever physical disability may have resulted.

3. *Ventricular Dilatation*:

Davies and Falconer,[27] in summarizing their findings, state that the frequency with which ventricular dilatation is found, increases with the severity of the trauma, usually appearing within two or three weeks of the time of injury and reaching its maximum within a month. The ventricles were usually dilated in cases with marked intellectual and mental deterioration (21 of 24 cases) and in contra-distinction were usually normal when the disorders were of a subjective character. In addition, cases with residual pyramidal signs frequently showed a significant dilatation of the ventricle on the appropriate side. However, post-traumatic epilepsy did not appear to be connected with changes of the ventricular outline. Although the findings of air encephalography cannot be used as a certain means of differentiating between cases with genuine disability and those with functional disturbances, the changes are sufficiently well marked and constant in occurrence to be important supportive evidence in cases of post-traumatic intellectual deterioration and in those cases with organic clinical signs.

4. *Electro-encephalography*:

As has already been pointed out in our consideration of post-traumatic epilepsy, serial changes in the electro-encephalogram, in which foci of abnormal discharges, occurring in an episodic manner, appear, definitely indicate that the patient is liable to the occurrence of post-traumatic epilepsy. It is when these persisting abnormal waves have a focus of origin that one can state in all probability that they have been caused by the injury. As Symonds[26] points out, although post-traumatic amnesia is an index of a general disturbance of the brain, local brain damage is the most important factor in the development of epilepsy, and the electro-encephalographic changes may give the first evidence of these local contusions or lacerations.

Other contributory signs suggesting the possibility that epilepsy may result are skull fractures (local ventricular dilatation is particularly apt to occur deep to a fracture of the skull), a prolonged period of post-traumatic amnesia, and persistence of signs and symptoms for an abnormally long period after the injury. In these latter cases, when symptoms persist after the injury, about 50% of the patients will show abnormalities in the electro-encephalogram indicating an organic basis for the complaints (Heppenstall and Hill)[11].

5. *Prognosis in Specific Lesions*:

(1) *Blindness.* Blindness usually occurs as an immediate effect of the injury and examples of delayed blindness are extremely infrequent. The causative lesion is a contusion of the nerve with compression due to haemorrhage beneath its sheath, and rarely is there any evidence of direct compression of the nerve as a result of a fracture at the region of the optic foramen. Consequently, therapy is expectant and if useful vision is going to be recovered, improvement starts about the third or fourth day and progresses rapidly

(Turner).[28] In all probability, full recovery never occurs, with residual scotomata or defects predominantly in the peripheral visual field.

(2) *Anosmia.* Anosmia is a common occurrence in severe cases of head injury and the prognosis for recovery is very bad. On the other hand, cases showing parosmia (distortion of the sense of smell) usually have a favourable outcome (Leigh)[29]. In cases where the taste is disturbed, it is usual for the primary tastes to be preserved while the appreciation of flavours is lacking.

(3) *Paralysis of Ocular Muscles.* Fortunately persistent diplopia is extremely uncommon after head injuries and when present responds well to orthoptic treatment or operations on the ocular muscles in refractory cases. Lyle[30] considers that if diplopia following head injury has not recovered spontaneously within four months, it is unlikely to do so, and surgical treatment is indicated to correct the ocular deviation by appropriate operation upon one or more of the extrinsic ocular muscles.

(4) *Facial Paralysis.* Facial paralysis may be immediate or delayed. The etiology is assumed to be a laceration of the nerve or an intraneural vascular accident in the case of immediate lesions, while pressure of blood on the nerve in the aqueduct of Fallopius is likely to be present in the delayed cases (Turner)[31]. Both types are probably associated with fractures of the petrous temporal bone, although this may not be demonstrable radiographically.

The prognosis in the delayed cases is good and the treatment recommended in all cases is expectant, with facial splintage, electrical stimulation and facial exercises during recovery. If no improvement occurs in three to six months, one may explore and decompress the nerve in the Fallopian canal, and if a nerve suture is found necessary, this may be carried out without tension by re-routing the nerve or performing a nerve graft (Lathrop)[32].

Summary

1. The sequelae of acute head injuries are briefly described with a general consideration of their recognition and evaluation.
2. An outline of treatment is presented emphasizing the advantage of active management during convalescence, and the values of combining medical, neuropsychiatric and surgical methods of therapy once the symptomatology has been carefully assessed with regard to its functional or organic basis.
3. The clinical findings of aid in prognosis are discussed with emphasis on the importance of determining the nature of the patient's pretraumatic personality and intellect.

REFERENCES

1. McKenzie, K. G.—One aspect of the post-traumatic syndrome in craniocerebral injuries—Surg. Gynec. & Obst., 77, 631, 1944.
injuries—Surg. Gynec. & Obst., 77, 631, 1944.
2. Rowbotham, G. F.—Acute Injuries of the Head: Their Diagnosis, Treatment, Complications and Sequels. G. & S. Livingstone Ltd., Edinburgh, 1945.
3. Penfield, W. and Norcross, N. C.—Subdural traction and post-traumatic headache: Study of pathology and therapeusis, Arch. Neurol & Psychiat., 36, 75, 1936.

4. Lehmann, H.—Post-traumatic confusional state treated with massive doses of nictonic acid. Cand. M. A. J., 51, 558, 1944.
5. O'Connell, J. E. A.—The Vascular Factor in Intracranial Pressure and the Maintenance of the Cerebrospinal fluid Circulation, Brain, 66, 204, 1943.
6. Reynell, W. R.—A psychometric method of determining intellectual loss following head injury. J. Ment. Sc. 90, 710, 1944.
7. Ruesch, J.—Intellectual Impairment in head injuries. Am. J. Psychiat. 100, 480, 1944.
8. Ross, W. D. and McNaughton, F. L.—Chronic post-traumatic head symptoms, Canad. M. A. J., 53, 12, 1945.
9. Penfield, W.—The Mechanism of Cicatricial Contractions of the Brain, 50, 499, 1927.
10. Williams, D.—The clinical application of electro-encephalography. Brit. M. Bull., 3, 613, (p. 18), 1945.
11. Heppenstall, M. E., and Hill, D.—Electroencephalography in chronic post-traumatic syndromes, Lancet, 1, 261, 1943.
12. Lennox, W. G.—The petit mal epilepsies: their treatment with tridione, J.A.M.A., 129, 1069, 1945.
13. Botterell, E. H. and Wilson, K. E.—The Active Management (non-operative) of Cranio-cerebral injuries. Canad. M. A. J., 51, 498, 1944.
14. Symonds, C. P. and Lewis, A.—Discussion on Differential diagnosis and treatment of post-concussional states. Proc. Roy. Soc. Med., 35, 601, 1942.
15. McConnell, A. A.—Prolonged Post-traumatic Amnesia: Findings at Operation Lancet, 1, 273, 1944.
16. Ver Brugghen, A.—Head Injuries. J. Iowa M. Soc. 34, 225, 1944.
17. Lennox, W. G.—The Pharmacopeia and the Physician. Second Series. Am. Med. Assoc. Chicago, 1943, pp. 148-171.
18. Correspondence. Greaves, R. J.—Sensitivity to tridione, J.A.M.A., 132, 44, 1946.
19. Harrison, F. F., Johnson, R. D., and Ayer, D.—Fatal aplastic anemia following use of tridione and hydantoin, J.A.M.A., 132, 11, 1946.
20. MacKay, R. P. and Gottstain, W. K.—Aplastic anaemia and agranulocytosis following tridione, J.A.M.A., 132, 13, 1946.
21. Price, J. C., Waelsch, H. and Putmam, T. J.—dl-Glutamic acid hydrochloride in treatment of petit mal and psychomotor seizures, J.A.M.A., 122, 1153, 1943.
22. Wealsch, H. and Price, J. C.—Biochemical aspects of glutamic acid therapy for epilepsy. Arch. Neurol. & Psychiat., 51, 393, 1944.
23. Gray, C. C.—Presented at the Inter-Allied Conference on Head Injuries, London, Eng., March, 1945. Paper in print.
24. Guttmann, E.—The prognosis in civilian head injuries. Brit. M. J., 1, 94, 1943.
25. Russell, W. R.—After-effects of head injury. Tr. Med. Chir. Soc., Edinburgh. In Edinburgh, M. J., 41, 129, 1934.
26. Symonds, C. P.—Prognosis in closed head injuries, Brit. M. Bull., 3, 612, (p. 14), 1945.
27. Davies, H. and Falconer, M. A.—Ventricular changes after closed head injuries. J. Neurol. & Psychiat., 6, 52, 1943.
28. Turner, J. W. A.—Indirect injuries of the optic nerve, Brain, 66, 140, 1943.
29. Leigh, A. D.—Defects of smell after head injury, Lancet, 1, 38, 1943.
30. Lyle, T. K.—Some cases of post-traumatic diplopia following head injury and their treatment. Tr. Ophth. Soc. U. Kingdom, 61, 189, 1941.
31. Turner, J. W. A.—Facial palsy in closed head injuries: the prognosis, Lancet, 1, 756, 1944.
32. Lathrop, F. D.—The repair of traumatic lesions of the facial nerve secondary to war wounds, S. Clin. North America, 26, 763, 1946.

Some Thoughts on Abortion

H. B. Atlee

FOR a good many years now about one-quarter of the cases coming into my service at the Victoria General Hospital have presented one or another of the various phases of abortion. It seemed therefore that the brethren might be interested in certain conclusions I have arrived at as a result of my experience with this common, and often trying condition. Particularly so, since modern endocrine investigation has so profoundly affected our views concerning its causation and treatment.

Let me first of all describe briefly the generally accepted theory governing the endocrine control of pregnancy. When a woman gets in the family way the corpus luteum continues to develop, producing a hormone, Progestin, which supports the developing ovum and produces all those mighty changes that occur in the organs of reproduction. But as the need for progestin in large quantities becomes greater and greater, the corpus luteum is no longer able to cope with the problem and in a relatively short time turns it over to the placenta which henceforth becomes the really outstanding manufacturer of the substance. This handover appears to take place somewhere around the 80th day. Unfortunately, in some cases there seems to be a certain amount of buck passing as between the corpus luteum and the placenta, with the result that neither supplies the proper quantity of progestin—the level of which drops seriously in the bloodstream—with the consequent onset of abortion, which may only be a threat or may go on to inevitability. While there are still some unexplained inconsistencies in this theory, it is, as I have outlined it, the one under which we now work. In other words, abortion occurs in the great majority of cases, perhaps in all cases, as a result of some factor inhibiting the secretion of corpus luteum hormone.

Prior to the enunciation of this theory, we thought that such conditions as retroverted uterus, uterine congestion and endometritis (whatever they are), general diseases, and such accidents as falling downstairs, were directly responsible for abortions. Probably thousands of retroverted uteri have been suspended and tens of thousands of D's and C's done for this reason. But we now more or less agree that the real trouble lies somewhere 'twixt the hypophysis and the placenta (via the ovary). While we see that physical accidents still appear to produce abortions, we hold them to do so not so much as a direct result, but because of some inhibiting effect they have on the production of progestin. Probably the actual accident itself is not so important in bringing about this inhibition as the emotional upset that goes with it. There seems little doubt that emotional stresses have a very real effect on such endocrine-dyscrasia-produced conditions as dysmenorrhea, amenorrhea and menorrhagia—why not therefore on pregnancy, which is the very child of the endocrines? So goes the theory.

Its impact on the purses of those suffering from abortion has been terrific. For with a theory so clear and beautiful, what more logical than to treat all threatened and habitual aborters with corpus luteum hormone? If the placenta won't take over from the corpus luteum, why not substitute with a hypo full of gonad, and so become—even more than we are at present—little brothers of God? That's exactly what we have been—and still are—doing. Corpus

luteum hormone is an expensive ichor—in the heyday of my enthusiasm for it I once ran up a bill of $64.00 for one unhappy husband. Is it worth the money?

There seems to be a difference of opinion on this point. On the one hand there are those who feel that corpus luteum hormone is a valuable and useful biological which has definitely lowered the number of habitual and inevitable aborters. On the other, there are those who, as a result of a fairly long trial, are unable to satisfy themselves that it does much good, but continue to use it because they don't know what else to do. I must confess that for some time I have believed and acted with the second group. I do not think that progestin has much, if any, effect on the aborting woman, but I still give her the stuff because I haven't yet achieved the moral courage to withhold it. Let me say, however, that I have almost worked up to having the courage of my convictions, for having used progestin in very large doses over a good number of years, I can produce no real evidence that it has lowered the incidence of habitual or inevitable abortion in my practice.

What about Vitamin E? This looked like another hopeful line of therapy. Here was the vitamin that stimulated the growth of germ cells; here surely was the answer to the maiden's prayer. But with regard to this preparation I really have achieved the courage of my convictions, and no longer use it. For years I gave it—as Wheat Germ Oil: as Ephynal—in small doses and in large. I ordered it fresh from the manufacturer, and prescribed it stale off the drugstore shelf. It made no difference what preparation I gave or in what dose I gave it. I therefore state quite definitely, dogmatically, categorically —other authorities to the contrary notwithstanding—Vitamin E is napooh. Use Wheat Germ Oil for your salad dressing, to lubricate your sewing machine, or to rub into your sore back—but you'll stop no woman aborting with it.

Let me illustrate one vista of my scepticism with regard to progestin and Vitamin E by describing what I might designate as an abortional holocaust that occurred in my private practice about six years ago. I had thirteen cases of inevitable abortion in a row in nine threatened and four habitual aborters. Every single one of those cases aborted despite fresh Wheat Germ Oil in doses rising as high as 16 drams a day, and proluton in 10 mgm. intramuscular dosage every day during the critical period. The habitual aborters were started on the Vitamin E as soon as they got pregnant, and were given from the beginning of pregnancy two doses of the proluton a week: the moment they showed signs of aborting the dosage was increased as indicated above. It was this phenomenal—and rather unusual—run of bad luck which first dampened my boyish enthusiasm, and led me—even though I have continued the same treatment subsequently on other cases—to do so with my tongue in my cheek, and an Oh Yeah? in my heart.

With regard to the treatment of habitual abortion I am getting back to where I was 10 years ago. Build up the patient's and her husband's health before she gets pregnant. When she does get pregnant—quiet living, mentally as well as physically—*and no coitus* (if you can call that living)—with as much time resting as she can manage for the first four months, and half a grain of phenobarb two or three times a day over the same period. When threatened with abortion: *Complete* bed rest while there is any bleeding, phenobarb grs. ½ t.i.d. for two weeks, and morphia or pantopon if there is any uterine pain.

Along with motor cars and radios abortion is going to be cheaper in 1948 . . .

Some of you may remember that I rather startled the local pundits twenty years ago by having my obstetrical patients do bed exercises, starting on the day after labor, and getting them up walking to a chair two days after that. Having satisfied myself on the evidence of above 2000 cases that God was going to strike neither me nor my laboring women dead for this ghastly heterodoxy, I proceeded to put the experience to work on those of my abortion cases that had become complete. Previous to that I had usually kept them in bed for about a week, which was the fashionable thing to do. But why, I asked myself one day, do one thing in one hospital for a full term labor, and another thing in another hospital for a mere abortion? So I started getting my complete aborters out of bed the day after they became complete and discharging them from hospital the day after that. In short, I adopted the policy of keeping them moving. Here, too, nothing serious happened. Occasionally, I would get—and still do—a patient banged back at me because she was bleeding again—which meant, of course, that there was still a fragment of placenta in the uterus. But this will happen if you keep your aborters in bed for a month. But what did I find, apart from this exception? That I had practically got rid of all my post-abortal complications! In other words, these aborters do better if you keep them up and about where their moving muscles and upright position encourage drainage from the uterine cavity and prevent their leg veins from getting thrombosed.

A year or so after starting this procedure, I read an article by a Russian gynecologist, describing their experience in that country during the epoch when abortions were legalized. You'll remember that this used to be quite comme il faut in Soviet land before communism froze out into Stalinism. According to this authority, they found that if, in doing their abortions, they followed three rules, they cut their post-abortal complications to a minimum. These were the rules: (1) Return the woman to her work immediately and without bed rest (thus confirming what I had already discovered for myself); (2) Give no anesthetic for the emptying of the uterus. (3) Do not pack the uterus unless absolutely forced to do so.

Having seen the value of (1) above, I decided to introduce into my policy in so far as I humanely could, emptying the uterus in cases of inevitable or incomplete abortion without giving an anesthetic. To my surprise I found, as time went on, that this was possible in a surprisingly large number of cases that previously I had anesthetized, and possible with very little discomfort to the patient. What happens in the average case that needs emptying is this: By the time there is so much bleeding that something requires to be done, the cervix is fairly well dilated and the placenta, or a large part of it, is either sticking through the cervix or lying in the lower uterine segment where it can easily and safely be grasped by an ovum forceps. Furthermore, in a large percentage of abortions with temperature, the temperature is raised because the placenta, or some part of it, is plugging the cervical canal, thus damming back into the uterine cavity secretions loaded with sapremic organisms. It is absorption from this dammed back secretion which causes the temperature. But if the plug of placenta is removed from the cervix with an ovum forceps, drainage will be re-established and the temperature fall to normal, even though

some placenta still remains attached higher up in the uterine cavity. As long as drainage is established it is not necessary to go up after this other fragment—indeed, it is safer to leave it there for the time being.

Let me make the following other brief points:

1. If, in a bleeding case, you remove a piece of placenta from the cervix or lower uterine segment and the bleeding stops, it is not necessary to do any more even if you can feel with your finger another piece of placenta high up in the uterine cavity. This still-retained fragment is quite likely to come away by itself, or to be pushed down later into the cervix so that you can get it more safely.
2. But if, despite removing a fragment from the cervix, the patient continues to bleed, it is better to then give an anesthetic and thoroughly explore the uterine cavity with finger and ovum forceps.
3. Do not pack unless, despite apparent emptying of the uterus, the woman is still bleeding—and pack only under an anesthetic; it can't be done properly without an anesthetic.
4. And let me repeat: Don't do anything more for a septic abortion than remove the placental plug from the cervix without anesthetic. If there is no placental plug in the cervix *keep out of that uterus.*

I find that one of the most difficult things to determine early is whether or not the uterus is empty. As a rule one can depend on three signs: (1) There is a daily lessening in the bleeding or lochia. (2) The cervix closes and becomes hard. (3) The uterus feels small and hard. But sometimes, with all these signs present, the woman will later begin to bleed more than she should and on curetting her it will be found that she has a small fragment of placenta still attached. I have done a vaginal hysterectomy four years after a miscarriage for menorrhagia and found the cause to be a still-attached fragment of placenta, the size of the tip of your little finger. On the other hand I have examined patients four days following a complete abortion, that was later proved to be complete, and found a large amount of lochia, a large, boggy uterus, and a patulous cervix into which I could stick my forefinger as far as the internal os. But two things are certain: (1) If the uterus is empty, the lochia will eventually settle down, the cervix close, and the uterus involute. (2) If the uterus is not empty, the patient will develop menorrhaia or metrorrhagia that will require exploration of the uterine cavity. But the practical thought that comes out of all this is that one should not be in too much of a hurry to explore what may seem to be a still-occupied uterine cavity. Give things time to settle down for a week or two. In the meantime fluid extract of ergot in half dram doses three or four times a day is often a help.

Penicillin, of course, has been a great boon in the handling of seriously infected abortions. The important thing is to give it early in effective doses and keep it up until the critical temperature period has passed. I do not believe, however, that it is necessary always to give it until the temperature is flat. Often a seriously infected case settles down from a high temperature to one that sawtooths up to 100° for a week or more thereafter. In such a case—I have one in my wards now—the penicillin does little good after the first few days, and to continue its use is to waste it.

Every aborter whose hemoglobin has dropped to 60 or lower, whether as a result of hemorrhage or infection, should be given blood transfusions. We tend to think of transfusions only where there has been actual bleeding; it is surprising how rapidly the red blood cell count and hemoglobin index will fall in a seriously infected case where the infection keeps up despite penicillin. Every woman who has had a pelvic infection for a week and is still febrile, should have her Hb. index done and her red cell count taken. Until we got penicillin I saw more seriously-ill septic abortions cured by repeated blood transfusions than by any other therapy. The fact that we now have such a valuable remedy in penicillin doesn't mean that blood transfusions have lost their usefulness in septic abortions.

I do not believe that sulphonamides have much place to-day in the treatment of septic abortion. Nor was I ever able to satisfy myself (in the days before penicillin) that these sulpha drugs were of value in this connection. In my sinful youth I often gave them to seriously infected abortions in huge doses. I doubt if I ever accomplished anything but add their toxin to that already floating in the patients' bloodstreams. To-day I do not use them at all in the treatment of infected abortion.

May I wind up by restating—for the sake of emphasis—two of the points I have already made:

1. It is highly doubtful if either Vitamin E or Progestin (in their present available form) is of any value in habitual or threatened abortion.
2. A woman who has an abortion will return to full health quicker, and be more likely to escape complications, if (1) she is not kept in bed but put on an active regime immediately, (2) her uterus is emptied wherever humanely possible without an anesthetic, and (3) her uterus is not packed.

Aerosol Penicillin in Upper Respiratory Infections in General Practice

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IN the following observations it is hoped to point out that the administration of penicillin by an extremely fine mist or nebulization, with oxygen as a vehicle, is a reliable, efficient and readily available method of administration.

An added feature of the arrangement is that in many pneumonias, it obviates the annoying needle q.3.h.—and on the other hand, it is rather a pleasant, refreshing agent of medication. It saves the practitioner many added trips to the home.

In certain respiratory diseases it is undoubtedly far superior to the usual parenteral avenues. In this respect it deposits the calculated amount of penicillin at the site of the pathology. In this fashion it acts not only locally, but also in a more remote effect by absorption into the blood stream and lymph channels.[1]

The work of British investigators proves that normal atmosphere can carry per litre, 35 units of penicillin by aerosol impregnation. They also show that upon section there is a very considerable concentration of penicillin found even in the peripheral lung,—as much as 2 units per gram. They also show that after 15 minutes of aerosol penicillin, nose, throat, and sputum tests are all loaded with penicillin itself and produce no growth of penicillin sensative organism.[4]

It is to be noted that in the virus pneumonias, it limits the deadly cross and mixed infections of penicillin susceptible organisms which are usually the killing agents.[3] At the same time, a flow of pure oxygen permeates the penicillin resistant virus. The course of the disease must be favourably altered under these conditions.

Along this same line of thought, it has been proven that it is possible to maintain a high blood level of penicillin by inhalation alone. It can be raised to 0.05 units per c.c. by the inhalation of 20,000 units[1] by a man weighing 180 lbs. Actually, any desired blood level may be obtained by aerosol method if more or less frequent inhalations be prescribed.

In extensive investigation it has been definitely proven that the deposition of penicillin in the lung tissue by aerosolization is safe.[1]

Dr. H. N. Vermilyne (N. Y.) in 1946[1], employing the method of aerosol penicillin as outlined by A. L. Barach, in 1945[2], gives a report on over 200 cases in which various types of respiratory conditions obtained. Extremely satisfactory results were noted. This paper deals also with the direct and indirect results observed on such associated conditions as extrinsic allergies, e.g.—asthma, migraine, eczema, etc., consequent upon chest, sinus and tonsil infection. We feel we want to limit our remarks to the chest infections, where the results observed were certainly more consistent and undoubtedly more dramatic.

Technic and Dosage

The apparatus consists of an oxygen tank (large for office, small for more

remote administration), a Heidbrink regulator to gauge oxygen flow in liters per minute, an atomizer (nebulizer) and a simple rubber mask with rebreather bag. The cost of these items, without the oxygen, is about $65.00.

The calcium salt is generally accepted at present due to the more favourable odour it possesses. It is diluted with saline so as to contain 20,000 units per c.c. (A purely aqueous solution is irritating to membranes.) This amount is placed in the nebulizer and the mask strapped to the face. By simply regulating the flow of oxygen to about 4 l. per min., there is produced a steady flow of penicillin-mist laden oxygen which proceeds to the mask and so to the respiratory tract. Deep breathing is employed and it is advised to hold the inspiration to facilitate deposition of the penicillin particles, then gently exhale.

In ambulatory patients such as those with bronchiectasis or unresolved pneumonias, office treatments are usually given four to six times daily at three or four hour intervals. One treatment (1 cc. =20,000 Units) lasts from 20-30 minutes. The soft rubber mask permits of easy adjustment to the face and many patients read while taking the treatment. In the home, treatment isgiven q. 3-4 h.

Case Reports

I shall not enumerate a tiring series of cases. One of pneumonia and one of unresolved pneumonia will typify the average case of each.

Acute Pneumonia—April 15, 1946.

Mr. C. 50 years. Male. White. Brought to my office. Overwrought, apprehensive. Dry irritating cough. T. 103.4. P.—120 min. Physical exam ination revealed moderate dullness over lower right lobe. Some fine rales- and bronchial breathing present. Fleuroscopically—area of consolidation at base, about three fingers breadth. Leucocyte count 20,000. Was given 40,000 units penicillin by inhalation and sent home. Small tank of oxygen set up and 20,000 units administered q.4.h. Upon second day, cough had disappeared, T. 99.6, P.—80, felt excellent. In five days patient insisted upon sitting out. The return to normal was uneventful and complete.

Unresolved Pneumonia—July 15, 1946.

Mrs. S. White, 35 years. Pale, tired individual. History of influenza seven weeks previously. Persistent, tiring cough since that time. Marked lassitude, periods of perspiring, weakness and general malaise. T.—98. P.—80/min. No specific history of tuberculosis or pleurisy. Chest plate taken and the following report from the Sanatorium in due course.

Left Lung—Descending trunks are accentuated. Peri-bronchial parenchymal changes from 5th to 7th rib, mid and inner zone. No cavity.

Right Lung—Descending trunks are accentuated. Suggestive parenchymal changes in association with them opposite 5th interspace, inner zone. No cavity.

Summary—Suggestive of bronchiectasis. Advise bronchograms of both lower lobes.

In the meantime the patient had been given aerosol 20,000 units q.1.d.

On the second day the seven week old cough was greatly retarded. On the third day the tiring, unproductive thing was gone. Treatment was continued for 5 days, when the patient had to go out of town.

She returned in two weeks, well, rested and putting on weight. A plate was taken some days later and the Sanatorium reports:

Right—As compared to your previous film, improvement has occurred. This lung is now normal.

Left—Improvement. Previously noted peri-bronchial changes are not seen. This lung is now normal.

Summary—Both lungs are now normal.

Side Effects

I observed but one. A marked uiticaria, entire body, 10 days after initial treatment. This responded promptly to Benadryl.

Barach and Vermilyne report that if aerosol is given a few hours after pollen immunization or vaccine, severe allergy manifestations appear. These may be characterized by urticaria, severe nervousness, abdominal pain or angioneurotic oedema. Isonipecaine 50 mg. controlled the situation within a few minutes. A few complained of substernal soreness. A few complained of sore throats during treatment.

Comments

Aerosol penicillin is not claimed to be an all-encompassing curative measure.

In home cases it is time-saving, pleasant, efficient.

It has, certainly, logical application to upper respiratory infections.

It has saved some patients what would have been more extensive and more expensive investigation.

It deserves a wide trial by the general practitioner as a not unduly great investment. On the other hand, it possesses an unusually comprehensive scope in its uses—and promises.

It has proven of great service to the writer in the past year in general practice.

REFERENCES

(1) Vermilyne, H. N.—Aerosol Penicillin, I.A.M.A., Sept., 1945.

(2) Barach, A. L.—Prelim. Report. Ann. Int. Med.—April, 1945.

(3) Cecil, R. L.—Text. Med. Ed. 6—Saunders Co., 1943.

(4) Knott and Southwell—Archives of Disease in Childhood, March, 1946; Digest Treatment, January, 1947.

DOCTOR WANTED

The community of Wallace and surrounding districts is in need of a resident doctor. Any doctor interested in settling there should kindly communicate with Mr. J. M. Charman, Secretary of the Citizens' Committee, Wallace.

A Case Report

Von Jaksch's Syndrome in Congenital Syphilis

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IN 1889 von Jaksch described a syndrome of early childhood consisting of a severe anemia associated with splenic, hepatic and lymph gland enlargement and a high leucocytosis. This has been known as "von Jaksch's anemia.'. There seems to be little unanimity of opinion, however, concerning the nomenclature, etiology and pathogenesis of this anemia and there is much controversy over the question of whether or not this syndrome represents a distinct disease entity. It has been found that the signs described by von Jaksch may be caused by a variety of conditions, including rickets, congenital syphilis, infection, under-nutrition, iron deficiency, or even the initial stages of true leukemia. Consequently, there is a tendency on the part of many American writers to discard entirely the term "von Jaksch's anemia" while British authorities tend to restrict its use to a very limited group of cases which fulfil certain criteria and which they consider comprise a distinct disease group. Their criteria are not exacting but embrace all cases within the age group of six months to three years, exhibiting a chronic anemia of haemolytic type and the von Jaksch symptom—complex, and distinguishable from Cooley's anemia in their rarity, their tendency to recovery in many instances, and their appearance in Anglo-Saxon people. It is thus apparent that the term "von Jaksch anemia" should be employed, if at all, only rarely in diagnosis. The term "von Jaksch syndrome," on the other hand, is still not infrequently used in a broad sense to denote a "clinical state" with no implication intended regarding the exact underlying diagnosis. This clinical state is not infrequently seen in infancy and early childhood and presents the original von Jaksch symptom—complex of a severe leuko-erythroblastic anemia, splenemagaly, hepatomegaly and high leucocytosis.

The case to be reported here presented this "clinical state" and, in addition, a marked jaundice, thus making it an interesting diagnostic problem.

Case Report

C.B.—a white, female infant was born on June 22, 1946, at the Grace Maternity Hospital, Halifax, N. S. Her mother had been admitted to hospital only eight hours previously so that little time had been afforded for appraisal of her general condition. However, her past history, pre-natal history, and functional enqjiry had been essentially negative including a denial of any knowledge of venereal disease. Her physical examination had revealed a thin, pale, poorly-nourished, primiparous woman with no apparently significant physical abnormalities other than this general malnutrition and the presence of multiple perineal condylomata. The foetus appeared small but otherwise normal, and labor was progressing satisfactorily.

At delivery, however, this infant was in very poor general condition. It was asphyxiated and resuscitation was very difficult. It was premature weighing three pounds, eleven ounces. Its skin and sclerae were markedly

icteric and the body was covered with numerous petechiae and small ecchymotic blotches. The abdomen was distended and tense. The liver was enlarged to the level of the umbilicus and the spleen was markedly enlarged, reaching the level of the anterior superior iliac spines. In spite of incubation, oxygen therapy, subcutaneous Rh-negative blood transfusion and other supportive measures the infant rapidly deteriorated, the jaundice and purpuric manifestations progressed and the child finally expired forty-five hours after birth. During its short life, the case was investigated as extensively as possible. The mother's history was carefully rechecked but no history of previous blood transfusions or pregnancies was obtained. On the other hand, she did admit that she had been an infrequent attendant of the Dalhousie Public Health Gynecological Clinic. The records of that institution disclosed that she had reported there on three occasions, twice in February, 1946, and once in May, 1946. On the first occasion she received local treatment for perineal condylomata and at this time investigations for gonococcal and leutic infections were negative. On the second occasion she received further local therapy but this time her blood Kahn test was positive (three plus) and the uretheral and cervical smears were again negative for gonococci. On her third attendance, anti-leutic therapy was instituted (her initial treatment consisting of 0.04 gms. of mepharsan and 1 c.c. of bismuth subsalicylate) and she was advised to attend the pre-natal clinic for pre-natal care and further anti-leutic treatment. This she failed to do. In addition, no history of her receiving anti-leutic treatment elsewhere was ascertainable.

Laboratory data included:

(1) *Rh factor*—This was positive in both the mother and the infant and the mother's blood was negative for anti-Rh agglutinins.

(2) *Blood picture*—This was as follows:

The Red Cell Count	3,840,000 per cu. mm.
Haemoglobin	80% (11.7 gms.)
Colour Index	1.04
The White Cell Count	40,400 per cu. mm.

Stained Films

The Red Cells reveal a marked polychromasia with anisocytosis. The average size of the red cells appears to be above normal. Many nucleated red cells are noted.

Differential Schilling Count (200 cells counted)

Myeloblast	1.0%
Myelocytes	4.0%
Juveniles	6.0%
Band Forms	7.0%
Polymorphs	34.0%
Lymphocytes	6.0%
Menocytes	0.0%
Eosinophiles	1.0%
Erythroblasts	11.0%
Normoblasts	30.0%

(3) *Serology*—Wasserman and Kahn tests were strongly positive (four plus) in both the mother and the infant.

The post-mortem findings were as follows:

Anatomical Diagnoses

1. Congenital Syphilis.
   (a) Acute Luetic hepatitis.
   (b) Leutic Interstitial Nephritis.
2. Extra medullary haematopoeisis in Liver and Spleen.

Post-mortem Findings

General Appearances: small, thin, white female infant of premature appearance with the skin stained a deep yellow in colour and flecked with numerous small petechiae and ecchymoses.

Thorax:

Pleural Sacs: no fluid or adhesions.

Lungs—no gross pathology, well aerated, bile-stained colour.

Trachea and Bronchi—nil.

Heart—normal size and shape, valves healthy, no congenital abnormalities, aorta healthy but deeply bile-stained.

Abdomen:

Liver: markedly enlarged down to level of umbilicus, deep yellowish colour, very firm and fibrotic to the feel and on cutting the normal surface markings were lost due to deep bile staining.

Gall-bladder: normal size, contained a very pale, thin watery bile.

Spleen: markedly enlarged down to level of iliac crest, deep red colour, moderately firm. On section it had a homogeneous glistening appearance. The malpigian bodies were not seen.

Stomach and Intestine: nil. Bile stained. No glands in mesentery.

Kidneys: normal size with foetal lobulation; bile stained; capsule stripped easily; no haemorrhage. On section, multiple small petechial haemorrhages were scattered along the margin between medulla and cortex and also along the lateral borders of the medullary pyramids.

Adrenals: nil.

Bladder: nil.

Head and Neck: brain not examined.

Bone: epiphyseal areas of femus (distal end) and tibia (proximal end) showed no gross abnormalities.

Histological Examination

Liver: The normal architecture was completely lost due to a marked fragmentation of the normal liver cell cords. The liver cells were widely separated from each other, irregular in size and shape and deeply bile stained. In the intervening spaces there were congested blood sinuosoids, active foci of haemopoiesis and heavy foci of round cell infiltration with a loose fibrillary connective tissue. There was no true fibrosis. The appearances were typical of a congenital luetic hepatitis. Blocks specially stained by Levaditi's method did not reveal any spirochaetes.

Kidney: There were large areas of perivascular haemorrhage with round cell infiltration scattered in both the cortex and medulla. The glomeruli and tubules were normal.

Pancreas: normal.

Spleen: No Malpigian bodies were present. The entire pulp was hyperplastic and congested. Many foci of erythropoiesis were scattered throughout.

Adrenals: only showed congestion.

Lungs: revealed a patchy atelectasis but no "pneumonic alba" was present and no spirochaetes were demonstrated by the Levaditi stain.

Tibia: sections through the epiphysis did not show any osteo-chondritis.

Comment

As before mentioned, this case was an interesting diagnostic problem. The diagnostic possibilities were multiple and included: icterus gravis neonatorum (erythroblastosis foetalis), acute haemolytic anemia of Lederer, subacute and chronic haemolytic anemia of von Jaksch, acholuric familial jaundice, severe infective hepatitis, congenital obliteration of the bile ducts, congenital syphilis, haemorrhagic disease of the newborn, purpura, hemophilia, leukaemia and simple physiological jaundice.

The age of the infant tended to exclude several of these possibilities. The acute haemolytic anaemia of Lederer and the subacute and chronic haemolytic anaemia of von Jaksch are diseases seen in the age group of six months to three years. Simple physiological jaundice is maximum on, and is usually not noticed until, the third day. Severe infective hepatitis usually manifests itself about the fifth day. Acholuric jaundice, though usually revealing itself during the first decade of life and even in early infancy, has not been reported as occurring before the second to third week. Purpura, hemophilia and leukaemia are also very rare in the newborn. In congenital obliteration of the bile duct, the infant usually seems healthy at birth and jaundice seldom appears before the second week.

The presence of splenomegaly also limited the possibilities, ruling out physiological jaundice, haemorrhagic disease of the newborn and the acute haemolytic anemia of Lederer.

Consequently, from the beginning the most probable diagnosis was either icterus gravis neonatorum or congenital syphilis. As this was the mother's first pregnancy and, as the history was negative regarding blood transfusions, it seemed improbable that, even should her Rhesus (Rh) factor prove to be negative, there had been any possibility of her blood becoming sensitized and therefore containing anti-Rh agglutinins. However, as rare cases of erythro blastosis foetalis in the first-born have been reported and as approximately 10% of cases of this disease are not due to the Rh factor but to other immune agglutinins such as the A or B factor, the possibility of this diagnosis still had to be entertained.

The laboratory investigation reported above soon clarified the picture and labelled the case as one of congenital syphilis and this was definitely confirmed by the post-mortem findings. The failure to demonstrate spirochoetes by the Levaditi—method in the liver and lungs is unusual as they are reported as being demonstrable in eighty to ninety per cent of cases not having received anti-leutic treatment. The explanation in this case is not apparent but, although the staining technique may have been at fault, the one anti-leutic treatment received by the mother cannot be disregarded as a factor. In any case, their demonstration was not essential as the histopathology of the liver was diagnostic.

Personal Interest Notes

THE BULLETIN extends congratulations to Dr. and Mrs. W. E. Boothroyd on the birth of a daughter, Marcia Anne, in Toronto; to Dr. and Mrs. Philip MacDonald of Sydney on the birth of a daughter, Catherine Frances, on January 16; and to Dr. and Mrs. Gordon W. Bethune on the birth of a son on January 23 in Halifax.

The BULLETIN extends sympathy to Dr. Roy A. Moreash of Berwick on the death of his mother, Mrs. Jessie Ann Moreash, who died on January 2 at the age of 70; and to Dr. and Mrs. C. H. Reardon of Halifax on the death of their 10-months old son on January 27.

"Courage and Devotion Beyond the Call of Duty"

Through the cooperation of Mead Johnson & Company $34,000 in War Bonds are being offered to physician-artists (both in civilian and in military service) for art works best illustrating the above title, as applied to physicians in war and in peace.

This contest is open to members of the American Physicians' Art Association and will be judged June 9-13, 1947, at the Atlantic City Session of the American Medical Association. For full details, write Dr. F. H. Redewill, Secretary, Flood Building, San Francisco, Cal., or Mead Johnson & Co., Evansville 21, Indiana.

DOCTOR WANTED

The community of Bass River is without a physician. Any doctor interested in settling there should kindly communicate with Mr. Jas. S. Creelman, General Manager of the Dominion Chair Company, Limited, at Bass River.

OFFICE SPACE FOR RENT

A suite of three offices with modern conveniences, suitable for a doctor, is available. Apply to McFatridge Drugs Limited, Gottingen Street, Halifax.

Obituary

DR. FRANK V. WOODBURY, for many years Medical Examiner for the City of Halifax, died suddenly at his home on February 5th.

He was born at Babylon, Long Island, New York, on September 22, 1881, the son of Doctor Frank and Jessie B. (Troop) Woodbury, both of Halifax. He attended the Halifax County Academy, and later the Halifax Medical College, from which he graduated in 1903. After two years of practice in Newfoundland he pursued postgraduate studies in Edinburgh. Returning to Halifax, he continued in practice until 1915, when he joined the Dalhousie Medical Unit and went overseas. Leaving that Unit for a Field Ambulance, he was eventually promoted to the rank of Lieut. Colonel, and served as Assistant Director of Medical Services, Canadian Army Overseas until the close of War 1.

Soon after his return to Canada in 1919, Doctor Woodbury became Medical Examiner for the City of Halifax and the Town of Dartmouth. He continued the general practice of medicine, however, with some emphasis on anaesthesia, and also served as United States Immigration Medical Examiner.

As an outcome of his intense interest in Medical Jurisprudence, Doctor Woodbury lectured on that subject for many years at Dalhousie University. From its origin in 1908 he was active in teaching at the Dalhousie Dental School, and after 1912 was its Professor of Oral Surgery.

In the death of Doctor Woodbury, the students of Dalhousie have lost not only a valued instructor, but a lifetime friend. No matter how junior they might be, he treated them as confreres in the study of problems of pathology and jurisprudence. He loved to tell of the pathological laboratory at the Halifax Medical College, and how he used to act as a human incubator for bacterial culture tubes because there was no mechanical substitute available. Because he never forgot his student days he never lost touch with the problems and difficulties of the students, and in return won their affection, and high regard.

There is a feeling of intense loss in the hearts of his fellow physicians. He was their team mate or opponent on yacht or tennis court. In any sport he played a good game and he played it with keen fairness. In the same way he played the game of life, up to its last moment. They will miss his helpful skill as a physician, and as a sincere and forthright friend.

To all the members of his family the BULLETIN extends its sincerest sympathy.

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