A Lecture

ON

ACUTE CEREBRAL LESIONS AT
DIFFERENT AGES.DELIVERED BEFORE THE WEST SUFFOLK DIVISION OF THE BRITISH
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ACUTE cerebral lesions may be reckoned among what my old teacher, the late Sir William Gowers, once called "the uncommonplaces of medical practice." Not occurring often enough to become familiar, if not banal, yet not so rare as to render ignorance of their nature and treatment pardonable, they are seen by the practitioner more frequently than by the consultant, and in nearly every instance necessitate his coming to quick decisions and adopting prompt therapeutic measures. Obviously he must have a certain confidence in his own diagnosis when such circumstances prevail, and must have at ready disposal some formulation of diagnostic elements on which to rely during the first hours after the lesion reveals itself. A waiting policy does not always serve at these times.

In this, as in many other instances, the problem is to know how to approach the problem. Working rules are necessary if he is to grasp the essential features of the situation and avoid being misled by non-essentials. Text-book descriptions doubtless cover all eventualities, but their application to the practice of medicine leaves him on occasion in perplexity. I do not pretend that the problem can be simplified by any rule of thumb devoid of exceptions and infallible in its working; all of us commit errors of diagnosis and of judgement, and sometimes hesitate not so much from ignorance as from fullness of knowledge. When this situation develops, another dictum of the same great clinical teacher deserves to be recalled: "Be decisive in your indecision"—in other words, be sure of your reasons for not being sure, and adopt measures calculated at least to do no harm.

One of the most convenient and practical ways of dealing with the problem of an acute cerebral lesion is by attending to the age of the patient. At any given age period, what are the probabilities? The generalizations sketched below have their value, relative though not absolute, and are worth bearing in mind.

I. IN CHILDREN AND YOUNG PEOPLE.

Numerically and proportionately less common in childhood and early adolescence, abrupt cerebral affections constitute no actual rarity and are often not a little disconcerting. Since the observer is able to dismiss all thought of degenerative lesions from his mind in such cases, the pathological possibilities are more or less restricted to toxic and toxi-infective processes, pressure, trauma, and blood-vascular states. Of these, the first-named form the majority.

1. Much the most frequent is one or other variety of *encephalitis*, by which pathological term is to be understood a brain infection, whether specifically known or not. A sporadic encephalitis (not, in all probability, identical with what is now called epidemic encephalitis)—sometimes described as acute non-suppurative encephalitis—is a fairly common affection of childhood, producing usually a hemiplegia and less often a diplegia. When a preliminary stage of malaise, fever, and vomiting, with headache, possibly with delirium and stupor, is succeeded by generalized convulsive attacks with unconsciousness, rigidity, cyanosis, and so forth, and followed by paralysis on one side of the body, the syndrome is not so sudden as to preclude a very good idea of its nature being formed. But in a minority of cases the paralysis supervenes abruptly on a convulsion, or even without the latter, just as it appears unheralded in a small proportion of cases of acute poliomyelitis. Diagnosis then is perhaps less definite, but the

probability is not; lumbar puncture is likely to reveal a finding comparable (in greater or less degree) with that of acute poliomyelitis should the case be one of acute poliomyelitis—a poliomyelitis variant; if the fluid is negative, however, the case may none the less be one of encephalitis, though not occasioned by the virus of infantile paralysis.

In a recent instance a schoolboy was sent to bed with an ill-defined ailment—slight rise of temperature, headache, pains in the neck; on the third day he developed diplopia, followed by complete aphagia and a remarkable dysphonia, a high-pitched, "reedy," "piping" intonation which he was incapable of modulating. Lumbar puncture showed a fluid differing immaterially from the normal, and the case was fairly, as I think, classified in the group of acute bulbar encephalitis.

I have often thought that some of the obscure cases of quick death in seemingly healthy children, the sole symptoms being clouding of consciousness with respiratory or cardiac embarrassment and failure, are attributable to poliomyelitis of the medulla, of the class already mentioned—namely, that without recognizable prodromata. Here, indeed, lies one of the serious difficulties connected with poliomyelitis; no definite relation has been made out between the mildness or otherwise of the preliminary syndrome and of the paralytic consequences. The former may actually escape notice, or be ignored, yet palsy may set in after a truly fulminant fashion.

Of less diagnostic difficulty are the varieties of cerebral disorder arising abruptly in the course of one or other of the *specific fevers and exanthems*, and set down (in some instances at least) to encephalitis. These juvenile encephalopathies are well recognized, however rare their incidence and variable their actual pathology. They are known as an occasional development in scarlatina, diphtheria, measles, mumps, pertussis, typhoid, small-pox, etc., as well as in influenza, pneumonia, and other infections. The underlying pathological condition is more frequently a matter of surmise than of proof; sometimes a haemorrhagic encephalitis, in other instances it is a vascular state of haemorrhage, embolism, or thrombosis, or a cerebral oedema associated with nephritis, or a serous meningitis. In a valuable series of papers J. D. Rolleston¹ has collected information bearing on these rather out-of-the-way cases, as has recently also my former house-physician and present colleague W. P. H. Sheldon.²

Attention may next be directed to the occurrence of *encephalitis after vaccination* (ordinary cow-pox), a sequel that has aroused some concern in the course of the last three years, over one hundred cases having been recorded in that time (Luckesch,³ Bastiaanse,⁴ Turnbull and McIntosh,⁵ Wiersma,⁶ Perdrau,⁷ Bouman,⁸ and others). There can be little doubt that such cases were formerly extremely infrequent, and the conclusion seems justifiable that they are somehow connected with the times through which we are passing; at any rate, one theory at present offered is to the effect that encephalitis after vaccination is due to activation by the vaccine of another "invisible virus," conceivably that of epidemic encephalitis or a herpetic one. Valuable experimental evidence in favour of the view that the encephalitis is not due to the vaccine itself has been furnished by Pette.⁹ Clinically, convulsions and hemiplegia seem to be rather common in vaccination encephalitis; an initial stage of general cerebral disturbance is usually definite and of some little duration, although localizing symptoms develop with rapidity. The pathological lesions resemble those of acute disseminated encephalomyelitis of the adult.

2. Of interest and importance is the group characterized by symptoms suggestive of *meningitis* or pointing to the existence of *meningeal reactions* of toxic or toxi-infective origin. Classified as meningismus or as circumscribed serous meningitis (both terms being as a fact rather indefinite from a pathological viewpoint), the type of case is that in which intracranial symptoms (cerebral, cerebellar, pontobulbar) develop unexpectedly and alarmingly early or late in the course of an indefinite illness, with mild meningeal indications, for which no source of infection is discoverable. Serous meningitis is the least sinister of all meningeal states; it consists chiefly in an increase of cerebro-spinal fluid in the appropriate cerebral and meningeal spaces, producing an internal and/or

external acute hydrocephalus, apparently always of a non-microbic character—in some instances (and these are the simplest diagnostically) a reaction to sepsis at some near but extra-meningeal spot (ear, nasal passages, tonsils). To this variety the term "meningitis serosa comitans" is sometimes applied. In numerous other examples of the affection, however, the meningeal reactions are localized (meningitis serosa circumscripta), producing focal symptoms and signs, although the underlying source seems to be a diffuse toxicosis of diverse kinds. Lumbar puncture yields an essentially negative fluid, sterile, with cytology little altered, but with perhaps a protein increase, and usually under considerable pressure. The remarkable feature of most cases is also a fortunate one; after withdrawal of fluid the symptoms are mitigated, and in not a few cases soon entirely disappear. I have been misled by cases of this kind on more than one occasion.

A little boy of 8 developed in the course of a few hours a complete right hemiplegia and aphasia, signs of general disturbance being minimal. No source of infection could be found. I diagnosed the case as one of encephalitis and gave a guarded prognosis. Lumbar puncture yielded a normal fluid under pressure, and within twenty-four hours thereafter the symptoms had completely vanished.

Cases of this kind are highly deceptive, but other examples of meningitis serosa simulate cerebral tumour (so-called pseudotumor cerebri) and are less calculated to give rise to acute cerebral symptoms.

3. We do well to bear in mind the possibility of insignificant *traumata* being succeeded after a variable interval by sudden cerebral disturbance. Trifling knocks raise haematomata on youthful heads with surprising ease. The serous meningeal reactions already alluded to may follow tearing of arachnoid tissues by trauma; in other cases subdural or subarachnoid haemorrhage results. I have seen more than one example of trivial injury turning out to be serious, but none so dramatic as that recorded long ago by Erichsen.

"A little girl was going downstairs with her mother to dinner. She said, 'I will go first, mamma,' and started to run downstairs, but she missed her footing and fell forwards, striking her head slightly against the wall. She felt a little dazed at the time, but went to her dinner, ate, and afterwards felt slightly sick. She was sent to bed, slept soundly, and was dead next morning. There was a clot found between the dura mater and the skull on the side of the head that had been struck, but no fracture."

The fatal injury may be so slight, indeed, that no concussion symptoms are produced, a point to which particular attention is directed. When we remember that in the growing child both periosteum and meninges are highly vascularized for purposes of nutrition, it is likely enough that corresponding to the visible "bump" where his head has been knocked is a haematoma greater or less in degree of the intracranial membranes. This state of affairs, in point of fact, has been found by Wood Jones,¹⁰ who adduces evidence strongly suggesting a venous and not an arterial origin for the haemorrhage. Sheldon's² series embraces several cases belonging to this group; in one instance a child hit his head against a table; in another, a boy fell off a kitchen chair.

Consideration of this part of our subject naturally leads to the problem of what has been called *traumatic delayed apoplexy* ("traumatische Spätapoplexie"), a matter of peculiar medical and sometimes medico-legal importance. The condition is not confined to youthful cases, but may be conveniently referred to in the present section. A latent period of days or weeks may elapse between the date of the injury and the onset of ominous cerebral symptoms. Numerous instances of delayed apoplexy are now on record (Bollinger,¹¹ Marie and Crouzon,¹² Lambert,¹³ Buzzard and Cuning,¹⁴ Warrington,¹⁵ Allen,¹⁶ and others). For admission in the class the appended criteria require attention: (1) cases in which apoplexy might develop spontaneously must be rigorously excluded (syphilis, nephritis, alcohol, heart disease, arterio-sclerosis, etc.); (2) the injury must have been definite though not of necessity severe, or producing unconsciousness; (3) the symptoms of the lesion must have appeared within a short space of time and under observation. During the interval between injury and apoplexy cerebral symptoms of a general kind and slight in degree may be noted; some-

times they appear to be non-existent. In one of Sheldon's cases a child fell against a cupboard, and "several weeks" of seeming health elapsed ere vomiting and loss of consciousness set in, these symptoms preceding death by only a few hours. But in the Buzzard-Cunning case, after a trivial head injury through a fall, seventeen days of headache were succeeded by drowsiness and other symptoms of pressure, and a craniectomy revealed bleeding from the superior longitudinal sinus.

It is impracticable to lay down any exact time limits for cases coming within this category; only those can be admitted in which causal connexion between trauma and symptoms is reasonably definite and in which from the date of infliction of the former a reasonably brief period elapses ere the latter make their appearance. I have had to adjudicate in a case of right hemiplegia and aphasia developing suddenly four weeks after a trifling knock on the skull through the patient (a man of 46) striking an overhead rafter, and I decided against the diagnosis of delayed apoplexy.

4. Where some more or less obvious *blood state* is in existence the explanation of acute cerebral lesions in youthful subjects is not obscure. I need only allude in passing to the occurrence of haemorrhage, thrombosis, or embolism, or combinations of these, in heart disease; in marantic cases, in leukaemia, splenic anaemia of infancy, purpura haemorrhagica; in any severe anaemia, in scurvy, haemophilia, and so forth.

5. There remain for discussion the always difficult and disconcerting cases of sudden death from *haemorrhage* in children and adolescents, of which numerous examples have been published (cf. a useful paper by Hawthorne¹⁷). Unless an autopsy is obtained cases of this kind are shrouded in obscurity, and their pathology is a pure speculation; if the patient recovers it is equally so. Here we touch what Sir William Gowers once called "the old and inevitable antagonism between pathology and therapeutics." Each flourishes at the expense of the other, and the ends for which we gain our knowledge can be the chief obstacle to its acquisition.

Briefly, in my experience the pathological possibilities are: (a) bleeding from an unsuspected vascular intracranial tumour; (b) bleeding from an otherwise undiscoverable miliary aneurysm ("congenital aneurysm"); (c) bleeding from a structurally defective blood vessel without aneurysmal formation. A fourth possibility has been suggested by Goldflam¹⁸—namely, (d) bleeding of the nature of capillary oozing during hyperaemia following constriction—a disorder of vasomotor function. The term "spontaneous subarachnoid haemorrhage" is in vogue for cases of this kind, though it is somewhat misleading. Clearly, if evidence of aneurysmal formation or neoplastic haemorrhage is discovered at necropsy the case should in point of fact be described under the heading of cerebral aneurysm or tumour, as the case may be. The literature on these types is considerable, clinico-pathological instances and useful reviews having been published by Symonds,¹⁹ Beadles,²⁰ Fearnside,²¹ Turnbull,²² Weber and Bode,²³ Marburg,²⁴ and many others.

As is to be expected, the symptomatology is highly variable, depending on both the degree and the site of the haemorrhagic extravasation. Elsewhere²⁵ I have given a full account of "spontaneous" leakage from a frontal tumour in a youth of 19; the extravasation spread through the ventricular system and passed under the basal meninges, reaching the vertex in one direction and the spinal cord in the other. In this instance the clinical picture for some seventy-two hours was that of decerebrate rigidity. The usual syndromes of ventricular, cortical, pontine, basal haemorrhage may be encountered.

A recent example was the case of a healthy schoolgirl of 17, who, after two days of insignificant malaise diagnosed as nothing more than a cold, became unconscious at 3 o'clock in the morning and was dead at 9. Lumbar puncture yielded a fluid laden with fresh blood. No necropsy being granted the case remains as obscure as it is tragic.

I have also had occasion to observe more than one instance of cerebral haemorrhage in youthful subjects supervening apparently on excessive emotional disturbance. Such cases provide food for thought even if objective corroboration is wanting.

A married woman of 20 ascertained that her husband was conducting a liaison with another person. In a state of intense emotion she went to confront them with the righteous indignation of a young wife. After a painful scene she left the house and fell unconscious on the pavement outside. Two days elapsed ere consciousness returned, accompanied by right hemiplegia and complete motor aphasia. Now, when she is 45 years old, the hemiplegia remains, and a trace of the former speech defect.

Here, doubtless, we may imagine a miliary aneurysm or a weakened spot in the wall of the left Sylvian artery to have ruptured under the influence of the altered vascular balance attributable to excitement.

II. IN EARLY AND MIDDLE ADULT LIFE.

In the age periods comprised by the twenties, thirties, and forties most of the etiological factors already enumerated are still in the picture. Trauma, encephalitis, blood states, cardiac disease, and "spontaneous" vessel rupture must always be considered possibilities. To these we now add acute cerebral lesions associated with (1) pregnancy, parturition, and the puerperium; (2) neurosyphilis; (3) haemorrhagic pachymeningitis.

1. Much that is still enigmatic surrounds the occasional development of acute hemiplegia (or paraplegia) *before, during, or after confinement*. Half a century ago clinicians were attracted by the conception of "reflex paralysis"—sometimes also described as "urinary paralysis"; Sir Samuel Wilks²⁶ contended that a paraplegia might be induced by an external irritant, that "the blood vessels are thereby diminished in calibre, the blood supply to the spinal cord is diminished in amount and its power departs." He was concerned mainly with cases of temporary paralysis, and sought an explanation in irritation of vasomotor nerves innervating blood vessels. *Inter alia*, he made the theory of reflex paralysis apply to the case of paraplegia succeeding labour. About the same time Fussell²⁷ stated that cows not infrequently suffer from paraplegia after calving, that they lie down, unable to move their hind legs, and apparently have no feeling in them when they are touched. In about a fortnight recovery ensues. Similar phenomena are known to occur in pregnant mares.

Present-day conceptions have widened in range without gaining in precision. Puerperal hemiplegia (inclusive of pre-puerperium cases) includes a rather heterogeneous collection of morbid states. Apart from the ordinary causes (syphilis, cardiac disease, etc.), to which, of course, the pregnant woman as well as any other may be subject, certain processes are apt to develop in connexion with this physiological condition. A review by Duhot²⁸ informs us that of 46 cases 17 were occasioned by cerebral haemorrhage, youthful though the patient frequently was. Physiological hypertrophy of the left ventricle and increased richness of the blood are thought by themselves to be insufficient, and the additional factor of albuminuria is incriminated. Localized cerebral oedema, rather than haemorrhage, and the local effect of toxæmia ought not, however, to be overlooked. Another variety is meningeal haemorrhage, but its explanation is unfortunately not clear. It may develop in the absence of albuminuria or cardiac disease, or of any evident visceral or toxi-infective process. I may mention an ingenious theory to the effect that increased activity of the suprarenal glands in pregnancy, as disclosed by a condition of hypercholesterinaemia, leads to a rise of blood pressure from time to time, and so to meningeal haemorrhage.

But in other instances thrombosis instead of haemorrhage provides the causation, and its pathogenesis is equally indefinite, unless we fall back on the analogies alluded to above and assume not so much a "reflex" paralysis as one originating in stagnation of viscous blood owing to a weakly acting heart or a low blood pressure. The paraplegia of pregnancy (sometimes called Moxon's paraplegia) may result from a thrombosis of this kind in the long anterior spinal artery.

The physiological peculiarities of pregnancy and the puerperal state deserve more attention from the neurologist. Three cases of epilepsy occurring solely during pregnancy and in the absence of eclampsia have been reported by Cürschmann.²⁹ Amelioration of the characteristic symptoms of myasthenia gravis in the course of pregnancy has long been known but never explained. The

evolution of abnormal clinical conditions during a normal physiological process should surely check the misconceptions of those who persist in the habit of making "symptoms" constitute "diseases," and who with equal persistence ignore the physiological nature of the former.

2. Little need be said of *neurosyphilis* in these days, when any suspected case of acute cerebral disorder can be made to run the gauntlet of serological tests and a verdict obtained within a few hours. Clinically, neurosyphilis turns up in unexpected quarters and in unwonted guise. A sudden loss of consciousness with epileptic fits and consequent paresis may be the first indication of incipient general paralysis; an acute hemiplegia or monoplegia may betray an endarteritis syphilitica, or a little-suspected gummatous meningitis; syphilitic cerebral haemorrhage, less well recognized and less often envisaged, should not be overlooked.

Only a week or two ago sudden headache, sickness, malaise, and rise of temperature, followed by right facial weakness and considerable aphasia, led me to diagnose encephalitis in the case of a man of 35; but routine insistence on the tests of the clinical laboratory enabled me next day to correct it to neurosyphilis, with corresponding improvement in the prospects of recovery.

3. A rarer affection is that known as *pachymeningitis haemorrhagica interna*, the pathology of which is explanatory of its somewhat unusual symptoms. Occurring more frequently among those addicted to alcohol than others, it is signalized by intense headache, vomiting, sometimes convulsions, and abrupt loss of consciousness. There is not uncommonly a rise of temperature accompanied by signs of meningeal irritation; a bloody spinal fluid is obtained by puncture, with marked increase of albumin and globulin; smears are always negative and cultures sterile. Recovery ensues, hastened by withdrawal of spinal fluid, but recurrence of the attacks constitutes the danger. In the cases I have observed myself neurosyphilis was excluded, yet the basis of some recorded instances appears to have been of this nature (Hassin³⁰).

Thus on his return home from a city dinner a middle-aged man suddenly fell unconscious on his bedroom carpet, with generalized convulsions and immediate rise of temperature. Meningeal symptoms appeared and a clinical diagnosis of some form of meningitis was made. Only on the fifth day was lumbar puncture resorted to, when a clear fluid of an extraordinary pink-orange-yellow colour was withdrawn. Spectroscopy showed it to be full of haemoglobin derivatives. In view of the history and of the habits of the patient a diagnosis of pachymeningitis haemorrhagica interna was made, and confirmed by the subsequent course of the case.

This example of the condition is almost identical with one reported by Fried.³¹ Another instance, however, occurred in a younger man, in whom three separate attacks similar to the above developed at approximate intervals of six months; recovery was much slower after the second, and a fourth was fatal. At necropsy the peculiar stratiform alternation of membranous and haemorrhagic layers over the postero-superior and postero-lateral aspects of the encephalon was at once recognized.

III. IN LATER LIFE.

If we assume with some arbitrariness that the fiftieth year of life ushers in a degenerative period, we may expect thereafter acute cerebral affections, of which the commonest symptoms are hemiplegia, aphasia, and hemianopia, and the commonest lesions vascular. Naturally the clinician must not forget the exceptions to the general rule; Bright's disease may bring cerebral disaster in its train long before 50, and families are known that are unhappily distinguished by hereditary apoplexy.

Vascular lesions, then, haemorrhagic or thrombotic, constitute by far the major portion of the cerebral attacks of later years; these should always be regarded as cerebral *traumata*, and treated by rules applicable to lesions of the vascular system. Differential criteria between haemorrhage and thrombosis often appear more impressive on paper than during anxious moments at a bedside; diagnosis is reached in numerous instances fully as much by scrutiny of the exact order of events as by consideration of the objective signs. In the subjoined paragraphs an outline is given of the points to weigh before committing oneself to a precise opinion. The condition of the vascular system is at least as significant in this connexion as the nature of the cerebral syndrome.

1. Inquiries directed towards the question of previous attacks may throw a sidelight on the present one. If slight seizures have occurred previously thrombosis is suggested; if a single severe previous attack, either. There may have been thrombosis originally, and haemorrhage now, or vice versa.

2. Asking next for the details of the present illness, the clinician notes that a more or less definite aura and a certain measurable duration for the unfolding of the attack characterize thrombosis, whereas there is little or none of this in the other process. Physical or mental exhaustion precedes thrombosis, it may be, but has little if any bearing on the occurrence of haemorrhage, as has, for that matter, the question of previous excitement. Ernest Jones³² has shown that quite a proportion of haemorrhage cases (subsequently confirmed) have their onset during rest or sleep. These are points of some moment, for if the ictus is attributable to a combination of special circumstances rather than to severity in degree of vascular change the prognosis as regards recurrence is proportionately better.

3. At the bedside, commencing with the state of the pulse and heart, the examiner concludes that a feeble pulse and weakly acting heart suggest thrombosis; a full and perhaps bounding pulse, with cardiac hypertrophy and a blood pressure above normal for the patient's years, is in favour of haemorrhage. Thickened or tortuous peripheral vessels indicate either, as does the presence of albuminuria demonstrable in catheter specimen or otherwise. Similarly, retinal haemorrhages noted with the ophthalmoscope do not favour one diagnosis rather than the other unless they happen to be severe and massed, in which case they point to cerebral bleeding.

4. Attention is also given to the loss of consciousness. Only if a large vascular area is out of action is unconsciousness likely to accompany thrombosis, whereas the laceration caused by extravasated blood inhibits the cortex, according to some, by irritation of corticopetal systems. Enduring unconsciousness strongly favours haemorrhage; a short initial loss, thrombosis. In basal, pontine, and ventricular cases the mechanism of production of the symptom consists in general (cerebral) vaso-constriction by excitation of a vasomotor control centre in the medulla. Should the obscuration of consciousness, trifling at first, steadily deepen as the hours pass, an intractable extravasation is indicated; yet in progressive thrombosis a not dissimilar phenomenon is occasionally remarked. The duration of these is never so prolonged, nor is there the same dubiousness of continuity, as may sometimes be observed in the cases of delayed traumatic apoplexy alluded to above.

5. As for concomitant epileptic seizures, if Jacksonian their repetition manifests a thrombotic process; if generalized, either that or haemorrhage. Since cortical vessels are more prone to the former than the latter the differential value of the symptom is explained.

6. Rise of temperature does not of itself furnish a means of distinguishing. It points to implication of the upper pons and is seen typically in pontine, basilar, and ventricular cases. Its steady and fairly rapid heightening is suggestive of early death.

7. If now the observer turns to other possible localizing indications apart from Jacksonian attacks, during the period of unconsciousness he may readily enough be able to diagnose capsular, pontine, unilateral, or bilateral sites of invasion, but of themselves these do not serve in differentiating between the two chief processes under discussion. The case is rather altered when consciousness returns. He may then be in a position to note that an enduring aphasia favours occlusion, since, as already mentioned, cortical thrombosis is as a fact more frequent than cortical extravasation. Again, hemianopia suggests a similar vascular origin, haemorrhage in the distribution of the posterior cerebral being distinctly rare.

With various interesting phenomena that can be demonstrated during the period of physiological decortication or decerebration (for example, the reflexes of Magnus and de Kleijn) I am not here concerned, as they do not shed light on the nature of the vascular process underlying the neural semeiology.

Since embolism may occur at any age, roughly speaking, I have left its consideration to the end. It does not present

those difficulties which seem to be inseparable from the other two, except perhaps when the circumstances are unusual. Thus if the practitioner finds a patient with mitral stenosis and a weakly acting heart at the degenerative period, is his acute cerebral lesion embolic or thrombotic? Again, war experiences have shown how traumatic thrombosis of the internal carotid may cause a sudden cerebral attack by detachment therefrom of an embolus (Colledge and Dunn³³).

When examination reveals a lesion of the vascular system in the emboligenic circulatory area, as it may be called, diagnosis of an acute cerebral ictus without premonitory symptoms can rarely if ever be erroneous. Infective endocarditis can scarcely be overlooked, although a presystolic murmur may be missed and milder endocarditic manifestations disappear.

Concluding reference to the controversial question of temporary or transient hemiplegias and other analogous cerebral disorders must of necessity be brief. The reality of these clinical disturbances is, of course, undeniable, but their pathogenesis is in the nature of things likely in most instances to be matter of speculation rather than of proof. Too facilely has acceptance been voted to the theory assigning them to an angiospasm—a transient constriction of a cerebral vessel thought to be itself in a degenerating condition; several weighty considerations militate against this view. (1) Leonard Hill³⁴ has shown experimentally that the cerebral circulation passively follows changes in general arterial and venous pressure, and is controlled by bulbar centres acting on the general circulatory system. If cerebral arteries are constricted corresponding capillaries become congested with venous blood—not emptied, with resultant local anaemia. (2) The pial network of vessels is so closely interwoven that symptoms from strong and persistent localized cramp in some of its twigs seem improbable, as collateral supply would supplement the arrest (Allbutt). (3) All arteries under the control of vasomotor nerves react to adrenaline by contraction, but the cerebral vessels do not (Florey³⁵). (4) The tacit assumption is made that arterio-sclerotic arteries are specially prone to spasm, yet in that state the muscular elements have lost their contractility by undergoing fibrous degeneration. (5) Advocates of the theory of angiospasm do not appear to have faced the difficulty that the local cerebral anaemia of the motor zone which they postulate is unaccompanied by Jacksonian or general epilepsy; yet evidence has collected to demonstrate the association of hyperexcitability with cerebral vascular constriction, even if temporary.

The truth is insufficient attention has been given to the occurrence of microscopic organic lesions as a foundation for the symptom-complex, transient and recurrent, set down to angiospasm (Achard, Foix, and Leblanc,³⁶ Evans,³⁷ and others). Again, if a vessel is degenerated transient reduction of general blood pressure may result in a failure to maintain the column of blood against peripheral resistance, or to overcome partial obstruction; hence the development of transient paresis or paralysis from relative ischaemia. I do not doubt, further, that this is the correct explanation of those cases in which transient amaurosis is stated to coincide with observed spasm of retinal vessels (Lundie,³⁸ Bruner³⁹); because of passing hypotension the artery may seem to diminish in calibre and perhaps actually do so, yet of arteriospasm in the strict sense there need be no trace. In fine, other possibilities than angiospasm may legitimately be utilized in the elucidation of transient cerebral attacks; and since this is so the physician need not seek a pathogenesis capable of being applied universally, and should realize the intrinsic variations among them on the clinical side as well.

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THE TREATMENT OF COLLES'S FRACTURE.

BY

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THE admirable short paper by E. D. Grasby and S.-R. Trick in the *British Medical Journal* of March 2nd (p. 391), following upon that of Edwards and Clayton in the *Journal* of January 12th, prompts me to offer a few remarks on the treatment of Colles's fracture.

Colles's fracture is at once the commonest (sharing this distinction with fracture of the clavicle), one of the easiest to treat, and generally the worst treated, of all fractures of the human body.

The first-mentioned paper is "an endeavour to ascertain the causes for the imperfect results that obtain from the usual methods of treatment at present adopted." The principal conclusions arrived at are:

"1. Colles's fractures without displacement yield uniformly good results, as observed two years after the injury.

"2. Where displacement was a marked feature of the fracture the results two years after the injury are invariably unsatisfactory.

"3. The disability was in each case due to osteo-arthritis in the inferior radio-ulnar joint, resulting from incomplete reduction."

From this we must infer that at a great London teaching hospital a Colles's fracture is never properly reduced. For, apparently, when the displacement is not considerable, reduction is not considered necessary, and, when displacement is considerable, the result is invariably unsatisfactory owing to incomplete reduction. We need not, however, infer that the results so candidly reported from this clinic are in any way exceptional. They are indeed the usual results of the methods of treatment at present adopted. I personally never, or hardly ever, see a case of Colles's fracture which has been properly reduced, although many of the cases that I see have had some sort of manipulation previously. The reason for these uniformly bad results is that the method of reduction almost universally employed—namely, that advocated by Sir Robert Jones—is entirely inadequate in the hands of the majority of those who have to treat these cases.

In the classical Colles's fracture—and most cases are of this type—there are three characteristic displacements of the lower fragment of the radius in relation to the upper—namely, (1) displacement upwards and backwards; (2) abduction to the radial side; and (3) rotation about a transverse axis—that is, backward tilting of the lower articular surface, the importance of which is rightly emphasized by Edwards and Clayton. Each of these displacements must be deliberately corrected if a good result is to be obtained.

Colles's fracture is produced by great force—that is, the whole weight of the falling body upon the fixed wrist—and it requires great force to reduce it. The reduction of a

Colles's fracture is a matter of sheer force; there is no trickery about it whatever.

General surgeons as a rule have no conception of the amount of force that orthopaedic surgeons can, and often do, employ in quite ordinary manipulations, and I think that some orthopaedic surgeons hardly realize it themselves. It may be that the powerful hands of Sir Robert Jones (and some others) can reduce a Colles's fracture in any manner that he chooses; but I am convinced that the method which he advocates is quite inadequate in the hands of the great majority of those who have to deal with Colles's fractures.

For general use there is no method to compare with that of *reduction over a wedge*. By this method the upper fragment is fixed, and the lower fragment is compelled by sheer force to go in whatever directions the operator wishes. Briefly the actual procedure is as follows:

The patient must be fully anaesthetized. Some expert anaesthetists can give gas or gas and oxygen for long periods, and can even obtain muscular relaxation (with luck), but this is quite exceptional. As ordinarily given, "a whiff of gas" is entirely inadequate for the proper reduction of a Colles's fracture. In these circumstances the reduction is always hurried, the patient is usually stiff or moving, and, above all, there is no time for a critical review of the reduction before he comes round, nor for a repetition of the manipulation if it should be found to be incomplete. The reduction should be done deliberately with the patient fully anaesthetized. I think that regional (brachial plexus) anaesthesia might be employed here much more often than it is.

For the manipulation the fracture is laid flexor surface downwards across the wedge,* so that the lower end of the upper fragment rests directly on the wedge. For the right side it is convenient to have the wedge lying on the table by the patient's side; for the left side (unless the surgeon is left-handed) it is more convenient to have it on an outlying small table. The upper fragment is firmly held by the surgeon's left hand on the wedge. The lower fragment is gripped by the surgeon's right hand with the thumb on top and the ball of the thumb directly over the prominence which the displaced lower fragment makes on the dorsum. The lower fragment is now forced downwards and forwards and towards the ulnar side. The amount of force required varies in different cases, but in all cases it is considerable, and I have many times put the whole weight of my body on to this fracture in order to reduce it properly. At the end of this manoeuvre the backward tilt of the lower fragment is corrected by a sharp forward flexion movement.

Many surgeons make a point of first disimpacting the fracture by dorsiflexing the wrist and temporarily increasing the deformity. This is harmless, but unnecessary. The only objection to it is that it is apt to make a surgeon think that he can reduce the fracture by some clever manoeuvre instead of by force. In reducing over a wedge disimpaction occurs just as readily as soon as the lower fragment is made to move, and then all that is necessary is to force the fragment into its proper position.

After the reduction the fracture should be critically examined. The positions of the styloid processes should be noted and compared with the normal. The prominence on the dorsum should be felt for, to make sure that it has quite disappeared, and it should be seen that the forward concavity of the lower end of the radius is restored. Lastly, the position of the hand as it hangs freely in relation to the lower end of the radius should be noted. If any part of the deformity remains uncorrected, the manipulation should be repeated.

There is very little tendency for the deformity to recur after a Colles's fracture has been properly reduced, unless the wrist is dorsiflexed, or the splint which is applied presses the lower end of the radius backwards.

The popular Carr's splint, as originally designed and as supplied to general practitioners and commonly used by them, is a bad splint, because it presents a flat surface to the normal forward concavity of the lower end of the radius and so causes the lower fragment to be tilted backwards. As padded up and applied by experts it is, no doubt, quite efficient, but then it is no longer a "Carr's splint."

* The so-called orthopaedic wedge is merely a short round horizontal bar covered with rubber, standing about four inches high on a firm base. For those who want one occasionally and have not got one, I may add that an ordinary flat iron, borrowed from the kitchen, with its round horizontal handle covered with a folded towel or other soft material, makes a perfectly good "orthopaedic wedge."