M.D. Thesis

"Observations on General Paralyses."

Harvey Baird

P.T.O.
In the following pages I shall endeavour to give an account of my observations on general paralysis of the insane, proceeding systematically by describing its pathology, symptomatology, etiology, etc. One cannot attempt in a thesis of this description to give a detailed account of the complex anomaly, but I shall record what I consider to be the principal facts. The observations made are based upon a three years experience of clinical and pathological asylum work, principally in the West Riding Asylum, Wakefield.

Pathology. Undue adhesion of the scalp is frequently met with. The skull-cap is usually thickened and sclerosed, and the weight increased. These changes in the coverings of the head of a chronic proliferative nature analogous to the meningeal and vascular changes in its interior. Haematoma auris is especially frequent.
It is not with general paralysis relatively more frequently than in any other mental disorder, in my opinion. This condition appears to be dependent on degenerative change in the cartilage cells. Groups of these degenerated cells become converted into small cysts. The capsules in the walls of these cysts, themselves degenerated, are specially prone to rupture. Here again there is some analogy with intracranial change.
The dura mater shows well-marked alteration in most cases. Undue adhesion and thickening are common. Sometimes to the naked eye little change is visible. Probably in all cases there is microscopic evidence of chronic proliferative and degenerative change. Proliferation of the surface endothelium and of that lining the perivascular canals occurs. Throughout the substance is an increase of fibrous tissue and arteriosclerosis is common. Subdural false membrane is very frequent.
frequent. In 131 consecutive post-mortem I found it in 43, or 33%. Most observers have given less. There is little doubt that many small delicate membranes can be easily overlooked. I agree with Ford Robertson's (1) explanation of the condition as being dependent on chronic proliferative changes in the endothelium and vessels of the dura. A sort of granulation tissue is formed with capillaries specially prone to rupture. Large effusions are, as he says, usually recently ante-mortem. Pannett (2) has experimentally failed to find any evidence of organisms or their products being in the false membrane. To my mind, this change, like haemato ma awui, thickening of dura and calvarium, etc., is due to the same profound tissue alterations of a proliferative and degenerative nature seen always in general paralyses. This morbid condition of the tissue cells may be due to an inherent
specific loss of vital energy, poverty congenital, more probably induced by syphilis, alcohol, etc., or to alteration in the blood or lymph, which supplies them, by toxins.

The pre-arenchoid is opaque and thickened. Attention to the cortex with erosion on stripping is considered by many as almost pathognomonic. I believe it occurs less frequently than many suppose. Of the above series, 73, or 55.7%, showed this. The majority of cortex were stripped in their entirety, but in some only the left frontal and parietal regions; therefore, allowing for a few that may have been overlooked, one may say that 40% of cases exhibit no erosion on stripping the pre-arenchoid. The cause of the erosion on stripping must be either unusual toughness of the connections between pia and cortex, or unusual softening of the cortex. I believe the latter to be the
(1) Pathology of Relation to Mental Diseases: p. 103

(2) Brain, 1903 — Summer.
the main cause, because erosion is mainly seen on the summits of the gyri, and because with a very thick pia, there may be no erosion on stripping. Though not pathognomonic, adhesions are still strongly suggestive of general paralysis. Their localisation is of interest, as in some cases it has been noted that the areas affected corresponded to those involved in convulsions during life. This I have been markedly exemplified. The amount of fluid contained in the meshes of the pre-arthroid is almost always largely increased. This is compensatory to brain atrophy. Böttcher has found choline in excess in the cerebrospinal fluid, also in the blood. Duflos has found lymphocytes in the cerebrospinal fluid in cases of senile and alcoholic dementia. General paralytic and senile decay brains show this excess of fluid more markedly than any other disease. The ventricles usually also are dilated, often markedly so.
The brain itself is usually soft and oedematous. In the great majority of cases, great wasting of the convolution is evident, and is mainly in the frontal and parietal regions. The brain usually weighs about 70 grammes less than the normal. The grey matter is diminished in depth, usually rather dark in colour, and the striation is indistinct.

Granularity of the ventricular ependyma is present in the great majority of cases. It occurred in 115, or 87.8%, of the 131 cases. It is much more frequent in general paralysis than in any other disease. This is probably due to proliferation of the neuroglia in the tissue immediately subjacent to the epithelium. It is fairly common in senile insanity and in organic dementia from vascular disease.

Disease of the basal vessels, recognized by the naked eye, one observed in 70, or 53.2%, of the cases. A large percentage usually show disease of the aorta.
(3) "Lancet", Aug. 2nd, 1902.

(4) "British Medical Journal", March 1902.
aorta: Visible disease is more frequent in paralytics than in persons of similar age dying from other affections. Microscopically profound changes are seen in advanced cases. The first change appears to be in the vessels, especially in those dipping in from the pia. There is proliferation of the endothelium, the cells often being large. The adventitia is considerably thickened, has a fairly clear, hyaline, look, and the cells are large and often spindle-shaped. Just outside the vessel one observes proliferated nucleated cells, often fairly large, and not staining very deeply - connective tissue cells. Leucocytes are seen there in some cases, but I have but rarely seen what could be called a leucoclosis. Probably connective tissue cells have been mistaken for leuco-ty. In cases dying from convulsions, the vascular changes are unusually prominent, and then leucocyte emigration is most likely to occur.
Fig. 2 - Visual changes under high power.

(a) mitotic proliferation of endothelial and connective tissue cells. 

(b) advanced state: hole in pigmented change nuclear proliferation.
The vessels are in such cases usually full of red blood cells. In the space
around the vessels granules of yellow
brown pigment may often be observed.
Atrophied has pointed out that in the
renal arteries and hepatic vessels
there are similar changes to those in
the cerebral.
The neuroglia usually shows proliferation.
This is specially seen in the outermost
layer of the cortex. The cells are
increased in size and number, their
vascular processes with their terminal
expansions on the vessels were being
unusually evident. But the neuroglia
hyper trophy is not essential, as in
some cases there is little change
seen. It is, I think, in cases of
cerebral softening that one sees
neuroglica cell proliferation most
marked. The best examples of Deiter's
cells are seen, I think, in the tissue
at the margin of softening, old
haemorrhages, tumours, etc. Many
observers doubt the accuracy of
Fig. 1. Section of cortex of typical case, dealt atop convulcrons (Hess's blue & erythrosin).

Note: thicker parts = vessels, & numerous nuclei in pia mater substance.
Devan Lewis' "scavenger cell" theory. It appears to me that the fact of these cells being specially evident round areas of softening, etc., rather support the idea that they can ingest degenerated products. But the neuroglia hypertrophy, when present, may be merely compensatory in general paralyzation.

The notice a large number of nuclei of cells scattered through the cortex. These are probably mainly the connective tissue cells of vessels, endothelial or adventitial, or neuroglia cells. Some may be the mesoglia cells of some observers.

The nerve cells show all phases of degeneration from a slight chromatic change to marked atrophy. Many of them disappear. Increase of pigment, loss of processes, and disintegration of the nucleus, occur.

The cord in paralytic may be slightly or greatly affected. The most common obvious change is a slight sclerosis of the lateral columns. There is a degeneration of medullated nerve fibers.
Fig. 3. a) Section of typical S.P. 4th Cervical x 5.
   a) Note asymmetry of anterior columns. There is comparatively pale staining in region of fewer pyramidal tracts due to degeneration also of periphery—possibly artificial.
   b) Healthy fibres from post. column near commissure.
   c) Degenerated fibres from lateral column (oruses pyramidal) — high power.
   d) Natural size — 4th Cervical — same cord.

   e) Ditto — 12th dorsal.

Even here the asymmetry can be seen, left anterior column is larger.
shown by pale staining with the heidenhain-
Pal method. In some cases one
observes degenerative changes in the
cells of the anterior cornua, or
disappearance of nerve cells. In one
case with marked pyramidal for three
weeks before death, I found
diminution in number of the cells of
the anterior cornua with chromatolysis
of those remaining, sclerosis of lateral
columns, and degeneration of medullated
nerve fibres. Incidentally I may state
there was even marked asymmetry of
the parts about the anterior median
fissure, due apparently to complete
decerebration of the pyramidal tract on
one side. There was no direct pyramidal
tract on one side, while there was
marked bulging on the other. This
could be traced into the lumbar
region, thereby showing that in this
case as an event fibres of the
direct pyramidal tract descended
as far. There was profound
trophic disturbance, large blisters of
almost
Fig. 4. Stages of degeneration of the large Betz cell of cerebral cortex.

a) The first cell is almost healthy, slightly chromatolytic; second has more chromatolytic with pigmentary degeneration, third even more.

b) Chromatolytic is complete.

c) Cell is shrunken and wasted. Outside in all the nuclei round the cells. The nucleolus is always well stained.
almost the entire cuticle developing on the lower limbs. This may have been connected with the nerve cell changes in the anterior cornea. The cord changes in general paralysis in non-tabetic cases are on the whole unimportant. Marie holds that both posterior and lateral changes are of a poliomyelitic nature, so that posterior column changes may be from the cells in the spinal ganglia via the nerve roots. There are cases with slight sclerosis in the posterior columns as well marked as in the lateral, apart from tabs. I noticed this specially in a case occurring in a boy of 16. Bevan Lewis explains the cord changes in non-tabetic paralysis by assuming that they depend on vasomotor paralysis of the vessels in the cord due to disease of the vasomotor centres in the cerebrum. The dilated vessels press on the nervous elements. I believe the cord fibre changes to be due partly to disease of the cortical nerve
(7) Journal of Mental Science, April 1894.

(8) Mickle, Brain, 1894.
nerve cells and sometimes cells of the spinal ganglia, and partly in those of the brainstems in the cord.

One fairly often sees tabs and general paralysis combined. The tabs, in my experience, appears to have existed first. I believe there to be no necessary connection between the two diseases. Such cases are probably always syphilitic.

The cranial and spinal nerves usually show some degeneration. Campbell (7) in 12 patients found the vagi extensively diseased in all cases, the mixed nerves usually so, and the more peripheral, the more severely. Imlach (8) says there is a degenerative remissions of peripheral nerves. The muscles are secondarily degenerated.

In demonstrating the above changes in the nervous system, the methods I have adopted have been as follows.

The fresh aniline blue thick method of Bevan Lewis is suitable for showing proliferation of axonoidia, and also to
to a certain extent vascular and nerve cell changes. For other sections I have always embedded in paraffin. Hirst's method shows the vascular appearance as well as amy, with its various modifications - bluish, thining, polychrome. The nerve cell change are also well shown. Van Giesen's stain is not of much use except for showing sclerosis of the cord. Sometimes with this stain however, I have seen bodies like horn bodies in the nerve cells. The hex-gal pal method is the best for observing degeneration of wiggledated nerve fibres. Stülcke's method shows sclerosis in the cord very well, so may anilin blue black. I have not used the platinum nor methyl violet methods.

Changes in non-nervous organs may be referred to. The cardiac muscle is often pale, soft, and undue friable. Aortic atheroma is usual. Embolar heart disease is not specially common. Arterio-sclerosis
(6) Gazette des Hopitaux, Jan. 1894.
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Hind's method shows the vascular appearance as well as any, with its parerous modifications - tincture, thionin, polychrome. The nerve cell changes are also well shown. Van Gieson's
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nearly friable. Aortic atheroma is usual. Valveless heart disease is not specially common. Arteriosclerosis
19) general Paralypi
is present in most vessels.

Bronchitis and some emphysema are often met with. Lumen of broncho-pneumonia is frequent. Any pneumonia is occasional. Gangrene is not uncommon. Phthisis is common.

In whole found present phthisis in 26%, cured in 12%, 18% in all. Congestion is usually present. The gastric masticating tract is often somewhat atrophic. Pigmentation at various places is often seen. Colitis or asylm dyspepsia is very frequent in paraplegia. All sorts of ulcers are seen, with recent or old haemorrhage.

It has occurred to me that old haemorrhages put down to previous colitis may really have been of an acerbate nature. Scoury of the gums is very liable. The mesentery glands are often enlarged. The liver is usually, I think, fairly healthy.

The kidneys often show cortical wasting, granular kidney may occur. Cystitis is relatively frequent.
One may make the statement that pathologically general paralytic represents premature senile decay. The appearances are practically identical. Any variation may be considered as due to the more acute course of the former malady. Erosion of the cortex on stripping the pia is, I think, the principal difference. But this may be accounted for by the great severity of the degenerative lesions in the cortex and the proliferative lesions in the pericortical attachments. The changes in the skull-caps and dura, the opacity and thickening of the pia, the increase of fluid in pia and ventricles, the brain atrophy, the ependyma granulations, the neuroglia proliferation, the vascular nerve cell changes are all of the same nature.

**Duration.** In 160 consecutive deaths from general paralytic at the West Riding Asylum, Wakefield, I found that the average length of residence
in the asylum was 18.9 months. Most of the cases, of course, show signs more or less marked on admission, and presumably then brains would be diseased six months before admission on an average. But even granting the disease to be demonstrable 9 months prior to admission, two years for the entire disease from the prodromal symptoms to death is surely less than was formerly thought to be the case. The cases mentioned are from January 1896 to December 1902. The fact of these patients living only a little over a year on an average in the asylum appears to me to show that the disease is more rapid in its progress than formerly. "Galloping" general paralysis is by no means uncommon, and the analogy to phthisis appeals to one. When at Newcastle Asylum I investigated the duration of the disease there, and found it to be 18 months on an average taken over 35 years. 18
Month was found to be the average duration of the disease at Wakefield by Hitchcock a good many years ago. Hitchcock refers to this in his text-book on general paralysis. I have, I think, proved that at all events in the West Riding of Yorkshire the duration of the disease has lessened.

(10) Racehe gives, however, 2½ years as the entire duration in 136 cases, and 2½ years in men, 1½ years in women, in 337 cases. My cases of course refer only to extra-asylum life. In women the disease certainly lasts longer as a rule.

Age. The age on admission appears to remain much as before. Of 202 consecutive cases from January 1896 to December 1902, 40. 6 years proved to be the average number. 2 were above 60 (63, 65), 11 under 30. One died at 16. Of 218 cases at Newcastle, 6 were less than 15, 11 more than 55.
Symptomatology. I shall not attempt to dwell fully on the symptoms. When one considers the pathological basis of the disease, involving as it does, practically the entire nervous system, no single mental or physical nervous symptom need cause wonder. It depends on which parts are affected first. Usually there is a prodromal alteration of conduct, self-assertiveness, etc. This goes on to mild manner, then later pronounced manner with in the classical type grandiose delusions. Later then is a happy dementia, then absolute dementia, then death. But many other types are met with. Thus then is the simple dementia type. The melancholic type (common in women), the uncloud type, the delusional type (i.e. delusions apart from fleeting grandiose ones). But all these are secondary to the one important fact, the mental sign of general paralysis, i.e. that mentally it is essentially a progressive dementia.
It appears to me that the simple dementa type is now becoming most common. Of the 202 consecutive male cases already mentioned, 92 only had grandiose delusions or an exaggerated sense of bereft. Further every year appears to diminish relatively the number of cases with those signs. Of the 92 cases with grandiose ideas, 56 were from January 1896 to June 1899, and 36 from July 1899 to December 1902. At the same time melancholia in general paroxysm does not appear to be more common. We have instead a simple dementia. There are also many cases resembling imbecies more than any other type of mental disorder.

As regards the physical signs, prominence has always been given to oculomotor abnormalities. Inequality of the pupils is said to be unusual. In the above series of 202, on admission 100 had equal pupils, to 91 unequal, the rest being unascertained.
Reference as before
In advanced cases, inequality does not appear to be more common. In 18 advanced cases, I found 13 equal, and 5 unequal. Of course temporary inequality may occur.

As regards the reaction to light, one finds marked alterations usually. In the above series, 40 reacted normally on admission, 96 sluggishly, and 62 were tried to light reaction is not ascertained. When, for example, one pupil is sluggish, the other, I have recorded the greater abnormality, i.e., fixation. Raedeke, in 136 cases, found 54 with 2 fixed pupils, 10 with 1 fixed, 38 sluggish. In most cases, even if the reaction is normal on admission, slow abnormalities in this respect later, and if abnormal on admission, the abnormality becomes more marked. In 18 very marked cases, I were normal, 6 sluggish, and 10 fixed. Papillary irregularity in outline is not infrequent. The earliest papillary sign is probably loss of

reflex.
Reflex dilatation to cutaneous stimulation. Then in order come diminution of or loss of consensural reaction, then of direct reaction to light, and lastly of accommodation.

The most constant physical sign in the writer's opinion, even in early cases, is the speech defect. Thus only 88 of the 202 cases showed a clear articulation on admission, 159 were slurred, 10 not ascertained. At the same time one must be careful to distinguish a true slurring from simple defect in enunciation. So suddenly ask a patient of the artisan class, who is probably somewhat "nervous," at the time to say "biblical criterion" is asking him to perform a work of some difficulty. Still there is little doubt that the occasional slurring is usually noticed early in general paralysis, and is the first physical sign to the patient also.
One usually observes alteration in the deep reflexes. Thus in the above series 39 only had knee-jerks normal on admission, 95 increased, 25 diminished, 40 absent, 3 not mentioned. In the few cases with knee-jerks varying to each limb, one records the greater abnormality. Increase of knee-jerk is then the most usual. In Ranke's 136 cases, more than half showed increase.

Tremor of the extended arm is nearly always to be detected, as well as of the tongue. In late cases this is extremely marked, the whole face frequently going into contortions at the attempt to speak. Tremor is a very early symptom, but is not of the same diagnostic value as slurring speech.

Convulsions occasionally are the focal signs to draw attention to the patient. Not uncommonly a patient is
months or even years in an epileptic ward before he is found to be a paralytic. The post-mortem examination may be where it is discovered. The convulsions may be epileptic, epileptiform, apoplectic, or apopleptiform. Possibly the last is the most common, then epileptiform, then epileptic, then apoplectic. I have been struck by the frequency of epileptic convulsions. Possibly the most usual phenomena are as follows. The patient loses consciousness, is paralysed, convulsive movements occur generally, usually principally on one side. They stop and a period of quiescence ensues till the next convulsion. Or a continuous twitching may go on. The temperature is raised, the breathing starts torow. The convulsions diminish in severity, then cease. Paralysis continues for some days. Localised twitching often goes on for days. After about a week the patient appears much as before.
though each attack of convulsions marks a step forward in the malady. Many varieties of convulsions may occur which space cannot permit describing. I would only remark that paretic attacks are very common.

Other physical signs may be more briefly mentioned. trophy skin disorders are fairly common. Blisters on the feet or hands are, I think, the most common. Occasionally the whole of cuticle is desquamated from large areas, especially on the legs. Some dispute the existence of neurotrophic disorders, but these blisters, etc., are seen in the entire absence of local irritation and it is difficult to account for them otherwise. The skin in general paralysis is often of a dull, earthy tint. Sometimes it is somewhat greasy. The temperature is usually relatively high, especially as so many of the insane have abnormal temperatures. Sensory impairments are common.
Thus we have diminished pain and heat sense, delayed reaction time, impaired muscular sense. Toulouse and Vacherot (1870) found 8 of 18 female general paralyses anemic. The anxiety sense is not often much impaired. There is no distinct we gait. A slight apraxia as in moderate alcoholism intoxication is not uncommon in fairly early cases. Spastic, tabetic, and cerebellar gait may be met with.

The "mark" expression may be met with.

Diagnosis. The diagnosis is often very easy, at other times it presents great difficulty. The grandiose mania, slurring speech, tremor, pupillary abnormalities, etc., combined, are pathognomonic. But many cases are much more subtle. First take acute mania. A patient is admitted violently excited, restless and noisy. Sometimes one fancies there is a slur in the speech. Pupillary examination is unsatisfactory, or some
Sluggishness or inequality may be imagined. One cannot then say if general paralysis be present. He is fed up, gets fresh air, bowels are kept regular, and in any time up to say 6 months he has apparently recovered. He is carefully examined. One detects slight traces of tongue, inability of hands, or slight papillary inequality or sluggishness, perhaps a slurring in speech. Mentally he appears sound. He is kept in at least a month after all mental symptoms have cleared up. There is no recurrence. He is discharged. I maintain that in such a case you still cannot be sure that in a few years' time that man will not be dead from general paralysis. Should the man last over three months, one strongly suspects general paralysis, and most of such cases are rightly so suspected. Other somewhat similar cases occur where there is little or no menia; indeed examination on admission may reveal little or nothing wrong mentally, yet he has these slight physical signs. Here again
(12) Journal of Mental Science, July 1902, p. 572
again one cannot be sure, but the chances are more against general paralysis than if mania had been present. Some maintain the disease may last 20 years in such cases. I have myself never seen a death from general paralysis of more than 6 years duration since diagnosed or suspected. It is, I believe, one of an asylum medical officer's most difficult duties to decide whether to recommend discharge or not in such cases. From my own experience, I should say keep in as long as possible, especially if there be signs of syphilis. Only too often have such cases been discharged on a remission occurring only to return within a few weeks or months utterly broken down, and soon afterwards to die of what is practically “galluping general paralysis.” Even a few days they may return. Usually too, when outside, they indulge in all sorts of excesses, and indescribable harm is done. The system of “discharge on trial” is a beneficial one in such cases.

Another type of case is that of a man,
day 64-5 years of age, who shows a
tendered mild dementia, speech slurs, his
pupils are unequal and fixed or sluggish.
This implies as an event that there is not
such chance of recovery, but such cases
are often cases of cerebral softening, or
proven cerebral vascular disease.
Semia symptoms may too be present.
Alcoholic elements may resemble paralysis
The absence of excitement, special loss of
memory, and the alcoholic that hemi diagnosis
from the last two types a few notions
acuteness will distinguish from general
paralysis by bringing out the essentially
progressive character of the latter.
Thus we see that from acute wax, some
cases of chronic wax, organic
dementia, and those cases with slight
physical signs and little or no mental,
general paralytic has more frequently to
be diagnosed. More rarely we must
diagnose from other states. I have seen
cases whose mental state almost exactly
resembles uncertainty fairly marked. The
expression is also similar. If there be
no history, they are very apt to be considered simple indiscretions. The history may indeed simply be one of inanity. Physical signs of course form the diagnosis here.

Cases of syphilis from disease may give similar physical signs. The absence of progressive dementia should distinguish such.

To before stated, convulsions may be the first sign and the case considered one of epilepsy. There is usually in such a case some dementia. If convulsions or dementia be both of short duration, general paralysis should be suspected. In most the physical signs in time assert themselves, but death may occur before general paralysis is diagnosed.

Occasionally it must be diagnosed from the varieties of melancholy, especially in women. The physical signs usually suffice.

From dementia due to alcoholic decay, it is questionable if it has to be diagnosed, for possibly they are the same thing.

From non-mental disease distinction must be made. Insular sclerosis is perhaps the most similar. Hypsagnus, gait, pronouns
tobacco habit, its longer duration, and earlier age of onset, are its most distinguishing features. From locomotor ataxia, it is to be distinguished by the absence of mental signs, behavior, and speech stuttering in this disease, with its different mode of onset. It may, of course, develop in the course of tabes. Milder stages 1% of paralytics have had tabes previously. Lead encephalopathy resembles general paralysis. Indeed lead may be an accessory cause, as alcohol ophthalmia.

The usual signs of palsy must be relied on for a diagnosis. Tumors is distinguished by headache, vomiting, choked disk, slow pulse, and localizing symptoms.

Treatment. A few remarks may be made. Needless to say, no cure is known. Could the theory that it is due to a specific toxin be proved, we might of course hope for an antitoxin. I shall refer later to diphtheria antitoxin. Some cases show well-marked
well-marked physical signs before mental.
Others once were, most both concurrently.
In the latter two asylums treatment is usually indicated at once. In the former it may
be delayed, and fresh air, good diet,
regular purgation, and a well-regulated
life insisted on, until the mental phenonem
render an asylum imperative. In early
cases one may try purgative treatment. Blue
pill 5 grs. given 3 times a day, with a
dose of sulphate and carbonate of magnesia
every day, appears in some cases to be of
benefit. I have seen a case which appeared
an early typical one discharged "recovered"
after the treatment. Dr. T. P. Hearder, now
of Clifton, York, was the administrator. In
later general paralysis I have found it
of no use. The rationale is the idea
of gastric intestinal auto-hystoeisation.
Restorial antispastics are of little, if any,
use. Antispilitic treatment was
regarded as not worth trying. Some
recommend mercurial mixing or
intravenous or sub-injection. For an
ordinary case, however, we must simply
observe the rules of hygiene, especially
avoiding constipation, and treat the
symptoms as they arise. If the mania
is severe, chloral hydrate is I think the
best sedative. 7½-15 grs. may be given
thrice daily, or 20-30 grs. may be ordered at
a time. Potassium bromide or chloral, in
the proportion of 2 to 1, may be given
instead. Paraldehyde from 7 to 15 grs.
may be tried for nervous and restlessness.
Sulphonal is rarely of much benefit,
being possibly rather better for general
paralysis is not the kind of mental
affection in which I recommend hypnotics.
Chloral is, I maintain, the best sedative.
In bedridden cases, who among other
parents and attendants by their constant
care, grinding of teeth, etc., small doses
of chloral are especially suited. Compound
are to be avoided mainly by preventing
constipation. When occurring, especially
if frequent and severe, or resembling
status epilepticus, an enema should
be ordered, then 30 grs. of chloral by
the mouth, or 7½ by bowel. This
may
may be repeated in an hour or two if necessary. It is surprising the amount that can be borne. Strongly is of benefit, if the temperature be 103.6° or over. Cauterization will probably be necessary. Paralytics are occasionally, though very rarely, suicidal. I have seen 2 cases, who attempted suicide, one by strangulation, one by falling forward on a projection, making it strike his forehead. Some cases are constantly picking up things in the airing court, stealing, etc. They should be searched. They may swallow stones, etc. As the disease advances, care must be taken to prevent falls, choking, etc. They should not be allowed to sit too much on hard seats, and no stove or saucers or butchers are liable to be so caused. Once bedridden, the prevention of bedsores forms one of the main duties of an attendant. A water-bed is advisable. Skin should be rubbed with methylated spirit, his position frequently changed, and means taken to prevent wetting with
Wine. He should lie on a drawn sheet. If a small bed sore form, it should be covered with lint. If fixed on by strip of plaster, large sloughing sores must have their sloughs removed by poulticing. He should, of course, be much as possible on sound skin. Care must be taken to prevent falling out of bed. Apart from colitis, a slight mucous diarrhoea is common. Astringent enemata may be given. Sewing is very liable. The slope they get should have vegetables, and 3-4 lime juice given three daily. One of the main phenomena requiring treatment is retention of urine. Bladders should be frequently examined, although urine is stated to be passed, although no urine is being passed naturally, catheterize night and morning. Crystals should be treated by bladder irrigation. Eucalyptus oil does very well to dip the catheter in before passing.
Etiology. Personally I have found a history of alcoholic excess more often than any other known cause of insanity. Alcoholic excess I found to be the principal cause in 101 out of 228 cases at Newcastle. But, whether or not as the main cause, it was found in the large majority of cases at Wakefield. Pathologically we know the resemblance between alcoholic dementia and general paralysis. But as much as alcoholic insanity has not the progressive rapidity of general paralysis, alcohol alone must only be accessory. Hereditary predisposition is possibly less marked than in some other varieties of insanity. I found it in 37 out of 228 cases at Newcastle. Probably a hereditary predisposition to the nervous diseases in many others. Statistics are not of much value on this point. Blanchard found it in more than half of 37 cases (alcohol nine, 26). Sexual excess is an important factor. The relative frequency of its occurrence amongst the married has often been shown. Only 3 of 32 female paralytics at
Newcastle were single.

Trauma is often given as a cause by the friends. It may in some cases cause a "locus minoris resistentiae."

Excess of work predisposes, or does any kind of exhaustion, mental or physical. Other ascribed causes may be dismissed with his exceptions.

Syphilis may regard as essential.

One undoubtedly finds evidence of syphilis in paralysis than in insane or insane persons dying from other the usual causes of death. In 18 advanced cases, I found 4 with penis scars, 1 with other signs, 9 with no signs, I doubtful. A penis scar is not pathognomonic of syphilis, but in 3 cases out of 4 may I think be said to be specific. Further, the effective treatment of primary and secondary syphilis doubtless conceals lesions that would have existed otherwise. The coincidence of locomotor ataxia is in favour of syphilis. The failure to inoculate syphilis in several advanced
(32) Journal of Mental Science, July 1902, p. 776
paralytic would appear strongly suggest.
we, but there are apparently some
doubts as to the accuracy of this, and
the number of cases is not large enough.
Pathologically there is some resemblance
Thickening and infiltration of pia,
and the changes in the smaller bloodvessels
are similar. Again while it is stated
that the early general paralytic is just
the man to acquire primary syphilis,
I have never seen nor heard of a
case admitted with a primary sore, while
I have seen several with gonorrhea,
thus showing that they certainly expose
themselves to infection. But,
notwithstanding these facts, there are
many cases in which one fails to find a
trace of syphilis, and yet no history of
it whatever. Can one assume that a
person may get a mild attack of syphilis,
and never know of it, or develop
tertiary manifestations? There are cases of
syphilis with no primary symptoms, or
some with no secondary without treatment.
Many with treatment of course have no
secondary. It is conceivable that there may be a form of syphilis without these symptoms which might cause general paralysis as a tertiary phenomenon, but we have no proof of this. Has a gunna ever been known to be the first sign of syphilis? I think not. Again, if paralytic dementia be syphilis, why do we not get a history of syphilis as often as in locomotor ataxia, i.e. in about 90% of cases? One point I would concede. I have never seen general paralysis (except in juvenile cases where congenital syphilis may play a part) in a person who has not had the opportunity of getting syphilis. There are of course other arguments. In the tropics with thousands of cases of syphilis there are practically no cases. Again, numerous cases of syphilis can be traced in this country who never acquire general paralysis. Anti-syphilitic remedies in this country as an event are useless. Of course the same may be said of locomotor ataxia. Pathologically the evidence is rather stronger against than in favour.
of syphilis. I have never seen a gumma in a paralytic alive or dead. Typical
endarteritis is rare in general paralysis, as also are aneurysms, militancy or otherwise.
Indeed one very seldom meets with evidence of syphilis in the internal and nervous
syphilis as a post mortem. Internally the
penis scar is the commonest sign, with
suspicions parasites on the skin, and
possibly slight thickening of the bone base,
as far as I have observed. At present
we can only say that syphilis is a
frequent predisposing cause. 2) it
cannot be proved to be essential.

We now come to the toxic theory, viz.
that general paralysis is caused by the
absorption of toxins produced by organisms.
Recently much attention has been
directed to the observations of D.
Robertson, Macneal, and others in this
connection. They have found the
alimentary and respiratory tract to be
the seat of a severe toxic infection.
Organisms resembling the Klebs-Loeffler
bacilli have been found especially on
The mucous surface of the stomach, and on tonsils and bronchi, culture can be made on most media, but the best appears to be lyso-haemoglobin agar. Intraperitoneal injection in rats caused death in 5 days with severe local inflammation. Several rats were fed with bread and sterilized cultures for weeks.

For a month they were fairly well. Then slowness and uncertainty of gait, drowsiness, developed; later motor weakness, incoordination, dyspnoea, drowsiness, disequilibrium. Control animals remained healthy. The post-mortem changes were: gastro-intestinal catarrh, especially of the upper part of the small intestine, proliferative and degenerative changes in the liver, pneumonia, cortical nerve cell degeneration, acute periventricular neuroglia proliferation, especially in the first cortex layer, neuroglia proliferation, and infiltration of pia-arachnoid.

Similar changes were in the cord, but the degeneration of nerve cells was more advanced. Many nerve fibres were degenerate. Even if the culture feeding were stopped, the morbid
hoped phenomena went on. In criticising these observations, I shall first state what appear to me to be the points against the bacterial origin of general paralysis.

1) It is not stated whether these organisms cannot be found in similar situations in persons dying of other affections.
2) nor is it stated if they are found in early cases.
3) They are not found in all cases.
4) Attempts to obtain specific agglutination action of blood serum on culture are negative.
5) Antitetanic serum has diminished some complications, but has no effect in shortening the duration of general paralysis.
6) The pathological changes, while suggestive, are not identical with the typical signs of general paralysis.

In favour of this theory are the facts:
1) The bacilli is found in rare cases.
2) The clinical symptoms are those of a progressive decay of the nervous system.
3) Antitetanic serum arrested some complications.
4) nerve cell degeneration, neuroglia proliferation, and to a certain extent infiltration of pia-arachnoid are similar lesions to
those in typical general paralysis.

But, I summarise, the changes on the whole appear to me to be of an acute encephalitis rather than the slow chronic degeneration essential to general paralysis. It is, I think, very probable that the toxins act in general paralysis, but there is no proof, I fear, that their action is not terminated on an already weakened organ.

So far as our present knowledge goes, we may state to probable theories:

1) General paralysis may depend upon an inherent specific loss of vital energy in nerve cells, or possibly in many kinds of cells. This may be congenital. The process is then a progressively degenerative one.

2) It may depend on a loss of specific vital energy induced by alcohol, syphilis, arsenic, etc., with or without congenital defect.

3) That given this loss of energy, either congenital or acquired, the disease cannot progress unless toxins are absorbed which induce the profound degenerative changes.
1) The disease may be induced in a perfectly healthy brain by a specific toxin, or by several toxins.

For the first, we must assume the person to lead a perfectly healthy life. This is rarely found to be the case. The second accounts for the majority of cases if we disbelieve in a true agency. The third is, I think, more feasible. We have first of all a weakness usually acquired by alcohol or syphilis, but not proved to be always so, of the nerve cells, and possibly some of the vessels, dura, cartilage even, all the cells of the body. This does not go on to produce degeneration incompatible with life unless brain act.

We all absorb toxins from time to time, but with comparatively healthy cells, we can withstand them. The paralytic cannot. First a few cells get degenerated by toxins, more strain is thrown on others, they in turn more readily account, so things go on till an intercurrent malady cannot be resisted, and death occurs. This may also account for senile.
Menil decay. We do not know what menil decay is. Perhaps it is a progressive degeneration due to instability of neural functions due to invasions. The全力打造 certainly cannot be considered as proved. But the work in connection with the bacteriology of general paralysis must be continued, and at the present time it appears likely to be one of the most fruitful. That an acclimatisation physician can pursue for the benefit of mankind.