HAEMORRHAGE INTO THE INTERNAL CAPSULE WITH SPECIAL REFERENCE TO THE AFTER GAIT

THESIS

by

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During the last five years, I have had under my sole charge in private practice the care of ten cases of Haemorrhage into the Internal Capsule. I have made a practical study of these cases, by writing, graphic methods and by photography. These notes and records form the basis of this thesis.

I shall endeavour to make it as practical as possible and shall therefore give an account of these ten cases and then state my own observations regarding the Etiology, Pathology, Prognosis, Symptoms and Treatment of these ten cases.

A most important question after cerebral haemorrhage is "Will the patient be able to walk again, and if so to what extent?" I have made this subject of AFTER GAIT a special study. I shall show by photographic records the characteristic gait that comes on after a cerebral haemorrhage into the Internal capsule.

I shall describe the first case - that of A.P. more fully than the others as it is a very typical case and serves well to illustrate the symptoms of cerebral haemorrhage into the internal capsule. The after gait in each case has been recorded in which the patient was sufficiently able to walk, to make an intelligent record.
ACCOUNT OF THE CASE OF A.P.

A.P., male, age 37. The patient was an engraver. Previous to the hour of his attack he was in his usual good health. No premonitory symptoms were present.

While at dinner on the 1st April 1904 he felt a severe pain in the left side of his head. He cried out from the pain and put his hand to the left side of his head. Almost at once he subsided to his left side and fell to the floor.

I saw him about fifteen minutes after this. He was lying on the floor, unconscious. I was told that when the attack first began that the patient shook as if he was in a fit and ground his teeth.

When I first saw him the arms and legs were flaccid. The teeth were clenched. The pupils were equal and contracted, and no corneal nor light reflex was obtained. The knee jerks were absent in both legs.

The heart was beating irregularly and the pulse was irregular and small. The breathing was stertorous.

AFTER SIX HOURS he was still unconscious. The temperature was 97.4°F. The corneal and light reflexes
had returned and movements in the left arm and leg were observed. The head and eyes were turned to the left side. Feces and urine had been voided unconsciously.

**NEXT DAY** consciousness had returned and the extent of the damage was apparent. The right arm and leg were totally paralysed, as also was the right side of the face except for the right orbicularis and occipitofrontalis muscles which were only weakened. Total motor aphasia had come on. He could not originate words nor repeat words. Swallowing was performed with difficulty. Food and saliva escaped from the right angle of the mouth. Slight regurgitation of fluids through the nose occurred on swallowing on several occasions. This however passed off in a few days. He lay all day semiconscious taking no notice of any one, and not recognising even his own wife. Conjugate deviation remained very marked. He refused to make any attempt to speak.

The **THIRD DAY** he made attempts to speak but only
made a noise as if he was trying to say "Ah, Ah, Ah," and failed absolutely to make himself understood. His temperature rose to 99.4. The pulse was 96 per minute. The same evening he became restless and moved his sound limbs freely. He seemed annoyed at his paralysed hand and picked it up frequently with his sound hand and let it drop impatiently. Tactile and painful sensations were unaffected. He now attempted to speak but could say nothing. Many attempts were made to make him say "Yes" but without success. When shown a knife and asked if it was a pencil he signified displeasure and irritability and shook his head. When asked if a knife was called a "Knife" he showed pleasure and nodded his head in approval. He could not write owing to the paralysed condition of his right arm. On trying to make him write with his left hand I found he could not form a single characteristic letter. He thus had total aphasia, agraphia, and alexia, but could understand what was said to him.

By the EVENING OF THE FOURTH DAY the pulse was 104 per minute and the temperature 100.2 F. The pulse remained quick and the temperature up for a few days and then gradually fell to normal by the eighth day. This showed a state of irritation of the heat centre by inflammatory thickening round the blood clot in the brain. There was still no rigidity of arm nor leg. Slight movement returned to the leg by
the third day. and by the end of the first week the hip knee and ankle joints could be freely moved. Speach began to return on the fifth day of the disease. The first word he said was "Yes" and he kept on saying it in a very unmeaning sort of way. After another two days he was able to repeat simple words spoken to him though he still kept on saying "Yes, Yes, Yes" in the same unmeaning way. It was now apparent that this "Yes, Yes, Yes," was his attempt to say other words. He could recognise himself that he was not saying what he meant to say. This annoyed him and he tried harder to say something I could understand, but he only made a noise as if he were saying "AH AH AH Yes yes Ah Ah Yes Yes AH Ah Ah Ah Yes". After doing this for sometime he grew impatient and gave up the attempt. He gradually learned to speak again but he always spoke with great difficulty as if he were hunting up each word in the dictionary of his brain before he could utter it. He frequently pronounced a word wrongly but at once recognized it and proceeded carefully to correct it. After six months when his speech was at its best he talked in a staccato way and in a monotinous tone of voice. The power in his arm began to come back after eight days. He could first shrug his shoulder and then flex and extend his elbow. Power never returned to his hand. He was able to move his thumb and forefinger slightly but the movements in the other fingers of the right hand was
was always so slight as to be almost unnoticeable. Rigidity began to come on about thirteen days after the stroke occurred. The fingers grew gradually flexed, the wrist became flexed also the elbow and the arm became quite useless. His power of walking had so far returned after six weeks that he was able to go for a walk of half a mile. At the same time he began to be very depressed. The thought that he would never be able to work again was what depressed him most of all. He seldom broke down and cried though on one occasion I saw him do so. He gradually refused to go out of the house, but when he did go out he walked round a deep pond in a way that suggested that he contemplated suicide. He was carefully watched to prevent this, but he managed to take advantage of his wife's absence and hanged himself in his own bedroom. This happened ten months after his first attack. No autopsy was obtained.

During the chronic state preceding his death rigidity was well marked and affected his right arm and leg. Increased knee jerk, ankle clonus, and wrist jerk were present. Sensation remained unimpaired except over the upper arm where there was hyperaesthesia. The muscles of the affected leg and arm wasted greatly. A characteristic gait came on. The following photograph shows the marks left by his feet when he walked along a long sheet of white paper with the soles of his feet blackened with a black paste.
He had not been accustomed to walking barefooted and found it somewhat difficult to preserve his balance when he did so. After a little practice he did so with ease. This no doubt was due to the hemianesthesia of the sole of his right foot. He had a very characteristic way of walking. The accompanying photo shows the following points regarding his way of walking:

1. The right foot is not properly raised on taking a step. The result is a scraping of the toes (except the big toe) on the ground, this is due to paresis of the anterior tibial muscles causing the toes to be

   (A) pointed by extention at the ankle
   (B) inverted to the middle line

Thus the four outer toes are the most dependent and scrape the ground while the big toe does not scrape due to inversion at the ankle raising the big toe.

2. The foot is everted. This is done to give more balancing power and is not due to paresis of special muscles.

3. The right foot is not advanced so far in front of the left, as the left foot is advanced in front of the right. This due to the weak condition of the right foot. In fact the patient does as everyone does—he puts his best foot first.
Foot prints of A.P. to illustrate the points mentioned in the last page.

(4) The right toes scrape the ground twice, usually, with each step - once on raising the foot from the ground and once just before placing it on the ground.
Account of W.S.

W.S. male age 65 was in his usual good health till 5th Jan 1904. On that date he was going to a meeting at which he had to make a speech. He fortified himself for the occasion with a little whisky and shortly after taking the stimulant he complained of a numb feeling coming over the right side of his body. Extreme weakness of all the muscles of his body came on but he did not lose consciousness.

I saw him about fifteen minutes after the onset. He was then sitting propped up in a chair, hanging his head forward and mumbling words with no meaning. Both sides of his body seemed to be paralysed but on careful examination it could be seen that the right side was absolutely paralysed and that there was some power left in the left side. The face was congested, the pupils were equal and moderately contracted. The breathing was noisy and groaning but not stertorous. The right cheek was puffed out at each expiration. The left cheek was also puffed out but to a less extent. Improvement came within six hours. For a short time he complained of seeing everything double. This very soon passed off. No strabismus could be detected.

By the next day the patient was quite conscious and able to speak and converse. His speech was a little thick but this passed off completely in a few days and he was able to speak as he did before his attack.

The right muscles of the face (except the frontalis...
and the orbicularis palpebrarum) the right arm and the right leg were completely paralysed for motion. The sense of touch was much impaired. He could feel when he was nipped but it took him about five seconds before he realised that he had been nipped. By the second day his temperature rose to 99.6. The tongue was furred and the patient felt generally uncomfortable. The arm and leg affected showed slight early rigidity. The knee jerk in the right leg was much increased. There was no ankle clonus. The plantar reflex on the right side was also much increased. No conjugate deviation was present. Urination was throughout performed normally. The bowels became constipated and have ever since required drugs to cause defeacation.

The patient remained in bed for three weeks, quite unable to get up. During the first week he was unable to turn in bed, then power gradually came to him and he was able to pull up his leg and to turn in bed. Soon he could shrug his shoulder and after that the power came to bend his elbow and last of all came the power to move his fingers. The right side of his face gradually recovered its power during the first ten days. The occipito-frontalis muscle recovered completely. The orbicularis almost completely. The power to shut the right eye without shutting his left never returned. The other muscles of the right side of the face gradually improved but always remained a
little weaker than those on the left side of the face. The abdominal and thoracic muscles were unaffected. The sensation to touch over the right half of the abdomen and thorax was diminished. The same hemianaesthesia obtained in the right arm, leg, and side of face.

After being confined to his room for six weeks the patient was sufficiently strong to be able to move about his house, and after another fortnight he was able to walk outside a distance of a quarter of a mile.

He went on improving for about six months. He was hopeful and bright and attempted to improve his health. After six months no more improvement came and he began to get despondent. He gradually became weaker and could not walk so far as he had done. Numbness and tingling and actual pain came on gradually all over the affected side of his body.

His condition now a year and a half after his attack is as follows:--

Paresis of the lower two thirds of the face, right side. The right arm and the right leg. He can walk a hundred yards at a time slowly and then has to rest. He has a typical gait as follows:--

He walks slowly and carefully. He keeps his eyes fixed on the ground about a yard in front of him. He swings his right leg round with each step as a cow swings her hind leg. He keeps his knee straight. He does so because he feels his leg more secure and
reliable when the knee is straight than when it is bent. He points the right toes outwards so as to give a broader base on which to balance himself. He scrapes his toes on the ground as he walks, with the result that he has had to have his boots repaired in the same place several times, due to the scrape it gets on the ground. He has always been a very careful man and is so still; this accounts for the fact that when I made the accompanying records of the way he walks the first record shows the walk of an ordinary individual except for a little turning out of the toes of the right foot.

**FIRST RECORD OF W.S.**

**POINTS TO NOTE**

(1) Short steps taken

(2) Turning out of right toes

(3) Slight falling of arch of right foot

"good"

(4) The "good" foot is advanced farther in front of the "good" foot than vice versa.
The second record of W.S. shows the same points as the first but in addition the fact that he scraped the ground with his toes as he raised his right foot to take a step. The direction of the scrape is outwards and forwards, principally outwards. This is due to the fact that he swings his leg round when he walks instead of bending his knee.
The following is an account of a case of cerebral haemorrhage which burst into the lateral ventricle and caused death.

THE CASE OF Mrs. H.

Mrs. H. was a laundress aged 56. For a year previous to her attack she had had indifferent health due to aortic regurgitation and albuminuria. The heart was much hypertrophied.

On 7th Feb 1903 she suddenly felt a pain in her head. She sat down and lost the power of her left arm and leg. I saw her ten minutes after the attack began. She had then nearly complete paralyses of the left arm and leg. She was quite conscious. The pupils were equal and contracted to pin points. She was perspiring freely. Her eyes were closed. She half raised the eyelids when asked to open her eyes. She could hear a watch ticking at her right ear and not at her left. (previously I know her hearing was equally good on both sides.)

Clonic twitchings went on in the right arm and leg. On asking her to put out her tongue the tip pointed to the left. The muscles of the left side of the face—except the occipito frontalis and the orbicularis palpebrarum—were paralysed. The knee jerks were present. The left K.J. was not so strong as the right. There was no ankle clonus. The pulse was 75 per minute with high tension.

After two hours she was still conscious, in fact more so than when the attack had lasted only ten minutes. She could answer questions but her speech was very slow.
thick and indistinct. She was kept sitting up in a chair during four hours after the attack began, and then she had a general convulsion. This took place as she was being laid down on a sofa. The cheeks became flaccid, being drawn in and puffed out at each breath. Loud snoring also came on. Mucus collected in the throat and was not coughed up. The breathing became irregular and slow. The pulse became irregular and small. The temperature fell to 96.5 in the armpit. The paralyzed side of the body felt warmer to the touch than the nonparalyzed side. All reflexes became abolished. Urine and feces escaped uncontrolled. She never again rallied and she died 4 1/2 hours after the beginning of the attack. An autopsy was obtained at which the following points regarding the brain were observed.

About three ounces of blood lay under the dura mater of the brain and extended down the spinal canal under the spinal dura mater. The vessels on the surface of the brain were congested with blood. The brain was put away to harden for a month in formalin. On clearing the surface of the hardened brain of the superficial arteries a gush of blood came on pulling out the striate arteries of the right middle cerebral artery. In the position where these arteries enter the brain there was a layer of brain substance about 1/4 inch thick separating the surface of the brain from a mass of blood clot and serum which occupied a cavity in the brain, that
could have held about ten ounces of fluid. Both lateral ventricles were filled with blood and the blood had broken into the adjoining brain substance and broken it up so that it was impossible to find the point from which the blood had originally come. No aneurysms on the small arteries could be found.

No foot record of this case could be taken but from the study of the case during life and the autopsy, lesson was learned: The patient had increased tension due to

(1) Kidney disease - probably granular contracted kidney
(2) Hypertrophied heart

Due to these causes a vessel gave way in the brain in the region of the right internal capsule. Bleeding went on slowly and stopped. For this reason the patient did not lose consciousness at first and in fact improved till she was changed from a sitting to a reclining posture. This change of position increased the tension of the vessels in the brain and started the bleeding again. The blood broke into the lateral ventricles and caused the convulsion. Bleeding then continued till death.
The following photograph depicts a record made a year and a half after a cerebral haemorrhage. It is taken from

**THE CASE OF J.K.**

J.K., a compositor, age 56, male, had a cerebral haemorrhage on the 24th February 1902. The course the case ran was very like that of A.P. described before, and there is no object in going into details regarding the case. A few main points of difference however must be noted.

J.K.'s age was 56

A.P.'s age was 37

J.K.'s longest walk after his haemorrhage was only about 20 yards

A.P.'s longest walk after his haemorrhage was about half a mile

J.K.'s power of speech returned so little that a stranger could not understand his words.

A.P.'s power of speech returned so far that he could easily carry on a conversation.

J.K. is still alive

A.P. committed suicide.

Thus A.P. recovered his power of walking to a much greater extent than did J.K. This point is well shown by a comparison of the two photographs.
PHOTOGRAPH OF A RECORD OF THE FOOT PRINTS OF J.K showing the following points.

1) Left foot normal, right foot affected.
2) Very short steps taken.
3) Left foot advanced at each step right foot brought nearly up to the level of left at each step.
4) Toes of right foot turned out more than those of left foot.
5) Great toe, second and third toes of right foot scrape the ground at the beginning of each step.
6) Arch of right has fallen a little.

In each of the former cases the right leg was the affected one. In the following case the left leg is the affected one. The following record was made as soon as the patient could walk, after his cerebral haemorrhage, i.e., about six weeks after the haemorrhage took place. The following is a short account of the case.

THE CASE OF W.F.

W. F., age 56, a clerk, on 6th August 1905, had symptoms of haemorrhage into the right internal Capsule, followed by left haemiplegia. For at least a year previously he had been under my care suffering from albuminuria with granular casts in the urine. On two occasions during that year he had had two uraemic attacks which
manifested themselves by a general convulsion followed by paresis of the left arm and leg. In both cases this paresis passed off in from 24 to 48 hours.

The present attack began with a general convulsion and loss of consciousness. Consciousness returned after sixteen hours but it was not complete till ten days from the onset of unconsciousness. During these ten days of semiconsciousness he appeared to be on the point of death every time I saw him. Improvement began about the tenth day and went on every day till after two months he was able to get up and walk with help. Whenever he could walk along a straight line alone, with the aid of a stick I made the following record of his footprints.

**RECORD OF W.F.**

Points to note

1) Left foot affected, right foot normal

2) Short steps taken

3) Right foot advanced farther in front of left than vice versa.

4) The toes of the left foot are a little more turned out than those of right foot.

5) Scraping of the toes of the left foot on the ground.

6) In the first two steps made by the left foot the heel also scraped the ground.
The following two photographs are shown to illustrate the gait five years after cerebral haemorrhage into the internal capsule. The patient A.N. has largely recovered from the effects of his stroke of five years ago. It still affects his walking a little. The first photograph shows:

(1) That he took short steps
(2) That he turned out his right toes more than his left
(3) That otherwise there is no indication by the record that the patient was suffering from hemipligia.
The following photograph of a record made immediately after the former shows the following points.

1. The patient took longer steps than in the preceding case.
2. The toes of the right foot are more turned out than those of the left foot.
3. The foot (right) is not properly lifted off the ground at each step. The outer toes of the foot scrape the ground. The big toe does not scrape the ground.

The reason for this is pointed out in the account of A.P. where the same phenomenon was observed. Before taking this latter record of A.N's gait, I asked him to walk smartly across the paper. In the previous record he walked carefully, and lifted his foot (rt) high in the air to avoid scraping the toes. On getting him to walk smartly and in his ordinary manner the following record was got.
THE CASE OF Mrs B.

Mrs B, age 63, was a housewife. Both her father and mother died of "apoplexy." I saw her first on the 21st of June 1901. She was then suffering from difficulty of speaking.

There was no other paralysis. After three hours this aphasia passed away completely and the patient was able to speak as she did before.

After four days she had a similar attack. It lasted a little longer than the former one but also passed away completely.

On 28th June (a week after the first attack) she became unconscious and had symptoms pointing to haemorrhage into the internal capsule of the left side. She remained unconscious for six hours. When consciousness returned it was found that her right arm and leg were completely paralysed also the lower two thirds of the right side of the face. She had also total motor aphasia.

On 18th July (20 days after the severe haemorrhage) she was able to say "yes" and "no" and move her right leg a little. From this time onwards she made more rapid improvement in the use of her muscles, but her mental state became very depressed. On the 22nd day of the disease she said, "I want to go to Jesus." This was her first utterance. By the 28th day she had recovered the power of calling things by their proper names, e.g. when I showed her my watch and asked her what it was, she replied after a little hesitation "WATCH."
On the 28th day of the disease I noticed contraction coming on in the right arm. Her right arm has remained quite useless since that time. After two months from the beginning of the disease she was able to walk a few steps. She went on improving for another three months and then no more improvement came and she began to go back. She gradually became bedridden. When I last saw her about a year ago she was in the same state. She could not walk sufficiently well to make a record of her footmarks.

She thus went on improving for six months after the haemorrhage and then since that time has gone on growing weaker till now she is almost completely bedridden.

THE CASE OF Mrs. D.

Mrs. D., age 55, a housewife was hurrying for a train when she felt what she thought was a knock on the head. She could see nothing to have caused this. She felt pain in her head and soon fell to the ground. She had the appearance of a drunken person. She was picked up and made to sit on a chair. I saw her about fifteen minutes after the attack began. I found her propped up on the chair semiconscious, talking thickly and rambling as if she were drunk (she had had no stimulant nor did her breath smell of it). She was got home in a cab and laid on a bed and was now quite unconscious. She remained unconscious for seven and a half hours and then...
gradually regained consciousness. It was then found that she had complete paralysis of the lower two thirds of the face (right side) also of the right arm and right leg. She had also total motor aphasia.

She gradually recovered the power in her leg first, and then in her face. Her power of speech also returned sufficiently to let her carry on an intelligent conversation. A very little power returned to the hand and arm. Improvement went on for about six months, then no more improvement came and the patient became despondent and emotional. After a year patient could only walk with great difficulty and at the end of two years she seldom attempted to walk. Now at the end of three years she is almost bedridden. No record was made of this case.

The next case I shall describe is that of Mr. A.M. I have watched this case during the chronic stage only. He is said to have had a stroke in January 1902 and again another in February 1903. He now talks in a monotonous tone of voice and can otherwise talk well left
His right arm is weak, but is useful. He has numb sensations in the left hand. He can use his fingers well and can write. His right leg is weak but he can walk as far as a mile a day. He has thus made a fairly satisfactory recovery from a cerebral haemorrhage which has probably taken place in the right internal capsule. He now has very little characteristic gait.
and in this lies the interest of the case. A casual look at the accompanying record does not seem to suggest anything characteristic of haemorrhage into the internal capsule, but on closer inspection the following characteristic points can be made out.

(1) Very short steps taken
(2) Turning out of left toes more than right toes.
(3) The right foot is advanced farther in front of the left than vice versa.
ACCOUNT OF THE CASE OF B.D.

B. D. aged 61 had a fit on 24th March 1902 at 10.30 A.M. He was going about his duty at his office. He had previously had good health. The fit quickly passed off but he remained insensible. I saw him in about fifteen minutes after the fit and found him lying on the floor. He was then deeply unconscious. No light reflex could be got. Pupils were widely dilated. The arms and legs were quite flaccid. Respiration was deeply stertorous. Surface of the body was warm and perspiring freely. The face was very congested. Pulse was 78. The radial artery atheromatous.

After an hour movements of the left arm and leg were observed but right arm and leg remained quite flaccid. I drew off about half a pint of clear straw-coloured urine and found it contained neither albumen nor sugar.

I remained beside him for nearly 16 hours and saw him gradually grow more deeply unconscious, the breathing and pulse gradually grew feeblar and more irregular and he died 16 hours after the haemorrhage began. He never regained consciousness. No autopsy was obtained but from the great similarity of the case to that of Mrs B. in which I performed an autopsy it seems very probable that there was haemorrhage into the right internal capsule which burst into the lateral ventricles and caused death.
Having now completed the account of these cases I shall proceed to tabulate and discuss them. First as regards **CAUSE**

In the case of A. P. age 37-----cause unknown

In the case of W. S. age 65 -----increased blood pressure due to whisky and hypertrophied heart from aortic regurgitation

In the case of Mrs H. age 56 granular kidney and aortic regurgitation.

In the case of J. K. age 56-----cause unknown

In the case of W. F. age 56 -----granular kidney

In the case of A. N. age 61 -----granular kidney

In the case of Mrs. B. age 63-----atheroma

In the case of Mrs. D.---age 56 ---overstrain

In the case of A. M. age 61 --- cause unknown

In the case of R. D. age 61 -----atheroma and excitement

Thus 90% were between 55 and 65 years of age and 10% under 55 years of age.

30% were females, 70% were males

30 % had diseased kidneys

20 % had heart disease

20 % had atheromatous vessels

In 10 %, exertion was the cause

In 20%, excitement was the cause

In 30% no cause was discovered
**PRGNOSIS.** The most important symptom in forming a prognosis is undoubtedly the length and intensity of the primary insensibility. If it is long and severe, death or very imperfect recovery may be expected. Warning of approaching death during the initial coma in two cases was as follows:

1. Clammy perspiration
2. Irregularity in the breathing
3. Collection of mucus in the larynx
4. Absolute flaccidity of both arms and both legs
5. Marked fall of temperature

The following is a tabulated statement of results in the above ten cases made with a view to forming a prognosis.

<table>
<thead>
<tr>
<th>CASE</th>
<th>TIME UNCONSCIOUS-AFTER 24 Hrs</th>
<th>ONE MONTH</th>
<th>ONE YEAR</th>
<th>TWO YEARS</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. F.</td>
<td>24 hours dazed</td>
<td>walked half mile</td>
<td>suicide</td>
<td>----</td>
</tr>
<tr>
<td>W. S.</td>
<td>6 hours quite conscious</td>
<td>walked 20 yds.</td>
<td>can walk very little</td>
<td></td>
</tr>
<tr>
<td>Mrs. H.</td>
<td>½ an hour died</td>
<td>----</td>
<td>----</td>
<td>----</td>
</tr>
<tr>
<td>J. K.</td>
<td>20 hours dazed</td>
<td>could stand and walk 20 yards</td>
<td>cannot walk so far</td>
<td></td>
</tr>
<tr>
<td>W. F.</td>
<td>16 hours semiconscious</td>
<td>walked 20 yds.</td>
<td>could not walk so far</td>
<td></td>
</tr>
<tr>
<td>A. N.</td>
<td>Did not lose consciousness</td>
<td>quite conscious</td>
<td>walked well</td>
<td>walked well</td>
</tr>
<tr>
<td>Mrs. B.</td>
<td>6 hours dazed</td>
<td>could not walk</td>
<td>walked 20 yds</td>
<td>could not walk</td>
</tr>
<tr>
<td>Mrs. D.</td>
<td>7½ hours dazed</td>
<td>could walk 20 yds</td>
<td>could walk 20 yds to walk</td>
<td></td>
</tr>
<tr>
<td>A. K.</td>
<td>a few hours</td>
<td>?</td>
<td>?</td>
<td>? can walk well</td>
</tr>
<tr>
<td>B. D.</td>
<td>16 hours (died while unconscious)</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
</tbody>
</table>
Haemorrhage into the ventricles is fatal if of great extent as in the case of Mrs. H. Great mental depression may lead to suicide as in the case of A. P. In no case did any improvement take place after one year. In cases where improvement took place it went on till the expiration of about a year and then the patients got worse i.e. they gave up attempting to walk so far as they had done and became more helpless in every way. In those cases in which no power returned to the arm in a month no return of power took place. Every case that recovered was able to walk in a month, except one case and she was able to stand and take a few steps with help in six weeks. In every case power returned to the shoulder. In two cases more power returned to the leg than to the hand and only in one case did more power return to the hand than to the leg.

PATHOLOGY When haemorrhage into the internal capsule is severe as in the case of Mrs. H. it first separates the nerve fibres and later on tears up the brain substance. It thus forms a cavity for itself which extends in every direction. The walls of this cavity are very irregular and pieces of brain substance may be found mixed up with the blood in the cavity. When the blood reaches the lateral ventricles it extends into these and ceases to destroy the brain substance in the neighbourhood of the internal capsule. So great is the damage before this happens that there is little chance of the patient's
recovery. So much damage is done by the haemorrhage that it may be impossible to find the vessel from which the blood escaped. The blood extends on to the surface of the brain through the great transverse fissure. The pressure inside the brain is increased in a case of sudden large intracranial haemorrhage and the convolutions on the surface of the hemispheres are flattened. The blood within the cavity clots.

Other organs which present changes most frequently are the heart and kidneys. In three cases I have seen hypertrophy of the heart, and in three cases granular casts in the urine pointing to granular contracted kidney.

**TREATMENT** - Prophylactic treatment for cerebral haemorrhage consist in avoiding anything which raises the blood pressure, especially overexertion and excitement. The excitement produced by alcohol should be especially avoided. Simple living with easily digested food and the use of purgatives whenever required should be enjoined. I would also recommend a saline aperient regularly once a week.

When the haemorrhage has actually begun I recommend keeping the patient sitting up whenever that is possible. We want to draw the blood away from the brain. In fact the treatment for cerebral haemorrhage is the
Ice to the head and counterirritation elsewhere should also be adopted with a view to reducing blood pressure in the brain.

During the after treatment I have found constipation a most constant symptom which required to be attended to. For this purpose I have found the most satisfaction from the use of the following pill

\[
\begin{align*}
\text{P.} & \quad \text{Podophyllin} \quad \ldots \ldots \frac{1}{2} \text{ Gr} \\
\text{Ext.} & \quad \text{Hyoscyam} \quad \ldots \ldots \frac{1}{4} \text{ Gr} \\
\text{Ext Taraxacum} & \quad \frac{1}{2} \text{ Gr} \\
\text{Ext Colocynth. Co} & \quad \frac{1}{4} \text{ Gr} \\
\text{Ext. Jalap} & \quad \ldots \ldots \frac{1}{2} \text{ Gr} \\
\text{Leptandra} & \quad \ldots \ldots \frac{1}{2} \text{ Gr}
\end{align*}
\]

I have found Strychnine useful in the after treatment. I have found Potas. Brom. decidedly harmful. On one occasion I gave it to quiet excitement. It had the desired effect but the patient (W.F.) lapsed into an apathetic semiconscious condition from which he was soon roused by Strychnine. By keeping him on Strychnine he has had no return of this condition. I also saw Potas. Brom. 

\[
\text{Potas. Brom.}
\]

deharm in the case of W. S. to whom I gave it on two separate occasions with a view to allaying the numbness and tingling which went on in the affected side of his body. On both occasions he said it made him feel ill and he refused to take more.

The End.
exact reverse of that for syncope. The severe cases that I have treated by keeping them sitting up during the beginning of the haemorrhage have done better than those that I have treated by keeping them lying down with their heads raised. In the case of Mrs H. the patient remained conscious for four hours while she was kept sitting up and on laying her down she at once became unconscious and died in half an hour.

In order to reduce pressure of the blood in the cerebral arteries during the haemorrhage, a most rational treatment is venesection. I did this in the case of B. D. but he died without regaining consciousness.

It is difficult to gauge the amount of benefit done by venesection. I would recommend it in cases in which the haemorrhage is due to overexertion and excitement also in other cases in which the face is congested and the veins on the surface of the body are engorged with blood. Croton oil (5 drops) should also be given at the onset with a view to reducing the arterial pressure. Less than 5 drops should not be given or the action of the drug may be so long delayed that the cerebral haemorrhage may have stopped before the croton oil has had its effect. The attendants should be well warned of the action which is to be expected, otherwise the patient may be moved so much in getting him clean after the action of the oil that more harm than good is done. I have seen this occur once.