Dissertation on a case of Primary Ventricular Haemorrhage

In the following paper, I will take up in the first instance the clinical history of the case, and describe what was found at the post-mortem examination; secondly enter into the cause, pathology, mode of anatomy, diagnosis and prognosis of the disease; and thirdly I will discuss the significance of the various symptoms, comparing this case with a number of recorded cases of the same condition.

In doing this, I shall repeatedly have occasion to refer to the excellent papers on this subject by Dr. Edward Sanders published in the American Journal of Medical Science in 1801; also to what Dr. Gower, Dr. Hilton Pope and others have to say on this topic.

W. McLawrence Watson F.R.B. C.M. Edin 1888
5 South View
Elkley-in-Warfedale
Emene E. R.
Age 22 years
Occupation - Upholsterer
Place of Birth - Wesley in Walsall
Date of Illness - June 16, 1891

History:

Heredity:
- Tetter and Tetter to the aline and in good health. The family, however, on the father's side have a neuritic history. For example, one uncle and one aunt are subject to epileptic attacks;
- While one cousin has epilepsy, and another cousin suffered from the same disease.

Habits as to Food and Drink:
- Patient was a most regular and temperate liver. He was teetotal all his life.

General Surroundings at Home and at Work:
- These were all that could be desired; he lived in one of the healthiest parts of Yorkshire.

Previous Illnesses and Accidents:
- Except for one fit when he was eleven and a half months old, this youth has always previously enjoyed excellent health. He never had any
illness of any consequence.

About the middle of March 1871, three months prior to this attack, this youth received a blow on his head from a fall on the football field. He was not conscious insensible and was able to walk the rooms. He then had a warm bath, took his tea, and went to bed. He was sick several times in the night, and could not sleep. On the following morning, he took an opium tablet, had some milk and some water, which seemed to allay his sickness. He, however, stayed in bed till four o'clock. He then got up and walked to the station to meet a relative. Later, he went to the club and returned home about 11 p.m., went to bed and had a good night's rest. Next day being Sunday, he went to church; and on Monday morning returned to his work. He seemed as little the worse of the accident that it was not deemed necessary the longest be seen by a doctor. From this time, however, he began to suffer more or less from headache, and he appeared to his friends to be more easily put out of temper than usual. This state of affairs gradually got worse. He was quite able to attend to his work and indeed played football again on two or three occasions after his accident.

Time and mode of crisis & amount of present attack:

On the evening of June 16, 1871 (about three months after the accident), while walking in a •••, he suddenly about 7 p.m., became faint & sickly & stumbled in going upstairs. Yet, he was still able to walk ••• ••• distance of about three hundred yards. After he reached home, he was sick and vomited. He went to bed.
clock, when he "fainted." He did not recover complete consciousness for about half an hour. He had to be assisted to bed by his daughter. Later on in the evening Dr. Tomlinson saw the patient, when from all accounts he seemed better. Dr. Tomlinson did not arrive at any definite conclusion as to what was the matter. Patient complained of headache, and seemed altogether somewhat unsure. As already stated he had been sick or vomiting and had "fainted." These ideas entered into Dr. Tomlinson's mind. I. Justice's doctrine as to the youth had written some new ideas. 1. First epileptic seizure is a youth with a strong family predisposition to that disease. 2. Encephalitis lethargica is a youth with a strong family predisposition to that disease. 3. Encephalitis. Is the doctor undue a sedative draught and asked for a sample of his urine to be sent on to his home. About half past eleven on the following forenoon (June 17) I was sent in the street by the constable of the patient. The nurse to go at once to see the patient, as he seemed to be worse. I found from the history that the patient had had rather a restless night, had been "delirious," had vomited several times. His last few words I heard that he had had a fit about half past eight in the morning, and that he was convulsed in all his limbs. After the fit passed off he drank some soda water and then went off to sleep till two o'clock, then he woke up, had some milk and soda water — this convinced me vomiting.

The patient quite conscious for a short time, shortly after
this he became delirious and had another
convulsion. When I arrived on the scene,
this convulsion had just passed off. The
patient was delirious. He stretched out
both his arms and moved them about in
a convulsive manner (though there was
no actual contraction), clutching at the
bed clothes or anything he could lay
hands on. At all appearances, he
resembled any one in the excitant stage
of chloroform anaesthesia. He rambles
in his speech. He recognizes and
answers to his name when addressed
in a loud voice. When asked in a loud
tone to put out his tongue, he was anxious
enough to do as he was requested.
He complained of pain in his head—especially
at the back. His face seemed quite
natural and his expression did not
indicate any especial trouble. He seemed
drowsy. There was no drawing of the
mouth to one side. The pupils were
contracted and the conjunctive sensitive.
There was no paralysis in any of the muscles.
The patient was able to turn pound on to
his side in order to let me examine the
back of the chest. This he did after being
asked several times in a loud voice.
His tongue was clean and moist and was
protruded not quite straight. The pulse was somewhat weak, but rate was about average. The heart sounds were normal; there was no murmur. The respirations were not increased or diminished in frequency, they were perfectly regular, and there was no tendency to Cheyne Stokes' breathing. The bases of the lungs were quite resonant on percussion, and auscultation revealed nothing serious. The temperature was 100.5°.

Though the head and tongue of any one who spoke loudly to him, yet the patient did not seem to apprehend anything of what was going on around him.

I thought the patient was feebly drowsy and sleepy as after an ordinary epileptic attack. I ordered him a Bromide of Potassium for his supposed epilepsy; this relieved the patient very soon. Before I left the house, the patient seemed somewhat better, as I thought, quiet and as if dropping off into a natural sleep. I asked his friends to send along a sample of the wine. The specific gravity was somewhat high 1032. There was no sugar present, and no albumen. But it contained an excess of the salts of urates of soda and ammonia, as was
shown by the appearance of an opalescence
or the addition of nitric acid in the cell;
the clod being situated not at the junction
of the two liquids, but on the surface
of the wine. This clod disappears
on heating; or again, it was prevented
from forming by diluting the wine with
an equal quantity of water.
Later—about half past twelve, Dr. Johnston
called to see the patient. On his arrival
he found the patient in a fit, which presented
no appearance of an ordinary epileptic
seizure. The convulsion affected all
the limbs—upper and lower;
extremities. When the fit passed off,
the patient still remained in an
unconscious condition. Dr. Johnston,
knowing the family history, concluded
that the patient had just had an
ordinary epileptic fit, and was sleeping
in the usual way after the attack.
However, he seemed to be unusually
sound asleep. The pupils were contracted,
and the conjunctivae were red and sensitive.
The pulse was very weak, and once or
twice Dr. Johnston noticed a slight
catch in the breathing. He then began
to get alarmed about the condition of the
patient, and injected slowly and cautiously.
in different places. His treatment began to be interfered with, exactly like a person suffering from an acute diabetes. Dr. Johnson, therefore, tried artificial respiration. This seemed of slight temporary benefit, but its effect did not last any time. The patient soon began to turn blue. The pupils dilated, and the heart and respiration gradually ceased. Death took place about one o'clock—about 18 hours from the time when the symptoms of pellagra first came on.

Post-mortem examination:

As the history of the case points to some brain mischief and apparently to pressure on these important structures in the floor of the fourth ventricle, we examined that part first and found there the cause of death. There was no fracture of the skull. No swelling. The veins on the surface of the brain were somewhat congested. We found blood clots in all the ventricles—in the two lateral and in the third and fourth. The right lateral ventricle contained a smaller clot than the left. The clot in the left lateral ventricle practically filled up the whole space. Yet in neither of the ventricles was there sufficient haemorrhage to cause any cerebral compression. The clot in the fourth ventricle seemed
to be of more recent formation than were those in the third ventricle. In all probability, therefore, the bleeding must have originated in one of the lateral ventricles and spread to the third and fourth. The fourth ventricle was relatively more distended with blood than were the other ventricles. This also relates to the fact that haemorrhage began in the lateral ventricles and spread to the third and fourth (vide Grune, Diseases of the Nervous System - vol. II, page 367). We could discover no point where the haemorrhage originated. There was nothing in the walls of the ventricles to indicate its origin.

On the under surface of the left frontal lobe there was a patch of softened brain substance. However, this did not seem in any way to be connected with the haemorrhage, as it was separated from the ventricular cavities by a considerable area of healthy brain substance. There was no clot found in the substance of either cerebral hemisphere, or in the cerebellum, the medulla or pons. In fact, the case proved to be one of those rare cases of Primary Ventricular Haemorrhage.
Remarks — The peculiarity of this case is that the patient should have had any hemorrhage at all. To all appearance he was a typically strong, healthy young man; regular in his habits, had been teetotal all his life; had no attenuations, degeneration. With regard to this age when primary ventricular hemorrhage is liable to occur, D. Sanders of New York in his papers on the subject published in the American Journal of Insane Science, vol. 62, 1887, points out that this lesion is relatively more frequent in early life than is the case with ordinary cerebral hemorrhage. He further proves from the cases which he has collected together, that it is most frequent in infancy and in its age. There are fewer cases between twenty and forty than at any other age. This can therefore occurring in a youth of twenty two is somewhat exceptional.

The hemorrhage in this instance evidently began in one of the lateral ventricles and the blood made its way into the third ventricle and thence on into the other lateral ventricle and downwards into the fourth ventricle. It would be aided in this way by the force of gravity — the patient lying in the recumbent position. This is more likely than to suppose that the hemorrhage occurred simultaneously in both lateral ventricles. However
as we did not find the course from which the bleeding took place, it is impossible to say from which lateral ventricle the haemorrhage originated. It is very rare in such cases to find bleeding from both lateral ventricles at the same time.

But if the ninety-four cases collected by Sanders, he found only one instance of this and foundin has never seen a case.

But in 69 of Sanders cases, the haemorrhage began in one or other of the lateral ventricles. It was, however, exceptional to find blood clots in all the four ventricles. Sanders found this in only fourteen of his cases.

Cause — This seems to be conspicuous of its absence. It certainly is more common in males than in females — yet his age was such as to render such an attack unlikely. His family history was a bad one, so far as the nervous system was concerned — yet this would not be sufficient to account for the trouble. Sanders found this condition more common in winter; this was in summer — this haeomorrhage probably of little importance. In two of Sanders cases there was a history of former central haemorrhage — there was none in this case. He was not a vascular subject.
He was not over-zealous himself at the
time when the symptoms came on.
He had no history of former convulsions—
such that one fit when he was eleven
and a half months old. This however
could hardly have had any connection
with the haemorrhage. In the Ilander
instance there was a history
of several epileptic attacks.
And now comes the question of whether
the accident on the football field three
months previous to the haemorrhage was
in any way connected with the occurrence
of the haemorrhage.

Hewett (in Donders's
System of Surgery, vol. IV, p. 186) states—
that a laceration of the floor of the lateral
ventricles, even when very slight, may give
rise to an extensive extravasation of blood
into the cavity, should it so happen that
the injury corresponds to the situation of
one of the large veins in this region. In
a preparation in the museum of St. George's
Hospital there is a slight laceration of the
cortex tributary, as well as of the floor of
the left lateral ventricle, where a large
vein was torn free, and the ventricle was
full of blood." Again Milles (Lectures on
Diseases of the Nervous System, London 1878,
p. 65) reports a case of ventricular haemorrhage
due to traumasms — the case —

The man "had fallen and cut his knee, the
ventricle was found full of blood, but a most
careful examination failed to discover the
cause of the haemorrhage."

Again, in this connexion, the eighth case reports
by Sanders (in this paper in the July number of
the American Journal of Medical Science) is
of interest. "A boy, five years old, healthy,
fell, eighteen months back, three feet, striking
his head on a hard board, wounding himself
in the side of the neck. He lost much blood.
He soon recovered, but his post temper and vitality
changed into listlessness, drooping and weakness.
His right eyeballs were always swollen, and the eye
itself appeared more emaciated in the head and
cameller than the left. His feet were weakly
and trembling; appetite, repetition, and other
functions undergoing no change. In the winter
he was attacked with cramp. In twelve days
the symptoms of cramps suddenly disappeared,
and soon after the symptoms of inflammation of
the brain declared themselves, and the patient
died the next day, presenting the indications of
water-strike (acrid ad poppug)." In this case
(said by the way from Godet, Hydrocephalus
Acute, tran., by Godet, London 1821, page 264.)

there was found at the autopsy — acute
inflammation of the cranial contents, also,
in the right lateral ventricle an old block clot – or at any rate a mass about the size of a pigeon's egg which seemed to consist of coagulated lymph vessels by blood. This latter being the cause of the drop change of disposition & character; while the former, namely the acute inflammation was the immediate cause of death. So far as could be told in the case the old block clot dates from the time of the accident and was probably caused from the accident.

In the present instance, though the change in the youth's disposition could lead one to suspect the injury received three months previously to be the cause, yet the fact that, at the post-mortem examination, we found no evidence of old standing haemorrhage in the shape of a decubitalis configuration, but only recent clot, makes it seem very unlikely that the accident should have any connection with the occurrence of this haemorrhage.

Pathology: – In this case we did not mention the seat of the haemorrhage.

In all probability there was a lesion somewhere which we overlooked. Barton maintains
that the haemorrhage takes place
generally from a vessel of some considerable
size and cannot occur by a process
of diaplasia. True and Siwekiger occur
in this view.
The causes from which this may arise are
according to Sanders—1. The vessels of the
eochism plenum. 2. The vessels of the choroid.
3. The arteries ramifying in the ventricular wall.
4. The veins ramifying in the ventricular wall.
5. Large sinuses. 6. Lesions in and about
the ventricles. 7. Inflammation or ulceration of the
ventricular walls. Again it is liable to arise
in cases of atrophy or primary softening of the
brain also in disease or weakness of the
vascular walls; also when from any cause
there is insinuation to them necessary return.
Some of these are present in this
instance.

Morris's Anatomy:
The blood in such cases is generally found
to be coagulated—either finely or in part.
In the present instance we found, as already
stated, the blood was clotted. Frequently,
there is some coagulation of the ventricular
walls which in many cases is caused by the
pressure of blood. Of course one gets
also extensive coagulation of the walls in
secondary ventricular haemorrhage, but that
we have nothing to do with it in this paper.

I have already stated where we found the clot and the probable cause of the hemorrhage took place. In some cases the clot goes even further than in the present instance, and passes out from the fourth ventricle on to the surface of the brain.

With respect to the question whether or not these may be absorbed from the ventricles after extravasation, it has been shown that such a thing may occur. There it has been found in a number of cases, which had formerly suffered from central hemorrhage, with perforation into the ventricles, the remains of such extravasation in the form of hemorrhagic patches in the ventricular cavity. Thus Bright and Ransford found what they thought were actual blood clots in a number of cases in the choroid plexus (Savary). Under the pathology I have entered into the question of the seat of the hemorrhage—

**Diagnosis:** In order to arrive at a correct diagnosis in such cases, we must carefully observe all the phenomena that arise.

We cannot go by one symptom— as there is not one which is pathognomonic; but it is, as it were, the text ensemble that we have to look for.
Gowers in his book on Diseases of the Nervous System—page 377, says, "The diagnosis of a primary ventricular haemorrhage is purely possible. There is no distinctive symptom, and the combination of symptoms that attend it may be produced by haemorrhage into the substance of the brain or into the meninges. It may, however, be suspected if some apoplexy comes on in early life, without preceding symptoms to suggest an aneurism."

On the subject of the diagnosis of this condition, Sanders speaks more helpfully. He sums up this—"In the present state of our knowledge of the subject, a positive diagnosis is rarely easy, if ever, impossible. But given a patient with sudden complete coma, partial or complete paralysis, or even without any paralysis at all, constructor and consolator, with rapidly following death, in fact that collection of symptoms which we have come to recognize under the term "apoplexie frontale", the possibilities are that we are dealing with a primary intraventricular haemorrhage." Such was to a certain extent the case with one patient. The coma did not come on suddenly known; the illness was sudden in its onset, in its coma; yet the coma came on gradually and
by degrees deeper, till death occurred.

Then there was no paralysis — at least not until coma was advanced — that is, just before the fatal issue. Contracture too was not present, except in so far as a patient kept clenching at the bed-clothes in a somewhat convulsive manner — but it did not amount to contracture. Hence the diagnosis was not established in life. The conclusion we arrive at was, that there was some intra-cranial mischief, and in all probability pressure on the pons at the floor of the fourth ventricle. But what that pressure was due to, we did not know.

The case was one not likely to be mistaken for ordinary central haemorrhage. (1) The age of the patient (twenty-two years) was against this; central haemorrhage occurring generally about middle life or in elderly people. (2) Convulsions are rare in ordinary central haemorrhage. (3) There was no paralysis till near the very last. (4) The rapidity fatal issue — viz. in 18 hours after the first appearance of its symptoms; in central haemorrhage, life is usually prolonged for several days.

In many respects this case resembled haemorrhage into the cerebellum with secondary rupture into the fourth ventricle. The absence of initial paralysis is according to St. George's pattern.
a lesion. Again frequent vomiting (such as was present in one case) is symptomatic of cerebellar haemorrhage. Lastly, the symptom of pressure at the floor of the fourth ventricle was quite in keeping with haemorrhage into the cerebellum when rupture and consequent extravasation of blood into the fourth ventricle is liable to occur. However, we found at the post-mortem examination no clot in the cerebellum, and the clot in the fourth ventricle was of more recent formation than were those in the lateral ventricles, showing that the bleeding must have commenced in the latter, perhaps partially as already described.

In many respects the case resembles that of intraventricular haemorrhage; in fact, the symptoms seemed in many ways more characteristic of this latter disease. (1) The pain in the head is not frequent in primary ventricular haemorrhage, but is so in ventricular haemorrhage according to Ranville: this symptom was present in this case. (2) Intraventricular haemorrhage is less common in intraventricular haemorrhage than in ventricular haemorrhage. (3) Diaphoretic of mouth or tongue was not present in our case — it is more commonly met with in ventricular haemorrhage. (4) Coma was a marked feature; they are found as a rule in ventricular haemorrhage, not as common, although frequently present in ventricular haemorrhage. (5) Vomiting — rare present — it very common.
is intravascular haemorrhage but not in ventricular haemorrhage. However the paper
conceives that the case was more characteristic of ventricular than of intravascular haemorrhage,
as in the latter case life is usually prolonged for several days.

II In some of these cases there was the case simulated haemorrhage into the fossa.

1. The initial convulsions which were universal, affecting all the limbs in, according to Jones,
especially frequent in haemorrhage into the fossa. (Vide pag. 359 of this book).

Hartingh states that convulsions are more frequent in haemorrhage into the
fossa than in any other form of trauma
haemorrhage; while on the other hand
Dr. Dagny says that has not proved it so
in his experience - vide M. E. pag. 505.
Dagny took no medicine.

2. The contraction of the pupils is a symptom which
is frequently seen in haemorrhage into the fossa.

3. Frequent vomiting is usual in haemorrhage into the fossa.
Against the idea of haemorrhage into the fossa were

1. The absence of enunciation.
2. The absence of conjugate deviation.
3. Respiration not affected till the very last - while in
haemorrhage into the fossa it is frequently affected early,
after a very slow one to one and a half minutes.
4. Respiration was only 100; while in haemorrhage into fossa
it is frequently hyperpnoeic.
I from absence of the brain rupturing into the ventricle.
Although there was a history of an a fall on the head this month previous to the fatal attack, also of headaches more or less during the interim, yet there was the absence of the ordinary symptoms of confinement – fever, and the temperature was not such as to indicate abscess formation. officiers – common symptoms with abscesses – sufficient time the case ran a too rapid course; in abscess according to Sanders (pap. 127) death is almost always delayed for several days.

Prognosis: – I have already – in discussing the mal's anatomy – alludes to the fact that Sharpe, Bright and Rawshill have in some cases found evidence of the remains of old haemorrhage in the ventricular cavities. This proves that there are occasional exceptions to the rule which holds in the majority of cases, that extravasation of blood into the ventricle is rapidly fatal. The speed with which death occurs depends, according to Sanders on
0) Rapidity of extravasation, (2) amount of bleeding,
(3) Seat of the haemorrhage. The more openly the bleeding the sooner death occurs. The greater the amount of haemorrhage the quicker the death.

With respect to the seat; haemorrhage into the fourth ventricle is the most rapidly fatal; then haemorrhage into the third ventricle. Life, in
hemorrhage into the lateral ventricles, is usually prolonged for several hours, and may be for some days. Recovery has been known to occur when hemorrhage takes place into the fourth ventricle. There is one case on record where temporary recovery took place after hemorrhage into the third ventricle, but the patient died from tuberculous meningitis a year afterwards, the result of the hemorrhage (Sanders paper). The cases where recovery has been proved than those when the bleeding was into one of the lateral ventricles.

And now these are a few points in the symptoms of the case worthy of notice.

1. The usual mode of onset in this disease is by partial or complete loss of consciousness without paralysis, usually there is complete loss of consciousness from the beginning. In one patient when I saw the patient about an hour and a half before death, he was conscious enough, when addressed in a low voice, to put his tongue out when asked, and to turn around on his side to let me examine his back. Liddell in his treatise on Aphoplegic Hemorrhage, 1834, gives it as this opinion that the more the hemorrhage the quicker is the loss of consciousness. This opinion would seem to be verified by this case in point, as although
...found in all the four ventricles, yet none was so distended as to cause any cerebral compression. However, Sanders says that Ubbelohde's theory is not borne out by his cases. He is of opinion that the evidences of extravasation have more to do with the symptom than the amount of the haemorrhage itself. His theory is that "the nerve cells receive a blow which temporarily or permanently deprives them of their power of functioning." Page 344, American Journal of Medical Sciences, Oct. 1881.

Of course in this case as in all cases of the kind unconsciousness came on in the common, then coma became advanced—just before death.


Contracture was not observed in our patient. It sometimes is seen in primary ventricular haemorrhage, but often it is absent. It is more common in secondary haemorrhage. Its absence may be accounted for from the fact that (so far as our cases) there was no laceration of the ventricular walls. Sanders states his symptom present in more than one third of his cases. He considers that the production of this symptom has a good deal to do with the condition of the walls of the ventricles. He says—"In this frequency of the occurrence of laceration with the occurrence of contracture, I believe there is more than a mere accident, although it cannot positively be shown..."
Gowers states that sometimes ventricular haemorrhage is caused by convulsions — this however seems improbable in the present case for these reasons — (1) The patient, though belonging to an epileptic family, had never before (except once when an infant of 1½ months), had an epileptic seizure.
(2) There was no sign of any subarachnitis. (3) The first convulsion in this attack did not come on till about 13½ hours after the first onset of the symptoms. I think therefore that can be no doubt that in the present instance at any rate the convulsions were secondary to the haemorrhage or were merely symptoms of the same.
at one time in twenty-three cases, and in
seventeen of these it came on early.

**Condition of surface** — There was no pallor or
flushing of skin of face to be noticed in
one patient.

**Temperature** — Sanders thinks there is probably
an initial fall which is very transient,
later there is a gradual rise till death
takes place. In one case, the temperature,
when I took it about one hour and a half
before death, was 100.5°F.
that lacanition always consists with this symptom, since in a small percentage it cannot be demonstrated, yet when we remember the likely host of this motoric condition being overlooked, it would seem that its presence is almost a necessity, the more so when we consider the almost uniform occurrence of this symptom with the secondary form of ventricular extravasation consecutive to lacanition of brain tissue." Prof. Todd, D.D., M.D., D.Sc., holds the same view in respect of the occurrence of this symptom in secondary ventricular extravasation.

3) Convulsion — occur in about 31 of all cases according to Sanders, and are due to the pressure of the ejected blood on the corpora striata. One patient had three well marked epileptiform convulsions — affecting all the limbs. See opposite page.

3) Paralysis — almost in one patient till just before death when coma became deep. Sanders found this symptom present in about one third of his cases. It is generally hemiplegia.

3. Disturbances of sensation:

Paresthesia or paresthesia was one of the first symptoms in one patient. In Sanders' cases, he found this mentioned eight times. He thinks these cases which commence with these symptoms are usually gradual in their onset and that life is prolonged for some time, the symptoms not being very violent.

Headache — complained of in one case, is of frequent
occurrence. It is due to various causes, including infection or haemorrhage.

4. Disturbances of organs of special sense—

Eyes—pupils were equal but contracted in the early part of the attack, later they became dilated just before death. The contraction was probably due to pressure of the effused blood in the fourth ventricle on the third nerve causing stimulation; later just before death the further pressure of more effused blood would cause paralysis of these nerves or the sympathetic still acting—dilatation of the pupils would occur. The equality of the pupils showed that the haemorrhage was the same on both sides; at any rate in these cases where inequality of the pupils existed—the haemorrhage was not equal on the two sides.

5. Disturbances in the Alimentary System:

Vomiting occurred frequently in one patient—

It is noted in only twelve of the hundred and fifty-four cases.

6. Disturbances in Circulatory System:

Pulse is usually slow, full and hard in early stage, later it becomes rapid and feeble. In one patient pulse, when felt it, seemed about average full and of ordinary strength.

7. Disturbances in Respiratory System:

There was no change here in one patient until the very last, but in twenty-eight cases recorded by Sanders when reference is made to this system, oedema was present.