Thrombosis of the Cerebral Sinuses.

My attention to this subject was drawn by a case which occurred in my practice in 1887. Not feeling satisfied on my own account at the time as to the actual cause of the disease in this case, I resolved to inquire as to what occurred therein was of similar cases. Hence the present paper. I will commence by relating the case above referred to, abstracting as far as possible from my notes, then pass on to a second case which occurred in my practice recently, and thirdly give a résumé of reports of 56 cases which I have collected from various sources with a view to trying to get a little light thrown upon a still rather obscure disease.

Case — A young woman, L. B., aged 25 years, a children's nurse in a small farm house with healthy surroundings, came under my care for her last and fatal illness on Jan. 26, 1887.

Family History. Both parents alive, father has been a heavy beer drinker, and suffers from nervous debility in consequence. (Since these notes were taken, he has been sent to the County Asylum at Thrope suffering from Softening of the Brain and Senile Dementia.) Father lost 2 brothers & 3 sisters from Phthisis. Mother and her side of the family strong and healthy. No history of any nerve disease or mental disease in the
Cases of Coma without the usual signs of
- difficulty of speech,
- ear disease,
- symptoms not uniformly headache, loss of consciousness.

Treatment — most vigorous.

Dr. Combe
North Walsham

Most sufficient data

3, 34, 35, 57, 58, 40, 41, 43, 43, 45.

Cases of Coma with and without Coma.

4, 39.
2.3.2 small holes in cornea due to cataract in another eye on the other side.

Recently observed blood in patch in two places in the inner side of the forearm.

Coma present.

Case 4 - child. Arachnoid abscess - brain - subdural.

4. Empyema - fibrinous clot in V. Inarabnia on right side, below V. Cava.


18. Empyema - recently into right anterior central lobe.

4. Coma without T.J. Sup. L.S.

5. Sup. L.S. without coma.

8. Not sufficiently described.

No coma.

Clot in mid cerebral vein after labour. Cause doubtful.

Recovery - cause doubtful. Specific.

Recovery - not explained (child).

Sputum with (Pneumonia ?).

Doubtful - probably acute Pneumonia lung.

Cause uncertain - data insufficient.

Not explained.

No particulars.

The Sup. L.S. with no coma.

Cases: 5/18, 19/20, 21.

14, 15, 16 not sufficient data.
### Thrombosis of the Central Sinuses

**J. Orloff Combe**

Circulate as follows:

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<tr>
<th>Professor Fraser</th>
<th>Received: June 24</th>
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<tr>
<td>Chirn</td>
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<td>Simpson</td>
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in the family— with the exception of that of the father. It was syphilis, or any other constitutional taint.

Personal History. Had twins (illegitimate) in July 1885, that is about 18 months before present illness. In October 1886 was treated by me for Anaemia, with headache, giddiness, shortness of breath, and oedema of legs. Menstruation was irregular. She soon recovered with the exception of palor and vague head sensations. Otherwise she felt well afterwards. No history of any hysterical or other fits, nor of any abnormal sensations other than those mentioned.

About 3 weeks before attack she had a severe fright. About midnight after that she fell down twice while hanging out some clothes; said it was giddiness, when she held her head back. Jan 7th. She was thought to have a cold, and was prescribed for by Mr. C. Morton, a Surgeon residing in North Walsham. But nothing unusual was noticed about her. Jan 10th. Felt worse, complained of pain like knives going through her head. This was in the evening. At mid-day she went to bed. For 2 nights previously had not slept on account of head—said to have pain. She went to sleep soon after going to bed on the 7th, and in 2 or 3 hours was noticed to be heavily amnog, a thing unusual for her to do. Mr. Morton was sent for; he tried to rouse her by slapping her face with a cold wet towel.
towel, and hands; by pinching and by
will shaking her. She partly succeeded,
but after a while she again went off to sleep.
The face was "flushed scarlet" and "bathed in
sweat", but no sweating or flushing were
noticed on other parts. The sleep continued;
face gradually became pale in about
24 hours, and sweating decreased to normal
by the time I first saw her.
6:th 6 p.m. I found her lying in bed, apparently
asleep. Dorsal cleabentes. The second well-
mourned: no marks of violence anywhere,
and no history of any previous injury.
Face and general surface Aucectic, no congestion
anywhere. Temperature normal; pulse 62,
regular, but weak. Respiration 17 and
normal in character as in sleep, no stridor.
Lung stone cold (thought to have been from above).
Hands and all the rest of body warm.
No orifice from the bowels for 2 days, poisoning.
Could not be aroused by any ordinary means.
I drew off about 1/2 pint of urine, which
on examination was shown to be normal in
every way. Conjunctivae and Cornea
sensitive to touch, but not highly so; that is
sensitiveness lessened. Pupils moderately
dilated, and do not react at all to light. Aspersion
put into the mouth causes aprimae,
as if patient were sensible of it. Muscles
slightly rigid all over, with slight clonic spasm,
they react at first but slightly on applying.
applying a weak interrupted current. This action increased very markedly in about 15 minutes. At constant applying, the current. She then appeared to wake partly, opened her eyes, and raised herself in bed; and, when lifted out of bed and propped up upon her feet with her back against a wall, she could retain herself so for about 2 minutes. In 10 minutes from the time of stopping the application, she collapsed into her former condition. Hot and cold applications had no apparent effect anywhere. Pulchra, Sapping and Pricking also had no apparent effect. Patient's Plantar reflexes about 2/8th size. By my direction patient was taken in a cab to her parent's house in North Walsham, a distance of 2½ miles. She bore the journey very well.

9.30 p.m. Condition the same. 7 of salvarsan, Moore. Ophthalmoscopic changes put into the right eye caused only slight dilatation of pupil in 10 minutes. Test still very cold. Temperature elsewhere normal. Nitrite glacial (1 capsule, 4 grains) inhaled caused flushing of face, deeper respiration, opening of eyes, dilatation of both pupils, yawning, and apparent attempts to answer questions. Patient then seemed to know what was said, and moved body limbs as if waking from sleep. Four of the 1/2 capsules of the nitrite were used consecutively, each with increasing action.
Unluckily I had no man by me, and could get none more until next he arose. After using the Natrium Acute the first became a packed warmer, pulse increased in volume and from 60 to 76; also respirations became more frequent and deeper. 15 minutes after the last capsule had been used the patient had collapsed into her previous condition, though perhaps hardly quite so completely unconscious. Nearly a pint of healthy urine was drawn off. I tried to feed her with some egg and milk, but do not think any was swallowed. There was no sickness at any time during the illness, nor discharge of blood or anything from any part except urine. Order is kept with quiet; feet to be wrapped in flannel coming out of hot mustard water, and nourishment, in the form of strong beef tea, egg or coffee, or egg and milk to be got down, if there were any opportunity.

Condition much the same, but can swallow a little jelly. When a spoon put between teeth there is a grimmace, which lasts as long as the spoon is left there. No effect on pressure over ovari. Pupils the same. Retina examined by the Ophthalmoscope, appears normal, chews no congestion. Not in the least startled by sudden noise. 8½ hr was easily disturbed. Remained the same. As before throughout the night. Swallows once easily and quickly. Took a fair quantity of liquid nourishment. Iris easily pulled to both plantar reflexes observed. Ordered Vallis Nervs. 31/1 Spirit 1/4 Sulph 31/1 40 C. 31/1 made stronger every hour.
2.30 a.m. Heavy, face pale, Inflation of feet, pain in nose, mouth, running from eyes, nose and mouth, but no sneezing. Inhalation current applied for 10 minutes, at first not much acting, then improved up to 10 minutes; after that, the effect decreased.

Pulse 108, temp. 99.8, Respiration 22.

From this time up to the 12th, when she died, the whole condition became worse marked. This was in spite of artificial feeding through an oesophageal tube with every conceivable kind of strong nourishment including some brandy, also subcutaneous injections of ether frequently repeated. On the 10th the temperature became normal, and after that sub normal, gradually going down to 97. Pulse became gradually weaker and decreased to 50 by the evening of the 11th, after which it was imperceptible at the wrist. On the 12th, 2½ hours before death, the cardiac contractions were only 41 per minute, as heard by stethoscope. Respiration fell to 6 per minute.
There were no convulsions at any time, as far as I could ascertain. Each time that
manœuvrement was given (up to 9 p.m. on the 14th) there was a slight response in the pulse.

Post Mortem Examination, 20 hours after death

[helped by Mr. Woodman, assistant to Mr. John
Shepherd, Surgeon of North Walesman], Rigorsmortis not strong.
Nothing unusual externally except marked
paler. On removing calvarium, the Dura
Mater was very slightly adherent along the line
of the Sagittal Sinuses. The posterior and sinus
contained a small quantity of dark fluid blood,
and extending along its whole length from
the Conva Salli to the Torcola Transversa, and
down 1 inch along each of the Lateral Sinuses,
falling quite firm, pale, and laminated,
and towards the anterior part rather soft
in the center. The Clot did not entirely fill
the Sinuses, but was adherent in many
places, caught apparently by the fibrous bands
crossing the wall of the Sinuses. It was paler
in the Sup. Long. Sinus than in the Lateral
Sinuses, and tougher in the Lateral Sinuses,
it was dark and more recent, and extended
about 1 inch further along the left side
than the right. The Occipital Sinuses were
so small it was difficult to examine them
properly, but no clot could be found there.
All the Vessels over the surface of both the
hemispheres, running into the Superior Lateral
Sinuses, were very distended with firm pale clots.
A few small extravasations in the substance of both hemispheres. A little clear fluid in the ventricles. No signs of the Fourth Vents, rather fuller than usual with clear fluid blood, but no clotting or mora in the Viri Galeni. Nothing else unusual.

Lungs healthy, Heart rather pale, but otherwise healthy. Abdominal organs healthy.

I should have mentioned that the young woman was of a rather nervous disposition, and for some little time before her illness had worried and fretted on account of being left alone in the house for hours.

Remarks. In this case we have an Anemia, then added to it a nervous depression, and lastly a sudden fright; all of them tending to impede the heart's action, and thus leading to arrestation of the venous circulation in certain parts, the effects of which however were only visible in the brain. There was no other discoverable explanation of the Thrombosis in this case; and in the light of some few cases, which will be related further on, I think we may safely consider it to have been due to the above mentioned conditions, the Anemia being the most important. That the Clot found was admixed with is evident from the fact of their being laminated, and adherent in part.

Further, that they were the cause of death, we are constrained to assume in the absence of any other discovered cause or apparent cause.
Strange enough, while I was transcribing a few notes of the above case, I had before the Norwich Medical Chirurgical Society, another case occurred in my practice, which was diagnosed during life. The following is a brief outline of the second case:

Matthew 13—aged 49. Single. A lady in comfortable circumstances. No family history of Epilepsy admitted. Was quite well and strong up to 17 of age. Until then escaped all signs of one. Had no illness other than young. At 17 years of age Epilepsy developed and gradually increased the fits increasing both in frequency and violence. Has been a confirmed epileptic, unable to do anything for 20 years. Never any history of injury to head nor elsewhere, nor shock. In February 1886 (3 years ago) after several bad fits deceased became very weak and almost completely unconscious, lying in a moribund state for 15 days. She was so insensible that I expected her death hourly during the whole time. At the end of that time she gradually recovered, and in another month was able to move about again.

There were no signs of any lung, kidney, heart or any other disease to account for it. Since then she had never been quite so strong.

1889, Jan. 3rd: Manner anomalous.

A week later several fits in one day, then a few solitaries occasionally, and several
half developed ones at intervals. Got gradually weaker, and more absent, and afterwards impossible to make her understand anything said to her. No violence. Sleep rather above normal—average 10½ hours out of 24. Developed Bronchitis rash on face.

But become somewhat drowsy, will sleep all through night—unusual with her—and a great deal during daytime. Rash on face very marked. Takes hardly any notice of anything. Pulse 100 and fairly strong for her. Bowels moved twice with help of medicine. Urine retained. Passed 2½ in 24 hours. No fits.

It should have said that she's anaemic, with dark complexion.

21st
Slept more, more difficult to move. Face pales.
 Pulse weaker. 1½ per minute. Respiration 18.
 Temp. 98.0. Place on face better.

23rd
Drowsiness increased, nearly always asleep.
 No notice taken of anything. Surface very cold.
 Place on face drying away, face much paler.
 Fingers & nails blue. 2 fits (at 5 a.m. & 10 a.m.).
 Pulse almost imperceptible at wrist. 10 per minute.
 & very irregular. Heart sounds very feeble.
 Resp. 11 & deep. No physical signs in chest.
 Urine healthy. 1½ pint in 24 hours.

28th
Quite Comatose. Temp. 97.4 Pulse 80. Resp. 15.
Death like expression on face. Pupils normal size,
quite insensitive to everything. Cornea hardly at all sensible to touch. Urine passed involuntarily.
No convulsions anywhere.
March 21st. Remained the same up to 9 A.M. this morning. When arising forehead, temples and sides of face were noticed to be abnormally and dark; also a white patch appeared on each side in front and rather below the ear. That increased slowly. No hemorrhage. No urine passed since 28th. Died quietly at 1 P.M.

4th. Post Mortem Examination 50 hours after death. Body very wasted all over. No congestion on surface anywhere, skin very pale. I was only allowed to examine the brain. Cranial bones thin. Dura mater normal. Venous over both hemisphere very congested, many containing clots which in several places were pale and firm. The superior longitudinal Sinus contained a clot extending from Conricta Galli to Vascular Sinus, pale and firm throughout, but paler in its anterior third. This clot was continuous with some occupying the smaller lateral Sinus. Other Sinuses contained recently formed clots that are ill-formed and quite dark. No signs of any inflammation. Inner surfaces of Sinuses quite smooth. The clot in the Supra longitudinal Sinus was adherent to the walls in many places. Brain not atrophied at all. But substance appearing with a few small brown extravasations (dark purple) about the size of a half grain morphine pill, some rather larger. These were situated chiefly toward in and near the gray matter. Nothing else remarkable about.
the brain except extreme palor of Optic Thalami and Corpora striata. No tumour
summae, atrophy, scar, nor abnormal fluid present. No clots found elsewhere.
Beyond those already mentioned. Cerebellum appeared rather small. The head was
of good shape and size.

Remarks. From the nature of the clot in
superior longitudinal series— I should
have mentioned that it was laminated,
being paler in the centre but not softer—
it was evident that it had formed some
time before death, though probably the outer
portion of the clot was not formed until
within 24 hours of death. The conjunctiva
vitreous faciale anterior not having been entered
until 4 hours before death points to that conclusion
as the whole clot all but filled the whole
vessels of the retina. In the case of such a
confirmed epileptic, one naturally looked to
find some old disease within the cranium.
But nothing was found beyond the thalamus
and its effect, except extreme anemia of the
brain. This was very marked throughout.
I cannot help thinking that this Central
Anemia was not recent only, and merely
an effect of an enfeebled circulation. The
brain was so very white, it made one think
of gelatine from a calf that had been systematically
bled during life in order to whiten the flesh.
It looked as if it could not have known the
the presence of blood at all, an evidence of Epiptopy with Acuminum brain, though that is outside the actual subject of this paper. In this case it is, of course, more difficult to assign the causes of the various symptoms, many of them being assignable to the previous disease. The question also arises was there any thrombosis (which may have cleared up by natural processes of digestion &c.) in the similar illness nearly 3 years before? I have thought for 2 reasons, first the condition may have been due partly to an increased Acuminum of Brain, & secondly with such a comparatively poor circulation if a clot once formed it would not be likely to have its elements cleared off in the blood current, but would, in all probability extend and prove fatal. The two illnesses were extremely similar; the only difference being in the addition in the last illness of insensibility of Conjunctiva and Cornea, 2 days before death, and Venous congestion of upper part of face for four hours before death. The former symptom may have been due to the Super-added Thrombosis or to a perhaps greater Acuminum of Brain. The Venous congestion could only be caused by the thrombosis. So that from a comparison of the two illnesses, though as very suggestive at first sight, we learn nothing new.
I will now proceed to the consideration of the 58 cases, besides any over, the report of which I have collected. None of these cases, as far as I can ascertain, have been included either in the monograph by Dusch in the eleventh volume of the New York State Society for works prior to that by Liebold in the American Journal of Medical Science Volume 67 pp. 39 to 65.

In analysing the cases, I shall include my two cases, making altogether 58.

And I shall adopt the classification by Dusch in the paper above referred to.

Out of the 58 cases, I must first subtract 10 that are either doubtful as to their cause, or insufficiently reported.

Of the remaining 48:

- 19 are from inflammation, including 6 from otitis
- 4 from disease of mastoid cells.

10 Anæmia
5 Whooping Cough
3 Disease of walls of Sinuses
3 Injury - beside one of inflammation following injury, included in first group.
8 Varies - effusing blood, claimula, sepsis

48

Many of the cases, although included under only one head each, might be included under 2 or even 3 different heads; different causes being present together. For the sake of the
Analysis. What appeared to have been the chief cause has been selected in each case. Of the various causes first excluded—none it is impossible to refer distinctly to any one group, except in very general terms. They will be taken at the conclusion of the other cases.

For the better understanding of the position of the various cases, I here reproduce the classification by Dr. Sch. above referred to.

A. Thrombosis by Prolongation (Secondary) for Inflammation

II. Injury.

III. Effusion of Blood.

B. Primary Thrombosis—i.e. occurring locally, from Injury or work on Circulation:

I. General Causes.

1. Deficient Cardiac Energy—due to:
   (a) Injuries, (b) Old Age, (c) Precedent acute or Chronic Diseases.

2. Diminished quantity of blood—due to
   (a) Loss of Blood, or (b) Profuse Secretions.

3. Impediments to Expansion of Lungs
   (a) In Lungs—Pneumonia, Tuberculosis, Asthma, &c.
   (b) In Pleura—Effusions, Adhesions, &c.
   (c) In Muscles of Respiration—Deficient Action.

II. Local Causes.

1. Pressure upon Sinuses

2. " " large Vessels near

3. Intruding foreign bodies into Sinuses, and lastly by Disease of Wall of Sinuses from altered Molecular attraction between Woll's tissues of the blood.
I. Secondary Thrombosis from Inflammation of troops in the neighborhood.

Firstly— from Otitis:

Case 1. M. A. G., female, Oct 22. Had always been deaf in left ear, lately gathering them. Deaf recently in right ear also. Has been ill about 10 days. Sept 27th.
Temp. 103.4. Frontal headache, pain in back; depression.
Bowel, torpor; on left, abdomen normal. Chest clear.
Great oscillations of temp. Mornings about 90.6.
6th Brain fever. 10th Regain continued, pulse 96.
Cough and blood stained spuza. Spleen large tender.
11th Temp. 106. in morning, 103.6 in evening.
12th Temp. 104.6. Death.

Post Mortem. Left temporal bone carious, chalk to lateral sinus & porous. middle ear. In left lateral sinue, soft, furred, amyloid, pus formed; clot continued into jugular vein, passed down to its termination.
In abscess overlying in brain. Several small embl.
Abscess in lungs. Liver swollen. Spleen 1103 gr. softened.

A Blue, Ope, Ditchworth, Janet 1889 Vol I. p. 18.
Case 2. M.P., a domestic servant, aged 20. After 7 years chronic discharge from right ear had an acute attack of otitis & mastoiditis. Temperature ranged from 96° to 101.6°. Pyrexia set in 27 days before death. Nystagmus commenced a week before, and continued up to death; one or more clearly. There was pyrexia trouble in both lungs and pleurisy. 2-5 days before death, the maternal process was febrile, resolving pers, but giving no improvement. 4 days later chills & fever set in. 4 days before death, appearance and increase of diarrhoea; tenesmus, frequency, increased vomiting and pallor, prostration of death.

Post-Mortem. Necrosis of right pars petrosum close to & involving the lateral sinus. Dura mater clotted and injected over the bone. Lateral sinus (right) filled by an acute-mortem clot, which did not pass into the jugular vein. The clot was not further described. There was double Empyema. Heart very soft, otherwise natural. Liver & kidneys very soft. No other abcesses. Brain natural.

Remarks. Sir Dyce Duckworth in this case looks upon the pyrexia as secondary to a thrombus breaking down in the lateral sinus. In his report of the case we are not told the condition of the clot in the lateral sinus after death; it is merely described as "acute-mortem". If it were more prevalent it could not have been said to have discharged "pyramidal tracts into the system." Though posterior, it may have broken away, and been carried off. Duckworth, 1891, p. 2.
into the system. It is quite as probable that pus from the mastoid cells found its way into the Lateral Sinus by the Vein Emisvarum mastoidenum and thus gave rise to Pyaemia; the Thrombosis in the Sinus occurring later.

In electricity central symptoms is recorded until 4 days before death, when there was drowsiness. This precedent delirium with delirious speech have been caused by the general pyaemia, and the temperature.

Case 3. Catarrhal inflammation of the dermal layer of the external auditory with Caries of the posterior wall; disease extending to the Lateral Sinus and Cerebellum." In a child 3½ years old.

The only symptoms mentioned, were pain, Mattnessness, delirium and the hands being kept applied to the head.

Path Motions. Amongst other conditions found were a considerable amount of pus, matter containing the Lateral Sinuses was discolored by a purl in and dark Coagulum, ½ inch in length.

Also Lateral Sinuses was full of dark Coagulum and purulent matter, and pus in the jugular veins.

Notes. In this case no mention is made of pyaemia. It is possible also here, as in case 2, that pus found its way into the Lateral Sinus through the Emisvarum vein. The clot in the Lateral Sinus may possibly have been Path Motions.

Joynt's Diseases of the Ear (1868) p. 73.
Case 1. - Catarrh of the mucous membrane of the tympanum, since years, an attack of scarlatina, abscess bone and caries, abscess in substance of the brain; adjacent cerebral matter healthy. Besides excreting pain the only cerebral symptom noticed was a condition, partial sleep, between the paroxysms of pain. At the time of death the patient became insensible & then convulsed. This case is interesting in this paper chiefly on account of one pathological condition found after death. That was posterior petro-carotid adhering to the internal surface of the lateral sinus of the affected side, thus showing the one way of commencing thrombosis from inflammation of neighboring tissues.

Case 2. In a lady of 15 years, Otitis Interna, with Thrombosis of Superior Longitudinal, left Lateral Sinuses and Internal, regular vein also gangrene, Lynx. The patient was restless, delirious, with frontal headache and divergent strabismus. The pupils were equal. There was also typhoid, like chronic headache, Lynx, Lynx. In this case the Superior Longitudinal Sinuses contained a clot adherent to its left wall, same side as the Otitis of a grey-brown, coarse granular and laminated, extending from the vertex to the Torcular Herophili, with prolongation extending a clot clavate into the small lateral veins. In the left Lateral Sinus, from the Torcular Herophili to the mastoid cell, was...
Another Clot, adherent but less gelatinous and darker, then for an inch the Sinuses was filled with puriform matter. The Internal Jugular Vein also was thrombosed for some distance. The Brain was otherwise healthy. Here there was pus in the Mastoid Cells.

Case 6. A young Woman, aged 22, who suffered from Eversion of Lefthand, followed by Abscess in both lungs and double Emphysema. She had been sickness, but that stopped a fortnight before death. The face was also flushed. The mind was quite clear all the time.

Post Mortem. There was, in addition to left ear, accompanying fibrinous matter between petrous bone and Dura Mater, no distinguishable from accompanying blood-clot in inferior Petrosal Sinuses and spreading into the Jugular Vein. When it was continuous with ordinary clot. The Mastoid Cells here also were chiefly the part diseased. There was a friable clot in the heart and concomitant turbid fluid in the pericardium besides affection (pyemia) of Liver, Spleen, Liver and Spleen.

Dr. Church in connection with this case relates a case by Abercrombie [in Pathological Researches on Diseases & Disorders of Brain & Spinal Cord 3rd Edition] very similar, where the lateral Sinuses was partly filled by clot & death ensued from Emphysema.

Case 7.—In a child aged 11 months, 'Sphærophalægia,' disease in horizontal portion of the mastoid cells; abscess in the mastoid process, formed by Caries; disease of the Dura mater; a small abscess in the cerebral. The child was ill for 1 week, had chronic discharge from right ear since birth; also had diarrhœa. There was paralysis of the left side of the face 4 months before death.

Post Mortem. Surface of Brain congested, with patches of dark blood over hemispheres, especially over right. The Central Nervous System disturbed by Congula. The large Central Arteries were thickened by firm film. The Dura mater over the right petrous bone was separated from it by pus, and very much thickened. The larger part of the mastoid process was entirely destroyed; petrous bone detached & both surfaces carious. The remnants of the mastoid cells contain a few Sphærophalægia matter.

Case 8.—'Decay of Mastoid Cells: deposit in the lateral Sinus; secondary deposit in the pleura.' In a young lady aged 15 years. Nausea, headache with pain in right ear 14 days before death. Headache extended over the right side of the head. Quick pulse; face pale; much diarrhœa; pain in chest; increasing weakness of death.

Post Mortem. Brain healthy except near right ear, where it was a dark leaden colour superficially. Dura Mater thickened from pyægeous & temporal bone. Contains pituitary lateral Sinus, pretty thickened throughout, and with an internal deposit of coagulated blood mixed with pus.
Case 9. "Purulenta mastoid cells; Caries of lateral Sinus; primary lateral Sinus; Secondary depression." In a young woman aged 20. Deaf in left ear. Headache, sleepless and occasionally delirious. Abscess above left Solar bone. Fever, delirium, after discharge from ears, finally Encephaloe, violent delirium, coma and death.

Post Mortem. - A large abscess in neck, communicating with and extending through whole left Carotid sheath. Internal Jugular Vein full of matter. Fibrous clot in Vena Cavae extending into descending Vena Cava. Lateral Sinus was dark brown, vitreous well formed by Dura mater entwined, and Sinus was full of coagulated blood mixed with prevalent matter. Dura mater forming its anterior wall thickened, left portion ulcerated away preceding the bone which was clearly rough & covered by masses of lymph and pus.

Case 10. "Purulent serous pus with mastoid cells; Communication with the lateral Sinus begins; secondary deposit in Sinus." In a girl aged 15. Chronic Ear discharge. Ill six days. Partially unconscious, strongly prostrated, pulse 140, small, thread-like, pupils dilated, especially on left (side chiefly affected). Pain in head & left ear. Head held to right side, growth also obvious to right. Stimulant relieved the condition somewhat. Slept badly, became delirious & died.

[Text continues on next page]
Post-Mortem. Cerebellum, against posterior part of petrosal bone, discoloured, softened beneath. Duramater against the inner side of skull. Lateral Sinus continued at its upper part a firm clot of clotted blood, its lower part fully filled with blood. Opening from mastoid cells into external auditory.

Remarks by Tavol: The only means by which the disease from the mastoid cells could be propagated to the cavity of the Lateral Sinuses, must have been the Venous.

These ten cases of thrombosis following disease of or in the neighbourhood of the ear bears out Dr. Chey's statement. The only point would draw attention to are:

1. Absence of any uniformity in the symptoms.
2. The comparative frequency of disease of the mastoid cells, with only 10 as the distinctive disease, and in 2 as accompanying other disease of the interior.
3. In one case the Superior Longitudinal Sinuses of the Root was affected (Case 5). Chey says this never rarely occurs (page 82 in the work quoted).

In one case (Case 7) only the Central Veins were reported as affected, and in another (4) a thrombus had just commenced to form.

The next case is interesting as showing the connection between necrosis of the lower jaw and thrombosis of the Central Sinuses, the affection been having to travel much further before reaching the Sinuses. Following is a brief summarum of the case as reported in the

Lancet for 1886 Vol I p. 593:
Case II. Notes read by Mr. Murray before
The Medical Society of London.
Post-mortem: Right cavernous sinus greatly dilated and contained pus mixed with yellow broken down pus and clots. Right ophthalmic vein similarly affected, and the circular sinus with the superior petrosal on the right side. Left cavernous sinus contained a clot yellow in the middle. Other sinuses healthy. There was subarachnoid abscesses in the lungs with pericarditis, well, and no signs of adjacent inflammation. Liver fatty and enlarged; kidneys healthy.
As Mr. Murray remarks thrombosis in all likelihood had extended along the petro-occipital sinus to the right cavernous sinus, thence through the circular sinus to the left side.

Case 12. A labourer, aged 24, said to have had
knocked his head against a wall suffered from subarachnoid abscess of bones of skull for some months. He suddenly, after a little daunted by present incision, developed symptoms of thrombosis of longitudinal sinuses with Cothoil epilepsy as a result. The epilepsy caused by the epilepsy rapidly caused death in 17 hours, having been quite unconscious all that time. There was chronic encephalitis of scalp.
Post-Mortem. Drainage right malar, left frontal and left zanatar bone. Cleft in longitudinal sinus posteriorly post-mortem, partly detached, soft and moist, clotted anteriorly from acute sinuses and adherent, extending to Cribrum Galli; clot dark brownish black except opposite middle of superior frontal convolution. When dry pale. There was a large vein absolutely blocked by acute sinuses, clot over side. On left side a small vein similarly blocked. There were three horn-shaped extravasations near the sphenoid sinus. Congestion of right ascending convolution and thin fibrinous exudation over whole of left hemisphere. Both lungs thickly studded with ordinary tubercles.

In this case the thrombosed lateral veins compared to the diseased posturing bone, the thrombosis evidently having commenced in these small veins and extended into the Superior Longitudinal Sinus. It is interesting to note that the portion of the brain thereby affected corresponded exactly to those marked out by Pierre Richer's centre for these particular movements noticed in this case (see his "Function of Brain" 2nd Ed. page 405 to 492. Ansas 2nd Ed. p16 & 112).

Case 19. Adult Male, robust, had a blow on the back of the head. Slightly stunned only. 3 days after headache, paresthesia & numbness, persisting taste, since of small amount. Vision cloudy,田空空. Pupils dilated. Optic discs swollen, patches of
Actual haemorrhage with nose large sttreous. Encephale. Scalp 4 days before death. Paralysis and coma. No convulsion nor rigidity. Temperature 100.2, pulse 160.

Post-Mortem - No fracture. Both cavernous sinuses completely plugged by fibrous clot. Central anhi healthy. Recent haemorrhage into right anterior central lobe, which contained a clot surrounded by punctiform haemorrhages. There had been complication of the orbit.

In this case one cannot help thinking that probably the encephale was antecedent to the symptoms noticed in connection with the eyes, and was the cause of the thrombosis although the order of symptoms in the report would lead one to think otherwise.

The 3 last cases (11, 12, 13) gave much more definite central symptoms, 12 and 13 especially pointing to the presence of thrombosis of some of the central sinuses.

Cases 84, 85, 86 are three cases of thrombosis of the cavernous sinuses occurring after cataract with malignant facial carbuncle, related by W. Davies Coley before the Medical Society of London (Curl Lancet I, 1886 Vol. I, p. 573). These cases show the connection between the facial veins and the cavernous sinuses.
The next case is one due to inflammation of a different part, namely the Dura Mater itself.

Case 17. A case was related before the Medical Society of London March 22nd, 1886 (Vide Lancet Vol. 1, p. 598) by Dr. Sidney Coupland. It was a case of basic meningitis in which there was total ophthalmoplegia and loss of sensation in the ocular tissues. At the post-mortem there was found mucous plugging of both cavernous sinuses. During life there were no signs of papillitis, and no choked disk.

In this case, although nothing is said of it in the report, there would undoubtedly be some phlebitis of the veins of the Pia Mater concurrent with, or else consequent upon, the meningitis; and thus the thrombosis would be hastened, just as happens in other sites.

Case 18. A healthy girl 62½ months old. Had scarlatina at 6½ months, recoverd well after. Had ophthalmia, improved in 3 weeks. Then a fit of convulsion, no other central symptoms. Convulsion did not return. Then an abscess formed at the umbilicus, then Pneumonia, Child thin, weak, hectic. Then sudden extreme faintness, almost perfect syncope. Rallied, and 24 hours afterwards faintness proved fatal, without any convulsion just 6½ months after the scarlatina.
Post Mortem. Emphysema & Petritis. Three
pieces of pus was found in the abdomen. There
was inflammation of the Superior Longitudinal
Sinus. The Sinuses on the left side were healthy,
but the blood was almost entirely coagulated.

The posterior half of the Longitudinal Sinus,
the torcular, the left lateral, and the left occipital
Sinuses were blocked up with fibrinous coagulum,
precisely such as one sees in inflamed veins, and
the clot extended into the internal jugular vein.
The coat of the longitudinal, and of the inner half
of the lateral Sinus, were much thinned, and
their lining membrane had lost its polish, was
uneven, and presented a dirty appearance.

The above case is twice referred to by Dr.
Sellers Smith of New York, in his work on
Occasions of Infancy & Childhood, that is on
pages 355 & 357. It is evident he made a mistake
and leads the reader to suppose there were two
separate cases. It should be noticed in
this case that death was 24 hours before
death had been recognized and the
child returned and proved fatal. The Throat
would not seem to have come on only about
2 days before death, in account of the absence
of the Superior Sinus being empty upon the
general condition. The latter was plainly
a remnant of the Petritis and gave
rise to the Anasarca pleuritic effusion,
Abdominal Abscess &c. Then is no mention
of the Clots being brownish or disintegrating.
Had the latter been the case, we should, in all probability, have heard of pyemic abscesses in the lungs. Unfortunately no mention is made as to the state of the heart. It is quite possible that the Syphillis with its affections in the heart, for instance, may have been due in part to a portion of the thrombus in the Inferior Vena Cava becoming detached and carried into the heart and perhaps caught there.

II. Secondary Thrombi from Injury.

The following 10 cases have all been due to some inflammatory process consequent upon disease; the next three cases illustrate the connection between injury and Central Thrombosis. They are placed here, as Inflammation is so frequently present in such cases, and may be the cause of the Thrombosis spreading; though, it is possible that mere exposure of open wounds of orbits may in some cases be the cause apart from any inflammation.

All the 3 following cases are from Hutchinson's Illustrations of Clinical Surgery, Volume I.

Case 19. J.W., a healthy boy aged 10, had a wound on left side of scalp from a dog bite. Exposure of bare bone. Excellent progress for 14 days, wound healing. Then double pneumonia set in with rigors (severe) during which (rigors) the pupils were dilated. 2 days later left papil became slightly contracted, face pale. He died in the...
23rd day after the injury. Quite conscious up to the last, no paralysis or convulsions. The pupils had become equal again on the 10th day. Post-mortem on the following day, pleuritic adhesions & numerous pyramidal deposits in both lungs, liver & spleen. Left common iliac vein plugged with large recent-clot, with a large sized yellow (pale) in the middle. In head, vertex, surface of Calvaria, on both sides of sagittal sutures green and closed down, also whole of left parietal bone. One between Duran Mater & bone patches with Virgi's leading from there to Sup. Long. S. Sinus dilated with puriform fluid. That sinus contained in part a recent black blood-clot, and in part a dirty puriform, thrombus, in anterior third no thrombus. Lateral Dura-mater vessels contained only recent-clot. The thrombus at Sup. Long. Sinus contained no red blood clots, but was made up of moiliary matter, debris & cells like those of Sinus.

Case 4261

A man, aged 30, fell on feet about 40 feet, and a chain fell upon head. He sustained a Pott's fracture of left ankle, and seven contusions and lacerations of scalp. Favourable progress for 12 days. Then rigors, all wounds healed but one; sickness, abdominal pain, some tenderness about left shoulder. Abscess formed in left sternoclavicular joint on 20th day. Face pale; skin related...
time, and sustained a lacerted wound of
scalp, with laceration or chipping of outer
table of skull at two points (no fracture through
both tables), the wound was rather to the right
of the middle line and parallel with it.
All went well until 14th day, most head symptoms
before them, sweating and headache for two days.
Daily fevers, rapid emaciation, pain in head,
proximis constipation, pulse quick, no appetite.
Ere delirium, & quite conscious until within
a few hours of death on the 18th day.
Post Mortem. Some bone was chipped throughout
whole thickness. The Sagittal longitudinal sinus
was filled with stomach lymph adherent
to its walls, and with a fluid exactly like pus
by which its cavity was distended. In its
Anterior part there was some coloured blood-clot,
and again some posteriorly. About 2 inches
above the fontanelle the clot became
recent. There was no mention of other parts
in the report.
In the last three cases it is interesting to note
that, all being healthy subjects at the times of their
accidents and having injuries similar and
in similar parts in all having lacerations
of scalp over or near the vertex, all had a very
similar period of favourable progress to
commence with. In Case 19, fourteen days,
in Case 20, twelve days and in Case 21 fourteen
days during which no central symptoms
were noticed. All after that developed eczema.
for it is reasonable to suppose that in Case 21 there was pyaemic infection of the system judging by the rigor, certain countenance, loss of appetite, &c. though we are not told what was the condition of the lungs and other organs. All presented alike uniform disintegration of thrombi. All were unconscious up to the last. Case 21 was only conscious a few hours before death, a not unusual condition when there is great weakness or prostration and approaching death. And, with the exception of a little wandering and defective memory for the last day or two in Case 20, there was no delirium, convolution, or paralysis in either of the three cases.

It is evident that clots formed first in the open exposed veins in the wound, and spread thence through various emissary veins to those of the Ova Mora, and so on into the chief longitudinal veins. This part of the pathological process would necessarily take some little time, and while the thrombus or thrombi were extending in one part, it would be softening and disintegrating in another. And as in all likelihood the thrombi would not at first block up the whole lumen of the vessel, some of the puriform matter from the softening thrombi would be able to pass along the sides of the vein and after infecting the prolongation of the thrombus in its way would at length find its way into the internal jugular vein and so into the
General symptom. Until this had happened, there might be no special symptoms of the coming trouble. The venous circulation would be able to be carried on, though perhaps not so vigorously; hence the reason of the early latent stage. As there was no marked trace of active inflammatory trouble in either of the cases 19 or 21, so there was no delirium nor convulsions: while in Case 20, when the Dura Mater was adherent to the Calvarium, there was "a little wandering and defective memory" during the last 2 days. This has been new paralysis nor Cereus is to be explained partly by the blood being able to some extent to pass the thrombi, but more fully by the localization of the thrombosis. All the veins not being blocked at the same time, other veins were able to take up the blood, and carry on the circulation without so much hindrance as in cases of more extensive thrombosis. It should also be noted that in Case 20, when there was the greatest amount of pus found over the brain (in Case 19 it was only in the patches), there was the longest interval between the onset of pyrexia (14 days, the other being 9 days & 4 days) and the death; and that in the case when there was the least pus namely in Case 21, death occurred soonest after onset of pyrexia, that is 4 days. The only conclusion that can be drawn from this
observation, is that it is possible that the longer duration of life allowed of more extensive breaking down and disintegration of thrombi.

Before passing on from Secondary to Primary Thrombosis I should like to add one note, which should properly have been made after Case 10.

That note is that in connection with the inflammatory processes Hughes Bennett in his "Principles of Practice of Medicine," 5th Ed., on page 133, says "Inflammation increases the amount of fibrin in the blood from 3 to 10 in 1000 parts, doubles the quantity of Cholesteroi, and diminishes the albumen." If such is the case, it is much easier to understand Thrombosis by prolongation in cases when any inflammation is present. As I have remarked above (page 29) in cases of injury some amount of inflammation--either little or much--is frequently present during or just previous to repair; and sometimes acting in later stage of repair, that one cannot help suspecting that a great many cases of injuries to head with wounds of head (or other part of the head) all of thrombosis of the cranial sinuses, and not of any direct injury to the Brain, are which is so often guessed at but not certified by post-mortem examination.
I will next pass to the considering cases of primary thrombosis — i.e. occurring locally. According to von Dusel's classification, which I am endeavouring to follow, the first of the general causes requiring the circulation is deficient cardiac energy. Of the two first causes of the latter, namely, infancy and old age, I have some instances; but as it is so difficult to be able to say that thrombosis is alone in either case solely at the time of life, I have classed these cases under the various other causes that were present in each case.

In fact as von Dusel remarked in his monograph most cases have more than one condition through which produce thrombosis. This makes classification of such cases a very difficult matter, and all the more so when we have to deal with so many cases of which very imperfect or incomplete reports are given. The third cause of deficient cardiac energy is precedent acute or chronic disease.

Of cases illustrating this I have collected 15 cases. 7 cases of Anaemia, 1 of Chronic Epilepsy with Central and General Anaemia (slight). 2 of Chlorosis, 1 of Mental Depressing Anxiety, and 4 of other chronic debilitated states. Of these cases the first of those of Anaemia, Case 22 is that of J. R. B related at the commencement of this paper.
Case 23. Mary J. - Aged 23, domestic servant. May 23, 1889 had sudden pain in right frontal region; headache and vomiting until June 6th. She took to her bed on May 30th. She was anaemic, and emaciated, but features sunken and with expression of extreme pain. June 6th: answered drowsily. Kidneys behind left mastoid process; nothing else; incubation of ear. Left pupil slightly larger than right; converging revisits of both eyes, distinct ptosis and slight hage. No anaesthesia or paralysis. Reflexes all normal. Pulse: 70-80.

June 9th: For 6 days got worse; continued drowsy and difficult to nourish; pain in head and behind left ear. June 16th: Right arm and hand slowly became paralysed; right foot very weak; nothing else. No modification given light placed before the eyes; but moved head when some smoke was sent up the nose. Of the neuritis and intense, paretic involuntarily. Tenderness at back. She was more sensible, recognized faces, but paralysed the same. Next day quite sensible. Use of right arm almost complete. Neuront. Headache slight.

May 14th: She improved up to the morning of the 15th, when laughed; power quite recovered. On the 16th at 4:30 a.m. she sat up in the bed, turned to the left, eyes turned to left; she lay down on the left side & vomiting. After that she turned on her back & drew her knees up, breathing deep & noisy. S 2 to 4 minute with coughs and then. Pulse
Pulse 80 very feeble; face only white. No paralysis; able to swallow, probably conscious. At 6 a.m. pulse 90 very feeble, fluttering; face deathly pale, cut blue at all. She died at 6 a.m. apparently conscious up to the last.

Pathology - All the veins on the surface of the brain were plucked by adherent and partially discoloured clot from the lateral aspect of the hemispheres to their opening into the Sup. Long. Sinus. A moderate quantity of dark fluid blood escaped from the Sup. Long. Sinus. The Veere Galeni contained clot like the other veins described. The whole of the left Lateral, the Straight Sinus and Torcular Herophili contained similar clot. A little pale clot was found beneath the right Lateral Sinus; elsewhere dark fluid blood. The walls of the anterior sinuses appeared healthy. No trace of disease in the left ear. The left frontal regular vein to a level of the angle of the lower jaw contained a clot like those described. There were two small haemorrhages of about the size of a pea in corresponding parts of the white matter in each hemisphere. No other trace of disease anywhere. In the uterus was a foetus about 6 weeks.

Dr. Brownes thought the death to be due rather to sudden collapse than to any definite cerebral cause; and draws attention to the Anaemia and possibly mental distress at the illegitimate pregnancy.

Comment: It is a little difficult to understand...
how it was that the paralysis ensued off
and recovery appeared probable. One suspects
naturally whether the clots found in the Lateral
Sinus and Internal Jugular Vein had come down
from the Superior Cerebral Sinuses, and then lodged
when found, extended back into the Venous
sinusphic Straight Sinus. It is probable that
some blood was still able to flow past the clots,
and if a clot in the Long Sinus slipped away
that would put the circulation in that part somehow,
as the clot going into the Lateral Sinus, the other
could carry on the blood.


Health good up to 12 months before, when she became
anemic and aneurysms began. She became tailed
with pain in left side; ankles edematous; anemic
murmur heard in the neck. Frontal headache at
first slight, got gradually worse, so that on April 2d
she was kept awake. On April 4th she vomited several
times; was delirious during the night. First day she
became comatose. Pulse got to 120 p. H. highly irregular.
She died at 10.30 a.m. the 6th.

Post Mortem. 24 hours after death.

Brain: of diminution with hemorrhage into Putamen,
Central ganglia, and posterior parts of each hemisphere.
The right side was worse than the left. Veins of Choroid
pleurae when interposition, Virchow-Robin also tended
to firm, partly yellow fibrinous clots. These extended
continuously along straight Sinus and for one inch.
In the Lateral Sinuses. In the latter they do not fill the vessels. Oldest clot at junction of straight & lateral Sinuses. Blood clotted then fluid. No disease of Central arteries. No tubercular disease anywhere.
Several branches of pulmonary artery obstructed by old clots, some breaking down in their center. Large echymoses beneath & dorsal vein in left Nucleus, the Nucleus (left) was poorly contracted. In the juxtanucleus were 3 recent intracerebral Hemorrhages. Comment. It should be noted that in this case the Superior Sagittal Sinus, and upper surface of the brain was free from clots; this being the most common situation for them in this class of cases. The central edema would be caused by the obstruction to the central circulation.

Case 25. A lady, below middle age, Anacurie, for a little while kept out of health. She was seized with headache all over head, but especially at the back, constant with paroxysms of great severity. Pulse weak.
After 8 days weakness came on. Next day slight weakness in right arm & leg, which passed off. She continued the same; pupils contracted. Intelligence intact. 8th. On the 11th day she became very sick, & towards evening half conscious. 12th day limbs became rigid, legs fleshy, then convulsive twitching came on, and she died.
Post Mortem. - Surface of brain tinged with blood; vessels of pia mater everywhere tightly filled with clots; also longitudinal Sinuses completely closed by fibrin. 

Wilks, Diseases of Nervous System, 2 Ed. p 179.
to its walls; in some places this clot was softening in the center. Laterally Sinuses filled in the same way as far as the Jugular Venæ. A little yellowish serum in the meshes of the perineurium, and in one or two places a patch of recently-effused blood. No disease of bone, perineurium, bodies, healthy, no signs of coagulation elsewhere.

Case 26. A young woman aged 20 years was admitted into S. Bartholomew's Hospital for Anaemia and Anaemia. A few days after admission she had intense headache, was dull, despondent, but answered questions rationally. She then became rambling and somewhat excited. Coma supervened followed by death. Post Mortem Vena Galeni, Straight & Lateral Sinuses occupied by ante mortem clot. Both Optic Thalami broken down by haemorrhage into their substance; & a small mass of extravasated blood in posterior part of left hemisphere. Meninges and general substance of brain cut particularly congested. Heart healthy, with no trace of old clot.

Case 27. This case is fully reported in the Lancet, so I shall simply note the leading facts: Jane H., aged 20, Anaemia, always delicate, was cried with eczema, & fear & pain all over head, which ceased for a while & then returned worse than before. She gradually became emaciated with left hemiplegia; motor power involuntarily, face flushed. Eyebrows drooping, eyes turned downwards, right somewhat inwards, pupils dilated. Seen by Dr. Church, S. Bartholomew's Hosp. Report 1869 p. 179. - Dr. D. Nunn. Lancet, 1869, p. 72.
8 week in the small cloud.noticed together. Respiration
30, shallow & irregular, with sighs among them. Pulse 72 and
irregular. Regular. Temperature 104.2, then to
105.8 during the last day. Legs extended; knees quite
elevated; tos generally markedly elevated. Toric
spasms with flexing adduction of shoulder, and
extension, yellow fingers. No chronic spasms.
Came complete at 7 pm in last day. Pulse 38. Pupils
dilated; retinal vessels full; no papillitis. She
was placed in bath at 90° reduced to 80° for 35
minutes. Temperature fell to 100 pulse to 60.
Temperature then went down to 90° and death
occurred suddenly two hours later.
Post-Mortem. Body well nourished. Whole
brain, especially right half, abnormally soft. A firm
contraction of sub-mortal clot in both Jugular veins
which was older about 2/3 inches above torcular.
Straight veins contained a firm particulated clot;
chiefly dark, but paler at branchings into tributary
vessels. The clot extended through the Torcular, where
it was older for 1/2 inches along the right lateral
Sinus. All arteries and other sinuses of brain
healthy. Right Cerebral Mural major studded
with minute multiple hemorrhages. Red softening
of right Capsule Striation Postihe Thalamus. Clot
in right Ventricles from coag from Choroid plexus.
Veins of brain much distended and like, completely
filled leg from half-discord clot. No local cause
of the hemorrhage discovered. All other organs healthy
with the exception of slight erosion of uteri.
Case 28. Margaret Meach - aged 19, a governess, getting anemic & liable to fainting for 12 months.

8th Oct 1886. Anemic with frontal-occipital headache, 13 fits, apparently true epileptic, but she did not bitter tongue vomit her water in the fits. Temperature rose from 98.6° to 102° in a few hours; when fits ceased, and she became difficult to move. For next 12 days, drowsy, but no more fits. Headache and often sick without obvious cause. Temperature on 8th Oct rose to 101.4° on 4th to 101.8°; 5th to 99.9° to 100.8°; during next 7 days, generally normal. Improvement soon on. On the 8th drowsy and very nervous, complained of right side being numb, no paralysis nor absolute anesthesia; sensation impaired over extension of right forearm. Knee jerks brisk, ankle & knee clonus on both sides, but chiefly on right. Sore in the morning; pain in back & neck.

8th Oct. Normal pulse 104. She had a slight fit, with shivering attended by scream over left arm. During fit she had pain in her right arm. On 16th fit it was better. Anesthesia gone, but she had frontal headache and acetamin phosphate right ear. No deafness, and no discharge from ear. Still had weakness applied behind the ear, and she was given 1 gr. graving. Then was tenderness swelling about the upper part of the external jugular vein, with slight deafness afterwards, which lasted for several days, and then subsided. Slight but obvious. Anosmia of optic nerves; supported the idea of thrombosis of lateral Sinus.

One or two days afterwards she was attacked on the left side, but not so badly, no deafness. This got better.
better in 8 or 4 days. Two days later phlebitis set in right leg in Hunter's Cuscal. This lasted a few days and subsided. She after that had one slight hysterical fit and slowly recovered.

Dr. Batro thought she really had phlebitis of one of the lateral veins and internal jugular vein. Another of the other. The carotic symptoms were not conclusive, but highly suggestive.

"It may be reasonably presumed that central phlebitis was determined by long continued anaemia, which is a well-known cause of thrombosis in the blood vessels."

**Case 29.** This was the case of Martha B. as related in page 9 of this paper, in whose case there was ascension, but especially of the brain. But, in addition thereto was the epilepsy.

**Case 30.** In a discussion at a meeting of the Medical Society of London in March 1886, Mr. Allingham, Jnr., mentioned the case of a girl aged 21, who died with thrombosis of the facial vein and Carotian thrombosis; there was extreme Chlorosis.

**Case 31.** Dr. H. M. Tuckwell mentions a case in a paper on "Clothing of the Blood in Some Chlorosis."

E. C. aged 16, with well-marked Chlorosis. The chief symptoms were headache for 10 days before death, expression dull, shivery, moist, speech slurred. Pulse 80 regular, systolic murmur at base.

and nausea continued in neck. A day before death, could not sit up steadily in a chair. Left hands and arms numb and paralyzed, headache severe and constant, referred chiefly to frontal region; this persisted, the paralysis of arm became more marked, until the last morning, when she was found foaming at the mouth and comatose, and died within an hour.

Post-mortem 24 hours after. Dura mater over right hemisphere thick. "Longitudinal Sinus in its posterior half blocked by a firm pale clot of some age." Central vein over right middle and posterior lobes very obliterated by firm dark blood clot. Blood effused in right middle and posterior fossae, and over right half of sensorium, & extruding down into the Optic Canal. Lateral Sinus, both filled out by firm pale clots, that in right one beginning to soften. Straight Sinus and the Meningeal sinus also filled with similar clot. Poor Visceral and Medulla surrounded by great clot. Only the anterior lobe of the right hemisphere was healthy (i.e., of that side only). Medulla and posterior lobes were soft, wet, pulpy, collapsed, and of a deep red colour, and in the midst of it was a clot as large as a small-sized walnut. Lateral Ventricles completely disorganized, containing much soft blood clot. Optic Thalamus soft on its surface, former incisus, Corpustercumbrense partly destroyed. The left hemisphere, the posterior half was softer than natural, but much less disorganized than the right. Lateral Ventricles contained bloody fluid. Optic Thalamus very soft. Corpustercumbrense much


Caudal and septum lucidum almost destroyed. No disease in any bone of the skull, nor evidence of any inflammation either within or around the sinuses. Cerebral arteries all quite healthy.Microscopic examination: Caudal brain showed broken-down fibers, granular matter of blood cells. No where was there any evidence of abnormal cellular or other structure.

Comment: The result of the post-mortem examination in this last case suggest very strongly that the derangement of the right side of the brain was due to impaired circulation from occluded blood vessels. As the arteries were all right and healthy the occlusion must have been in either the capillaries or the narrow channels. Had the obstruction been in the capillaries, it is hardly conceivable that the occlusion could have been so general over one side of the brain while the other side was clear. It could have been more local and in patches. Therefore we are reduced to the one remaining possibility namely that the obstruction was atherosis. And here, there was no evidence of any other possible cause, it must probably be atherosclerosis that started all the other disease described. It is certainly unusual for atherosis to cause so much disease, but in a case well marked atherosclerosis, as this was all the tissue would be in such a state of low vitality, that greater changes might be expected to result from any particular lesion, than is usually met with.
In considering the connection between Anaemia and Thrombosis of the Cerebral Vessels I shall include in it Chlorosis, owing that is so closely allied to Anaemia. In fact it would not be well to separate them here, as the terms "Anaemia" and "Chlorosis" are, I am sure, generally used in such an indiscriminating manner, that one cannot feel sure that all the cases here related are referred to nor rightly properly named.

Drusch does not mention Anaemia in connection with Thrombosis of the Cerebral Vessels. He is very approach to it made by Siddell in his paper on "Thrombosis of the Central Nerves and Sciases of the Dura Mater" in the first volume of the American Journal of Med. Sciences for 1874. Siddell only treated of "Traumatic and Inflammatory Thrombosis."

The first mention I can find of it is by Dr. Church in the St. Bartholomew's Hospital Reports Vol. 6, 1869, page 177 (or 178). He then says "In reference to a case related by him in his "Contributions to Cerebral Pathology" - Thrombosis of the Cerebral Vessels and Nerves, as occurs occasionally in anaemic and debilitated conditions, etc." Dr. Wilks in his work on "Diseases of the Nervous System" (2nd Edition page 179) refers to it but in very cautious terms, the also related these 2 or 3 cases, which I have inserted into this paper. Dr. Bristowe in his work on Diseases of the Nervous System refers to it (vide page 442 this paper). Mr. T. D. Rivers in his book on "Diseases of the Nervous System from Ross in his, nor
Nothnagel in his contribution on this subject in
Zwinger's Encyclopaedia of Medicine mentions
Anœmœma as a cause of Central Thrombosis.
So that the literature upon the subject is de-
cidedly scanty. For that reason I shall inter-
mezze fully into this part of the Subject of Central
Thrombosis, than I have done or shall do in others,
seeing that the other parts have received notice
from various writers.
Firstly—can we justify in connecting Anœmœma
with Thrombosis of the Central Sinuses? I think
we are. And that is for 2 reasons; firstly, the
references made to it by authors, notably
Dr. Church, although many writers omit it mention
it. And secondly on the strength of the 10 cases
collected together in this paper: Or 9 cases only,
if the objection is made against Case 6:28
that there is no positive proof that Thrombus
actually occurred. Nine cases are not a
great number from which to draw any
conclusive conclusion. But, if we include
well investigated cases, they would be sufficient
to enable us to affirm a possible connection.
Secondly—Supposing Central Thrombosis and
Anœmœma to be connected, how can such a
connection be explained pathologically?
In considering the formation of a Thrombus,
we have to take into account, the predisposing
causes and the special or exciting causes.
I shall deal with these separately.
Firstly, the predisposing causes are contained in the anatomical structure of the various channels in the Cranium, and include rigidity, non-compressibility, and want of contractile power in the walls of the Sinuses; their peculiar shape, being more triangular than round, and thus affording a larger proportionate amount of surface to the bloodstream; the dilatation at certain points, as in the Occipital and Lateral Sinuses, the angular projections and the fibrous bands crossing the walls of the Superior Longitudinal Sinuses. In the latter sinuses we have also the way in which the vessels from the hemispheres join the Sinuses, some joining against the bloodstream and others more or less at right angles to the Sinuses. We have also to bear in mind that the Cranium is at the top of the arch formed by the bloodstream, before it commences to descend again. It will thus have lost a greater proportion of the impulse of the heart than in other situations. Another, though slight, impediment to the circulation is that there are no large muscles or parts in active motion in the immediate neighborhood of the brain (excepting those outside the Cranium) such as are found in the limbs and other parts. Such muscles and mobile parts must help the circulation whenever present.

Secondly, are the special or exciting causes of Central Thrombosis in the Aneurism. Causis chiefly in Changes in the Heart and in the Blood. In the Heart we find fatty degeneration of the.
The vascular fibres of the heart, and palate of fatty degeneration on the central valve. The former condition by weakening the heart will greatly interfere with the force and rapidity of the circulation, and then tends to stagnation of the blood consequent thereupon. In the blood are the root of all the evils in Aneurina. The quantity of the blood is lessened. Thus (as explained by Von Gericht in his monograph in the New Hydriphane Society's Works) the flow of the blood on account of not completely filling out the vessels. As to the composition of the blood, it is not at all clear that that tends to produce thrombosis. Indeed Dr. Compland mentions the blood, as found by post-mortem examination after Aneurina, as "fluid, thin and watery and scanty": though he mentions at the same time "pale clot of an annulated with the fluid blood in the chambers of the heart." Also Mitchell Bruce says, the blood "coagulates slowly and loosely, or in aggravated cases not at all, settling into three layers." So that on the other hand Playfair in the second volume of his work on Midwifery (p. 343, 2nd edition) refers to thrombosis after surgical operations as being especially liable to attack a patient when "in a weak and anemic condition". In Jones & Sire's Pathological Anatomy it is stated (p. 38, 2nd ed.) that in Aneurina the aorta is quite unaffected; it was never found below the normal mean; and in cases where inflammation of some organ was present its quantity was notably increased.

The latter part of the sentence, when read by the light of the statement by Hughes Bennett as to the increase of fibrin in inflammation leaves but little room for considering that any increase in the proportion of fibrin this is due to the anemia itself. Still, as it does not go below the normal amount, clotting should not be much more difficult or slow than in ordinary healthy blood. Another point in connection with the blood in anemia is its effect upon the heart. Not only does a supply of impoverished blood through the coronary arteries lead to a starvation of the muscular tissue and a consequent degeneration; but also there is good reason to believe that both an account of diminished quantity and altered gas composition, it will provide a smaller stimulus to the cardiac ganglia than the normal fluid; and thus the heart action will be less vigorous, and the circulation retarded to a corresponding extent.

Beyond the condition of the blood itself, we have frequently in cases of anemia occurring in young women (or girls) a certain amount of abnormal contraction -- or rather want of expansion -- of the chest caused by tight clothing. I have met with tight clothing and consequent insufficient expansion of the lungs so frequently, and so frequently have observed that all the various remedies for anemia, diyeriments have failed to improve the condition of the blood until tight clothing had been given up, that I am fully convinced that abnormal compression of the chest and abdomen is frequently a factor.
in the production of the Anæmia. Not only so, but it must also act very prejudicially in rertarding the circulation both by the compression of the contents of the chest, and also by pressure upon the large galls which result in the abdomen.

In case 22, I had previously had to advise and warn my patient as to the evil effects of her tight-lacing. She certainly loosened her things a bit when under my care in the October preceding her death; but I have reason to believe from the statement of her mother, her mistress, that she had latterly dressed as tightly as ever.

Symptomatology. In order to compare the 10 cases (22 to 31) I have tabulated the most important symptoms— vide table in next page. In reference to that table it will be found:

1st. All the cases were in females. This is not at all remarkable, seeing how seldom we meet with either Anæmia or Thrombosis in male patients. Among the few cases included in this paper altogether there is one case only (28) to be related further. When there was an approach to Anæmia in a case that case is not included in the Anæmic class, as more likely caused both the Thrombosis and the Anæmia was in the kidney. Though as far as the Anæmia went, it might well have been included under this heading.

2nd. Eight patients out of ten were between the ages of 16 and 25; of that number only two were below 20 (cases 28 & 31). Of the remaining two
### Analysis of Cases of Cerebral Thrombosis in Anæmia

<table>
<thead>
<tr>
<th>Case</th>
<th>22</th>
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One was below middle age (25), while the other was 47. The matter of age goes very much with the sex, so that this point also is not at all remarkable, seeing that it is chiefly between the ages of 16 and 25 or 27 that young women suffer from Anæmia. This is no doubt partly due to the conditions of development, and partly to unhealthy conditions of life during that part.
of their lives, such as confined air in ships, work rooms &c. long hours of work, hurried meals &c.

§ 8 - Temperature. In only 3 cases (22, 27, 42) was a rise of temperature reported. Inflammation and a consequent heightened temperature are not common in connection with these cases. But of this it is impossible to speak confidently, as so many of the reports are incomplete, that it is possible something might have been noted in the temperature. We should naturally expect the temperature to tend to become sub-normal. In case 22 for the last two days of life it was sub-normal, while in Case 29 it was sub-normal for 5 days before death. With diminished vitality and depressed circulation it is difficult to understand how the general temperature is likely to be raised beyond the normal. Tissue change, exsanguination and the various other chemical and physiological processes going on in the body are all likely to be diminished, except in cases when, from some local or particular cause, any local inflammation was set up. I strongly suspect that in cases of cerebral thrombosis in Anemia the temperature, if carefully observed, would generally, at least, be found to be below the normal.

§ 9 - Pulse. In four cases we have a nearly normal pulse. In the other cases we have an insufficient one to go upon. This is an important point in considering the causation of the Thrombus. And it is unfortunate that the case
an not better reported in this matter.

5th Vomiting. This was present in 5 cases out of the 10. In two (228 29) it is reported as absent, while in 3 cases no mention is made of it. In the latter 3 cases it is probable that it was not present, but beyond that we cannot go any much. This only shows that vomiting is not a constant symptom.

6th Congestion of the face, which is mentioned by some authors (but restricted to venous congestion) was only noted in 3 cases - though it is possible it may have been present in much that number. In 2 of the cases (223 27) the face was arterially congested, in one only (29) was any venous congestion noted. Dureh, Koth nagel, Bowers and others mention this symptom as an important one for diagnosis when present: That is just it; it is so frequently absent, that we cannot look for it, and put aside our diagnosis on account of its absence. It is supposed to occur in cases of thrombosis of the Superior Sagittal Sinus, and to be due to the blocking of that Sinus. This is on the assumption that the foramen occiput at the anterior end of the Sinus is patent. In most adults it would seem as if it were closed - see Dr. John Knaus's paper on the Anatomy of the Cranial Sinuses, (reported in the Transactions of the International Medical Congress in 1881, Vol I pp 191 to 199) where in the say, that "through the foramen occiput it is the Sinus..."
Longitudinal sinus) commences in early life with the veins of the nasal fossae, but this anastomosis is usually cut off in the adult. If this is the case, it will account for the frequent absence of the symptoms of venous congestion of the upper part of the face. A certain amount of palpebral venous congestion may be present in occlusion of either of the cavernous sinuses, but there are so many other causes of the same symptoms, that it could only help in a diagnosis already strongly suspected to be correct.

Epistaxis was not reported in any of the ten cases, although mentioned by some authors together with last named symptoms.

Optic neuritis was only noticed twice (Case 23 and Posterior Longitudinal Case (Case 27). We should expect a more frequent record of these symptoms; but, their not having been mentioned, probably, arises from the eyes not having been examined by the opththalmoscope.

Paralysis was noted in only 4 cases, in 2 of which it was temporary. In my own first case (No. 22) it was not certain—account of the prolonged and deep coma—whether then was any paralysis or not. It is notable that in all the four recorded cases with paralysis, this was in the arm, hand, or both from on one also it affected the rest of that side of the body (Case 24). In case 29 when the right arm and hand were only temporarily affected, it is contrary to state the real cause.
This was occlusion of the left lateral sinus amongst others, then core also two small haemorrhages in corresponding part of each hemisphere, but their exact locality is not stated. In Case 25 there were patches of recently effused blood in the meshes of the pia mater. Whether these conditions alone is sufficient to explain the paralysis. It is most probable that plugging of the Vesi on the upper surface of the opposite hemisphere, towards its anterior part occurred first leading to paralysis of the centre marked out by Perrier for the upper limbs. The clearing off of the paralysis may be explained by a venous anastomosis with neighbouring Vesi allowing of a renewal of the venous circulation after a short time. In Case 27 when there was left hemiplegia, there was found extensive disorganization (chiefly and softening) of the right central ganglia. In Case 31 when the left hand and arm were "numb and paralyzed", there was found extensive effusion of blood over the right hemisphere, effusion of blood into the right middle and posterior lobes, and softening of those lobes, besides partial disorganization of the corresponding central ganglia. It would thus appear that temporary paralysis may be looked upon as a physiological result of local venous congestion and stagnation; while persistent or permanent paralysis is due to some pathological lesion, and is consequently of much greater importance.
Muscular spasm was noted in only three cases (2, 25, 27) if we except Case 20 when the spasms only occurred at the time of an epileptic fit. In Case 23 there was convulsive action of the o'facial and ocular muscles, turning the head and eyes to the left side. In Case 22 the tonic spasms occurred at quite an early stage of the disease, and only lasted for about 30 hours. In Cases 23, 25, 27 there was also paralysis, as mentioned before.

In Cases 25 and 27 the muscular spasm affected the same parts and it would seem the same muscle as were affected by paralysis in the Case 25 coming after the paralysis, in 27 before it. Whether the paralysis and the muscular contractions were really connected must be left until new data can be gathered upon the subject.

Reflexes in 3 cases only are noted. In 1 they were normal (Case 23) in 1 diminished (Case 29) and in 1 lost completely at one time, and afterwards slightly restored. These data are insufficient from which to draw any conclusion.

Headache was noted in 9 out of the 10 cases. In the remaining case (20) no particular headache was given, nothing can be said. In fact for the general use of the table on page 53 it would be better to exclude that case. Of the 9 cases with headache, in 4 it was frontal, in 1 frontal and occipital, in 3 it was general, that is more or less all over the head, and in 1 (Case 22) it was not ascertained exactly where the pain was. So that in 8 out of the 9 the pain attacked the frontal region, in 4 of these
it was felt elsewhere as well. This does not agree with the observations of Dr. W. H. Day, who says that the headache from Anæmia is most frequently vertical, though it may also frontal, or occasionally occipital. Again he says "the headache from intellectual strain and severe gastric disorder from over indulgence in alcohol and high living is almost invariably frontal, and the veins about the temples and forehead are full, and the face flushed." As the latter form of headache is more one of congestion than of Anæmia, it naturally suggests that the headache found so frequently in the cases under consideration is one of congestion. But this is most probably passive congestion from blocking of the venous channels. This will cause pain chiefly by mechanical pressure, the peri-vascular spaces having been already emptied in all probability; it may also increase that pain through the action of cholesterin products in the venous blood being retained in the part, and by a slow exosmosis reaching the cells in the brain substance in the gray matter.

13. Delirium was noted in two cases only; while convulsions were not noted in any cases but 28 & 29 when they were epileptic. In Case 29 these were not looked upon as dependent upon the Anæmia, but in Case 28 it is not ascertained whether this was so or not.

14. Drowsiness was noted in 6 out of the
I sufficiently reported cases. In Case 24, there was wakefulness from the headache up to 3 days before death, coma setting in 2 days after the last mention of it. In Case 25, the thermometer, I was told, that the day before death the patient was only slightly conscious. Drowsiness, unconsciousness, and coma were all probably caused by similar conditions, and will be considered together.

Unconsciousness was noted in 2 cases, 23 & 25 whereas

Coma was noted in 6 out of the remaining 3 sufficiently reported cases. In one case only (No 28) was there only drowsiness without absolute unconsciousness or coma. In this case the patient recovered. In all the rest the termination was fatal.

This is little doubt that the drowsiness, unconsciousness and coma were each due to the various congestion and stagnation preventing fresh nourishment and oxygen from reaching the superficial cerebral tissues. This was interestingly and plainly demonstrated in my own first case (No 22) where Aeryl Nitrite by dilating the small arteries of the brain allowed of a certain supply of fresh blood, and the consequence was a corresponding increase of activity in the brain. The retention of deleterious & actually poisonous products in the blood by the stagnation of the venous blood would also tend to have a similar effect as the withdrawal of oxygen.
The interesting question arises in connection with this part of the subject, and that is the
much greater frequency of coma or unconsciousness in thrombosis of the Superior Longitudinal
Sinus. Comparing these cases with those of Secondary Thrombosis one is struck with
the contrast. Out of the 21 cases recorded:

- 3 were not sufficiently described (No. 14, 15, 16).
- 1 there was coma with Thrombosis of Right Sinus (No. 12).
- 4 there was coma without.
- 9, 11, 13.
- 5 there was no coma, but there was (No. 5, 13, 19, 20, 21).
- 8 there was no coma and no Thrombosis of Left Sinus.

Of the 4 in which there was coma without thrombosis of the Superior Long Sinus, No. 4 had Arachnitis
and Abscess in the Brain substance, the latter being sufficient to produce coma by pressure; in
No. 9 there were Encephalitis Extensive fibrinoid clot in the Vena Innominate extending into the Descending
Vena Cava; in No. 11 there were abscesses in the lungs (purpura); and in No. 13 there were encephalitis
and recent hemorrhage into the right anterior central lobe.

Of the 6 cases where there was thrombosis of the Superior Long Sinus but no coma, 4 (No. 10, 14,
20, 21) were cases of minor or less inflammation in the Sinus or its neighborhood.

In cases where there was inflammation in or about the neighborhood of the Super. Long. Sinus,
then would be increased activity of the part of the brain on account of the increased blood supply, or it should rather be expressed thus:--the tendency to coma produced by stagnating the blood would be counteracted by the active hyperemia. This would explain the absence of coma in such cases. On the other hand, the stagnation of blood in the pensive or main congestion of central anemia thrombosis has no counteracting circumstance. The only other condition present, namely, the presence more or less of central anemia (of other parts of the brain) would tend in the same direction as the obstructed venous current, that is to starve the tissues.

The reason why coma should be more common in pterygoid thrombosis of the supra-sagittal sinus would be because thrombosis of that sinus affects the circulation more or less over both hemispheres, whereas in secondary thrombosis other sinuses are more frequently occluded and the thrombosis unilateral, so that the venous stagnation is not so extensive.


duration of cases of thrombosis of central sinuses in anemia varies very much, just as do cases without anemia. It may be 205 or 14 days, more.

Diagnosis: These cases are and must yet be very difficult to diagnose. All we can say is that given a case of anemia or chlorosis in which there is headache, disturbed pulse, temperature normal or subnormal with throes or sinuses, unconsciousness or coma we should suspect central thrombosis.
Progressive - This must be extremely grave, unless we can indicate some line of treatment likely to relieve the pathological condition. Cases
of themselves rarely recover. Should they do so a doubt must remain as to the accuracy of the
diagnosis, as there is then no means of proving
the actual formation of a thrombus. In case
of it was believed that there had been phlebitis.
and thrombosis of first one lateral series of them the
other, but this case only be conjectured.

Treatment - Our chief aim must be to
try and restore the circulation in the Cerebrum.
The patient should have the head slightly raised,
to help the return of the blood from the brain, always
avoiding any bending the neck or press upon
the vessels in it. Proceed with plenty of strong
nourishment, and if necessary stimulants. Then,
if the patient is in a semicorsem, must be given
either through a tube of hypodermic tube passed into the
Stomach, or by injection into rectum. I prefer
the former method, as I think the stomach is
taken up the nourishment better than the
wall of the rectum. I should feel inclined
to apply warmth to the head with a view to
decreasing the central circulation. Dr.
Lewis-Sarrett, in his work on Diseases of Infancy
and Childhood (page 358, 4th Edition), advises the
application of cold to the head. We all know
how blue parts that are exposed to cold are liable
to become. Here it seems that the cold stanches
the venous circulation, it brings more superficial
and less active than the arterial. Of course, before applying warmth to the head we must feel satisfied that the symptoms are not due to haemorrhage on or in the brain.

As to drugs, their use must greatly depend upon the individual necessities of each case. I should feel inclined to try a more extended use of inhalations of Nitrite of Amyl. No doubt the rousing from the Coma in many cases was due to dilatation of the small arteries of the brain by paralysing their muscular coats and so allowing pure blood to enter the brain. At the same time as this is used the heart should be especially attended to in case of failure. Still it is difficult to say what permanent good effect is to be gained by the prolonged use of Nitrite of Amyl. Unless the clot in the new allowed blood to flow past it, it might only have the effect of increasing the venous congestion; or possibly it might close the clot on into some other less dangerous part.

The latter effect must be extremely doubtful, seeing the many obstacles in the way of clot travelling through the veins to the heart and thence to the Pulmonary Arteries.

For the Anaemia give Iron, the best form would be the Citrate of Iron & Acammonia. Acammonia to strengthen the Cardiac Contracting Aid ed by Ether (Sulphuric) by the mouth or Intermittently.

Acammonia has been suggested in the general
Treatment of Thrombosis. The hypertensive injection of brandy and digitalis (1200 to 4000 grains) has been used with apparent success in recorded cases of coma in children. It might well be tried in cases such as those under consideration, so as to help the action of the heart. The last question in treatment is: Should we be justified in operating in any way in a case when the diagnosis of the case was clear? In the case of the clear diagnosis of thrombosis in the Sphenoparietal Sinus, I think an operation would be justifiable. I should then try to ascertain, firstly, what region there was most concerned by the focus of the thrombus, possibly the membranous front of the area of the central centres in the convolutions of the brain might excite. Then by care fully trifurcating over the sagittal section, if necessary in 2 places, open the longitudinal Sinus, longitudinally and with a pair of broad flat-bladed forceps attempt to withdraw the clot; close the wound by a pack of dog lint, or if I thought better, with the edge of the wound in the Sinus by means of very fine silk sutures. This seems at somewhat quixotic suggestion at first, but a little careful consideration of some recorded facts will help us to look much more kindly upon such a eminently formidable operation. Trifty in trying to find the position of the clot, percussion over the skull might help

Dr. Alexander Robertson of Glasgow says: 'Whenever, therefore, there is the least ground, judging from the general symptoms, for suspecting that disease may exist superficially within the skull, percussing the head should not be omitted. Failing any result, no harm will have been produced.' Secondly, as to the danger of opening the sinuses, Lassus in his memoir on Wound of the Superior Longitudinal Sinus (vide Memoirs of the Royal Academy of Surgery of France, Translation in the Edinburgh Society's Works, p. 63 to 69) relates 3 cases of recovery, which 'could scarcely have happened if the sinuses had become blocked up with coagula.' Again, Lidell in the same place refers to Pott's Chirurgical Works (Vol. I. p. 196) wherein it is related that Pott bled from the sinuses in one case and stopped the bleeding easily by applying a piece of dry lint with slight pressure. The patient in this case was restored to consciousness, and returned her senses for several days; but a fever coming on, she became delirious and convulsed, and so died on the 12th day after the bleeding.' Lidell refers from the above mentioned to the cases that wound of the Superior Longitudinal Sinus is generally curable, and, therefore that it is not often attended with occlusion of the canal of the sinuses with Coagula. Again, Dr. Chas. H. Hacincde in Ashurst's Internet Encyclopaedia of Surgery (Vol. I. p. 52) says that 'in nearly all cases when wound has been accessible, the application of a little dry lint,'
with the presence of the finger has at once arrested the flow. Also, "later, filled an opening in the Longitudinal Sinus with a branch of cutis ligation, and J. H. Bronston cauterized the lips of a wound of the Lateral Sinus with forceps, and tied it with fine silk." The result of the latter procedure is not stated. Again he says, "although not recommending such a procedure when it is available, the ease with which hemorrhage from these Venous channels can be arrested, proves that, so far as bleeding goes, trephining can be safely performed over the course of either the Longitudinal or the Lateral Sinus." Dr. E. C. Cameron of Glasgow reported a successful case of treatment of wound of the Supra-Longitudinal Sinus by a splinter of fractured bone, he only applied a little glycerin with very slight pressure. He also refers to another successful case of treatment of an accidental wound of the same Sinus mentioned by Dr. Childers in Mr. Le Grand's "Operations in Surgery." Then would be two difficulties to overcome in operating, one the withdrawal of the thrombus without breaking it, and the other the making sure that the whole thrombus had been removed. The first might be overcome by careful manipulation; the second only by a second trephining at some little distance from the first. Such an operation as referred to, should not be thought of, except in the eventually fatal termination to the case. Though, on the other hand, if too long delayed, thrombus will have extended into the smaller lateral Sinus and become the point from
Passing on now from the consideration of Anæmia and Chlorosis in their connection with Central Thrombosis, we come to the remaining five cases of Proceedent Acute or Chronic Disease.

The first is a case of great mental depression and anxiety.

Case 32. (Read before the College of Physicians of Philadelphia, by Dr. R. V. Meigs, Oct. 4th, 1876) — A young woman, aged 20, who had suffered for some time with extreme mental depression and anxiety, was attacked by vomiting and headache, without fever, followed by delirium, partial paralysis of the extremities, and later, chronic movements, sudden prostration, coma, and death.

Post mortem examination showed firm clots in the veins of the carotid and the vein of Salecius, with extensive softening of the neighboring parts of the brain. The veins of the Dura mater and the Circle of Willis were unaffected.

In this case there was no thrombosis found in the sinuses, so-called. But it shows an effect of extreme depression. Is doubt cast on its effect by depressing the heart, acting as a standing circulation. Still one can hardly understand how merely a站着 circulation from such a cause, could have such an effect without the aid of some other factor.

The next four cases are ones of chronic and debilitating diseases, in which it is reasonable to suppose that, besides the merely debilitating effect of the disease in each case, there was probably an attendant condition of the blood itself.

[Note: Philadelphia Med. Press, 1876, p. 1123]
Case 33. - A phthisical child in whom Hemorrhage of the central sinuses occurred, giving rise to delirium 12 days before death. The optic disc was rounded as a gray papilla surrounded as if with ocular vessels, large and pale, choroid and retina pale. The cause of the delirium was sought for in the sinuses of the Dura Mater, and the associated atrophy.
Post mortem.—Tubercles in both lungs. Longitudinal and transverse sinuses completely closed by blood-plugs, of which some were old, white, firm, and numerous, grown to the walls, others still fresh, blackish and soft, and had clearly formed during the last moment of life. The brain and membranes were sound.
Bouchut, in commenting upon the above case remarks that in cases where, after long chronic disease, children die in convulsions, we must look for trouble in the great veins of the brain.
The next case appeared in the Lancet after this commenced this paper.

Case 34. - Reported by Surgeon P. D. Barnard, M.S.
A soldier had ague in Sept. 1868. Five days later there was acute Bronchitis with pain in the left side of the head, and rolling of the eyeballs. Bronchitis was removed from but pain continued, got worse; intolerance of light, but not sound; pupils contracted; temperature 100.4. No loss of power or sensation. No brain overhead. On Oct. 15th the right half of the body partially lost power; he began gradually worse, breathing difficult; pulse weak and frequent; unconsciousness with involuntary contraction of arms & death on Oct. 18th.

Fat Mistake. Iris, think like clarified butter, over left frontal lobe, also underneath it and the middle lobe. Left carotid contained an ante-mortem clot. No disease of ear or nose or brain substance. Congestion of right lung at its base; and a defibrinated clot in the right cavity of the heart. Other organs all healthy. Surgeon Darnaud suggests the thrombosis being caused by the thrombosis, this leads to leakage of serum, which subsequently changes into pus. This certainly best explains the steps of the process otherwise it is impossible to explain the presence of the pus on the surface of the brain, without any disease of the brain substance or its membranes.

Case 35. Dr. Coupland at a meeting of the Pathological Society of London in May 1857 said he had seen one case of thrombosis in the cranial sinuses in Cancer. Particularly not present of this case, but it is quite reasonable to think that thrombosis might occur in such an extremely debilitating disease.

Case 36. Reported by J. M. Corner. A man aged 63, apparently healthy with ossified radius and aneurysms, was admitted to the Dreadnought hospital ship on Feb 15th for chronic dysentery. On March 9th there was acute right pleurisy with effusion. March 11th eyes mild and vacant; slow in answering questions. Delirious in the afternoon. At 8 P.M. convulsed, spitted, breathed sternly, and died hard at 11 P.M.
Post Mortem. 16 hours after. Much subarachnoid effusion, brain oedema, lateral ventricle, brain distended with clear serum. Pericardial fluid increased. Right side head clotted and filled by firm fibrinous coagula. Coagula filled the superior vena cava, Vena anonymis, the subclavians and internal jugular a perfect cast these vessels being formed without a breakage. Similar casts mixed with clot, blood was found in the Lateral and occipital sinuses and the Torcular Herophili. Similar minute fibrinous clots were taken from the pulmonary arteries and their branches to a second division, and in the inferior vena cava a smaller clot and more sanguinaceous coagula. The left auricle, left ventricle, was contracted and contained smaller fibrinous coagula. Abiot of turbid serum in right pleura also much fibrin. Heart otherwise healthy; lungs loaded with serum, remnants of capsular inflammatory exudate pleurae. Membrane of large intestine softened; kidneys in a state of far advanced granular degeneration. Comment. No doubt in this case the disease of the kidneys led to the various pressure effects on brain, lungs, pleura, and pericardium and intestine; and this by depriving the blood of some of its fluid led to thrombosis in the various venous and channels. The case is interesting and instructive on account of the extensive thrombi found. Doubtless the chronic anemia added materially to the effect of the kidney disease, or rather vice versa. Surgeon Corner in commenting upon the above case says that "It is generally
recognised that a chief cause of unusually extensive or firm coagulation is a protracted act of dying, and
refr refr refr refr refr refr refr refr refr refr
hydrophilous coagula in the heart. These coagula are not,
exceptionally, the cause of death (polyphagia of the heart), though
it is very possible in certain instances their presence
may prevent the rallying of patients in extremis, who
ought otherwise have been brought through.
In this case there was superimposed to the trouble
by dehydrating the blood; in the next two cases we
have examples discharges from the bowel as the main
cause of the thrombosis.

Case 37. Under Dr. H. Mallins of Winterton, Norfolk.
A healthy boy, J. office, aged 2 years, was suddenly attacked
with severe diarrhoea in the morning, Dec. 15th 1888; 9 p.m.
About 7 p.m. became cold and drowsy. 1½ hours later
violent convulsions, the legs being struck out with great
violence "almost as strong as a man's"; the eyes rolled
about; head moved quickly from side to side; he was pinching
his teeth loudly; breathing rapid; and quite unconscious.
The body turned jetily cold; the 7 hands and arms
were the seat of patching plump convolution; eyes upturned,
with pupils dilated and insensitive to light; pulse in-
perceptible; occasionally feeble convulsive movement
of right arm; and death soon afterwards.
The family had recently quitted the workhouse, and
the child was not well nourished. The only assignable
cause for the diarrhoea was a suddenly developed
严重的 cold of the atmosphere "acting on an ill-nourished
system. 2 other members of the family had diarrhoea soon after
M.ted by Dr. Mallins before the Norfolk Norwich Med-Chirurgical Society, Feb. 7th 1889.
Post Mortem. Dura Mater Othematia Superior Longitudinal Sinus and the Veniæ enetantæ it naturally were distended with well-formed pale clot and a small quantity of very dark fluid blood. The clot extended the whole length of the Sinus and into the Lateral, straight, occipital, Superior and inferior Petrovæ sinuses, all of which were distended. The brain itself was healthy, with the exception of a small but well-marked extravasation in the right lobe of the cerebellum. All other organs healthy, but right side of heart much distended, locking ready to burst.

Case 38. An infant - 7 months old was admitted into the New York Foundling Hospital in Dec. 1877. The pupils reacted freely to light, head turned from side to side constantly. Body much wasted, eyes sunken, anterior fontanelle depressed. The child died evidently after intestinal Catarrh.

Post Mortem, all the lesions of severe protracted catarrh present. In examining about an aqueous humour between the Dura mater and Brain, anterior part of brain (uppermost part) normal. Venæ Petrosalminus in posterior part engorged with dark blood. Cranial Sinuses distended with dark blood clot, a long white clot was drawn out of the Longitudinal Sinus, being firm & evidently acute mortem. There was no inflammation about the brain or sinuses. Both of these cases (37 & 38) and also Case 38 in part, illustrated at one of the causes of rapid death from diarrhoea, especially in young children.

Schenck’s Diseases of Infancy and Childhood (14th Ed) p. 144.
Drach in his monograph (p. 114) refers to
Gerhardt's explanation of the pathology and symptoms
in such cases, which is very simple and natural.
In case 37 the extreme dilatation of the right side
of the heart at first suggests the cause of the
thrombus is to be some obstruction to the circulation
in the lungs. And it is not unlikely that this
may be some extent true; for the viscidification
of the blood would make it very difficult to pass
through the pulmonary capillaries, and would
thus retard the general circulation.
Did we but examine after death from brainhaemorrhage
all the skulls and their contents in
the many children that die every year, especially
in the hot weather, I am sure we should find
a very great number exhibiting thrombosis
of the Central Sinuses. Though great care
must be taken to exclude all cases in which
an old clot may have formed after death. Of
course, the sooner after death the post-mortem
examination is made in these cases the better;
as it would give less time for the various
elements of the blood to separate and the clot
to be thereby decolorised.
Cases where much serum is thrown out
on the surface of the brain from venous
congestion may be sometimes mistaken for
hydrocephalus. In fact they have occasionally
been called false or spurious hydrocephalus.
A diminished quantity of blood is one of the
general causes of retardation of the circulation.
leading to Central Thrombosis. The two things that diminished the quantity of the blood are (a) loss of blood, and (b) profuse sweating. Cases 37 & 38 have been examples of the latter class.

The next case, which is closest under impetus into the expansion of lungs, might also be considered as belonging to past to class (a), mentioned above, ascertained bleeding by leeches was thought to have played a part in the cause of Thrombosis.

Case 39. J. Stowe, aged 20 months, always very pallid, used to burst at people with head. Had pain in head, head hot, no convulsions at first. Leeches were applied to temples and again behind ears; also blisters. Cough, sneezing on 17th day, convulsions; fever; eyes drawn to one corner; stiffness of hands; pupils rather contracted; pulse very quick. Then cough became bad, bowels acted well. On 24th face convulsed, pulse 120, weak; respiration 60; convulsions, grand drip, teeth, pupils contracted,acting badly to light. Died at 11 p.m.

Post Mortem. All the larger veins over both hemispheres were quite filled with yellow Coagulum; whole interior under the membranes covered with dark extravasated blood. Superior Longitudinal Sinuses fully clot, formed at the back, and clearly attached to the month of the vein. Sunken up brain pales; veins are engorged in substance of both hemispheres. On the outside of the right anterior
lobe was a patch completely softened. Several strakes of clear fluid in the ventricles, and an ounce and a half of serum about the base of the brain. Left lung hepaticized throughout; 12 uniform fluid in left bronchus, also left pleurisy. Liver rather pale and with a few superficial yellow patches.

Comment. As there is no mention of any pyogenic abscesses in the lungs in connection with the pneumonic state, and as the cough is related quite early in the case, it is evident that the pneumonic (and secondarily pleurisy) was the forerunner of the thrombosis, and apparently the cause of it. The hemorrhage over the brain must have been recent, and very probably a result of the venous pooling. The condition of the left lung was quite sufficient to have so obstructed the venous circulation as to have set up the thrombosis. For the age and strength of the child the bleeding by itself was rather excessive, and it may fairly be questioned whether that was not enough to have helped in the causation of the thrombosis. The bleeding in this case would not be able to stimulate or accelerate the circulation but a very little, if at all; at the same time if too much, it would partly empty the venous channels and so tend towards increasing the blood stasis.

The next 5 cases are further instances of obstruction to the circulation occurring in the respiratory organs. All are cases of Whooping cough. The first is so closely allied to the last case related (No. 31) that it is placed first.
Case 40. is reported by Bouchut in the Journal
Kild. 1868. A little girl had suffered for several
months from Whooping cough. Broncho-pneumonia
ensued; in complete anesthesia attended, quickly
emerging in a condition resembling asphyxia. Now
Convulsion lasting 4 hours set in, were repeated
twice in the same clay, and led to death.
Post Mortem. Considerable brain congestion, and
a little serum effusion in perier and under
the arachnoid. Besides this, old coagula were found
hard and colorless; one 15 centimeters long extending
along the lateral sinus, to the commencement of the
jugal vein; the other, harder and paler in the
Sulcus Longus Sinus, had grown to the wall of the sinus,
and completely obliterated it. No mention is made
of the lateral veins in the writer, nor of any other
part.

Comment. In this case, as in 40.39, the broncho-
pneumonia played the principal part in
causing the thrombosis. It is very doubtful
that the Convulsion or actual Pertussis
had any effect in the causation. More probably
the Convulsion assulted from the complete
plugging of the Superior Longitudinal Sinus.
This is not the view held by Dr Lewis Smith &
in connection with the next few cases.

Cases 41, 42, 43, 44. are four cases referred to by Dr Lewis Smith (pp. 354, 355) the symptoms, were alike apparently
due to the thrombosis, venous central congestion.

These were cases of pertussis with chronic convulsions in connexion with a severe spasmodic cough. Dr. Smith says also the convulsions which occurred in both (from the context I presume he means to refer to all the five) cases were apparently a cause and not a result of the thrombosis. If this were really the case, we should have to consider the convulsions in other cases of Central Thrombosis as the cause or one of the causes of the thrombosis. Though I do not admit that violent convulsions may have the effect of so interrupting the circulation, yet in many other cases noted, the thrombi found after death had all the appearance of having been formed prior to the commencement of the convulsions. In those cases in which the convulsions did not come on until a few hours before death, as in Case 40, here the convulsions only set in on the day of death, and from the hardness, pallor, and ashen appearance of the thrombi, also the severe effusion on the surface of the brain, the thrombosis must have commenced before then. Briefly, I put it thus—there must have been first a waste of affection of the lungs, (in some cases from pneumatic consolidation in others from spasmodic cough of pertussis) which obstructed the circulation already feeble from acute (acinar pneumonia) or chronic (as in pertussis of long standing) disease, this led to the thrombosis; this in its turn caused visceral and arterial congestion provoking convulsions, then either a severe spasmodic coughing or pertussis or...
a bad attack of convulsions, so further impeded the respiratory movements, as to prevent the right side of the heart from emptying itself, and sudden death was the result.

Although not actually a part of the subject of Central Thrombosis, I should like to connect with this branch of the subject to suggest that when we are called in to see a case of sudden violent convulsions leading to death (apparently artificial respiration and stimulant hypodermic metal or by oesophageal tube) should be tried if the apparent death has not occurred more than a few minutes before our arrival. Also, these means of resuscitation should be persisted in for an hour, if necessary. Many cases of sudden death from convulsions are due to the interference with the respiratory movements, preventing the right side of the heart from emptying itself. (Vide Case 31, when the right side of the heart was "ready to burst." If then, we could relieve that condition by getting the lungs to expand, and stimulating the heart, the latter might recover itself. I can recall to my recollection 2 cases in my own practice within 2 years that were in each of which the child had apparently died from convulsions about 20 minutes before my arrival. In one case I performed artificial respiration and plunged the child into hot and cold water alternately and had the pleasure of seeing the child revive in about 20 minutes.
In the other case I could not get any last water, so I suspected some brandy under the skin and performed artificial respiration as in the other first case; the child began to revive in about 35 minutes. Unfortunately some other convulsions occurring 2 days later when I was out proved fatal. Both children were absolutely paleless, no sound whatever being heard by the Stethoscope.

The next three cases come under and illustrate more or less the effects of disease in the walls of the sinuses in causing Primary Thrombosis.

Case 45. A lady at Strathmore suffered from intense pain in the head and sickness. She had no marked symptoms indicative of any cerebral disease, and the case was regarded as functional. She however died, and there was found extensive congestion and thickening of the sinuses in connection (as poissaid) with ossification of the dura mater.

In this case it appears that not only were the walls of the sinuses so prone to abnormal rigidity and also the molecular attraction between them and the blood would be altered by the changed condition of the tissues constituting the walls, so that in all probability in their changed state they would act somewhat as foreign bodies,
Carcinoma fibrosum elementis (or rather the fibrin, forming elements) to the culture and from adherent cells.


Post Mortem. 41 hours after death. General Droopy. Skull caps normal. A small old adherent thorn in the middle of the Superior longitudinal fissure, another in the vesicular hemisphere. Pit under over whole right frontal and opaque, much more crumpled over the left side. The left hemisphere much atrophied, the right perhaps a little so.

No further description of condition of brain or amyotrophy. The case is put down as one of.
Sclerosis of Cerephalon. — Talented Syphilis.
In this case I state it as the chief but not the
only, cause of thrombosis was the altered state
of the PM matter, involving the serious error or
less. The atrophy of the left hemisphere would
have aided this. But further, the state of the urine
showed some Kidney trouble, pointed to by the
American as well. So that it is probable that
this also was another factor in the causation
by impeding the circulation and enforcing
the heart more.

Case 47. Reported by Dr. M. Church.
Mary Anne P — aged 16 admitted March 27th.
Died March 30th. Had Typhoia (?) for 3 weeks.
March 26th severe pain in head, and very sick.
This continued & convulsions with screaming set
in next day 28th quiter. 29th became resemble &
comatose, pulse 60, temperature 102.0, pupils con-
tracted grays directed to the right. Face amuck
flushed. Left leg rigid arreted, right leg
arm flexed. Vessels of retina a little slack.
next day less flushed, pupils contracted less. 4
Death. Without any convulsion at 5 p.m.
Ht 4 feet 11. Female organs ill developed.
Expulsion of Smaller vessels of brain matter. A large
recent clot in the Cap. Longitudinal Sinus, yellowed
above & tough, passing into loose clots in the
torcular Heosphi. Congestion of convolutional gular.

Cloth in all the veins of the body, contained some tough yellow fibrous, others loose & solid. Viscous Baleni & straight veins filled with firm yellow continuous clot. "And certainly put motion" hemorrhage frequently angina engorged. Arteries at the base filled with yellowish fibrous clots similar to those in the veins & sinuses. In the right auricle & ventricle a similar clot continued with one in the Pulmonary Artery. Pneumonic patches in the lungs. Liver intensely engorged, left lobe adherent to diaphragm & spleen gall bladder filled with thick tenacious bile. Nothing else very marked found in the body. In this case we have to take into consideration two main factors, the inflammation of the brain & cone of nerves, and the evidently particular condition of the blood, tending to lead to extensive thrombosis. The latter (condition of blood) may well be put down as an excessive proportion of fibrin consequent upon the inflammation. So that the inflammation was the original cause of the thrombosis, partly by the altered molecular attraction of the walls of the veins and sinuses, and partly by the altered state of the blood. The arterial thrombi no doubt extended backward from the capillaries, aided greatly by the state of the blood. The coagula in the head and pulmonary arteries would also start from the various thrombi higher up, either by centrifugation.
prolongation, or by a portion of half-formed clot breaking away from a thrombus in the Ductus venosus or in the portal vessels and lodging either in the heart or in the Pulmonary artery and spreading from hence.

In Case 18 (page 278) we had a phlebitis of the Inferior longitudinal Sinuses, very much allied to the above case in many points, though not in outline. The most general mention of inflammation in Case 18, made one class it when it is, though, on further consideration of the case, it now seems true that its most proper place should be among the cases of Primary Thrombosis.

Before taking the 10 cases with unassigned positions in the classification, I must refer to Case 10. A case mentioned by Dr. Ord before the Pathological Society of London. It was seen by him, and was a case of thrombus of the cranial Sinuses in acute Rheumatism, in which phlebitis developed in the neck and arm on one side. There was hemorrhage into the brain. Dr. Ord said that the changes in the condition of the lining membrane of the neck was perhaps operative in the evacuation. In the absence of further data, we cannot say much with regard to the case, beyond the fact that it appears to belong to the class of cases such as No. 254534.
Of the 10 remaining cases that I have collected,... there was no actual thrombosis of a cranial sinus ascertained, but only of a cerebral vein following parturition.

2. Thrombosis was only suspected, as both patients recovered; in both also the probable cause was doubtful.

4. The cause of the thrombosis (proved to have been present by post-mortem examination) was doubtful; in most of the cases there are not sufficient data, upon which to found an opinion.

It shall take the cases, according to the above order, commenting, where necessary or possible, upon each.

**Case 43.** Dr. Forgan noted notes as a case before the Leeds & West Riding Medical Chirurgical Society.

It was the case of a woman, who, after a normal labour, was seized on the ninth day with pain in the right side of the head and numbness in the first 3 fingers of the left hand. This was followed by incomplete paralyzing of the left side, with transient ophthalmia. She gradually became emaciated, and the loss of power more marked. After death a clot was found in the auricle of the right atrium; more in cory of the sinuses."

Comments: In this case the strong presumption is that the thrombosis was in some way connected with the labour, probably through Pneumonia. Dr. Bristowe (in his Reynolds’s System of Medicine, Vol. V, p. 416) says that Pneumonia is peculiarly apt to supercede in the periphrisal state. Playfair gives the three following reasons for thrombus occurring:

1. Excess of fibrin in the blood; 2. Presence of a quantity of effete material in the blood from resolution of the uterus; 3. Losses of blood (or excessive discharges might be added to this last one).

As there is no mention of any thrombosis elsewhere beyond the middle central vein, there must have been some local exciting cause. The positive aspect of the thrombosis suggests an embolus in the middle central artery having started the thrombosis. That artery is the one most frequently affected by embolisms. And in the cerebral state a small piece of clot from elsewhere (femoral or iliac vein for instance) might have forced its way into the middle central artery, and yet have been so small as to have been overlooked.

Case 50. Dr. O'Hara related a case before the American Medical Association in the early part of 1882. It was one of supposed thrombosis of one or more of the cerebral sinuses, followed by recovery. The patient, a girl, had intense headache, ordinaffaire, slight ptosis, ecchymoses of the conjunctiva, slight left anæsthesia and right hyperæsthesia; also some paralysis of left side of body and slight deafness on left side. The mind was clear. She was treated with mercury and iodide of potassium, with purgatives; and recovered in 3 weeks. The chief point in her symptoms was paresis of the facial, postaural, hemi-nigral, pains, conjunctival ecchymosis, and paralysis of the eighth nerve.

Vide London Medical Record, 1882, p. 276.
Comment. Dr. Olmara thought it probable that there had been thrombosis of the right cavernous sinus which had spread through the circulaxons to the left cavernous sinus. There was no injury nor inflammation of the ear, nor any vesicles to account for the thrombosis of such large vessels. Dr. Olmara thought the cause was specific. No doubt his reason for so thinking was on account of the recovery when put on mercuric and iodide. It is possible it may have been specific, as iodide of potassium by itself is capable of greatly reducing thickening or deposit as might and would have occurred in case of ordinary phlebitis.

Case 51. One case is referred to by Murr. It was recorded by Voorman (in the Deutsch. Wochenschrift, 1836, 1892). A child 5 months old had paralysis of tongue, right limbs, rigidity of neck, and opisthotone, with clauternation of the left temporal vein and oedema of the scalp. The child recovered with some permanent damage to the left hemisphere.

Comment. The cause here is not indicated in any way. But, as recovery took place in both cases, 5 and 29, there is no any satisfactory evidence that thrombosis actually existed. The data in Case 57 are as few, that it would not be safe to draw any definite conclusion. In Case 28 (related on page 43) although there was recovery there were more suggestive symptoms, but no positive proof.

We must be careful not to reject it into all cases of suspected thrombosis which was removed from. In many cases—e.g. in phlebitis—there is ample evidence to show that the thrombus commences at one side of the sinus. And it is quite conceivable that the process might be checked, and the thrombus, not yet having filled the sinus, could integrate with the debris be carried on by the blood stream. Or under the acting of drugs or a marked assimilative or absorbing power, the clot might become partly absorbed, the more movable elements passing through the wall of the vein or sinus, while the fibrinous elements contract and flatten down against the wall, or get washed down smooth again by the current of blood constantly flowing over it.

Case 52. M. Remant made note of a case before the Med-Chirurgical Society of Paris in 1862. It amounted to phlebitis, and was called "Spontaneous Thrombosis". A girl aged 18, had been dead six weeks. Her temperature rose to 104°. Soon afterwards she was attacked in the right side, with phlegraphia, and all the symptoms of pulmonary consumption in the lower lobe of the left lung. These symptoms soon disappeared. On the 30th day, there was phlegmation of the right leg. By the 45th day she had all the symptoms of tubercular meningitis in present.
Post mortem there was complete thrombosis of the Superior Sagittal Sinus, and of the right transverse sinus, also thrombosis of the few branches of the pulmonary artery from which a haemostatic suture had proceeded to the pulmonary vein when induration had been diagnosed. There were a few tubercles in the apical one lung. No cause for the thrombosis was found. Nor could any sign of pneumonia, either old or recent, be found.

The heart was healthy.

Comment. No doubt then was a general condition of the blood probably tending to thrombosis, as shown by thrombi forming in the transverse vein and the cranial sinuses. The coagulation in the lungs must have been started by an embolus from one of the other thrombi.

Case 53. by Dr. Compland, in Middlesex Hospital.

A man aged 31. Single. Had had Bronchitis and Rheumatism in the winter for many years. He was "said to be temperate". The illness lasted only 24 hours. There was first heavy sleep with violent hicouph. Then delirium, wandering about apparently awake. He was put to bed; breathing became 80 times in 4 minutes with constant hicouph and yawning. Breaths smell strongly of Alkohol. The patient was silent when spoken to, and resembled an inarticulate answer. He said he had no pain. There was no reflex action of right leg diminished. Power and sensation in right arm vide Med. Times Gazette, 1881, vol ii, p 574.
also decreased sensation of right side of face. Right pupil contracted slightly, left dilated & does not at all. Tongue but strong to the right side. Abundant mucous mucus heard all over front of chest. Heart sounds normal, Liver healthy.

Post Mortem: At the base of the left lung there were some necrotic areas, also fibrous adhesions at the apex. The right auricle contained a fibrous clot which extended into the right ventricle and hence into the pulmonary arteries. Superior and inferior vena cava also contained post-mortem clot and fluid blood. Slight atresia of aortic valves. A quantity of slightly frothy turbid yellow fluid in trachea and bronchi; the mucus membrane of latter injected. Left lung emphysematous under face, or sertum deeply congested and edematous, with capillary extravasation in the upper lobe. Right lung the same. Spleen kidneys congested. Congestion in stomach, pancreas, suprarep and jejunum. Liver very congested. In the cranium, Durra Mater congested, no adhesions between dura mater brain except along the margins of the longitudinal sinuses. Pia mater injected everywhere. Clots partly fibrinous and partly dark and recent was found in the straight, both lateral, right cavernous sinuses, and right sylvean vein. Extensive haemorrhage about the base of the brain involving the substance of the force and crura. No atheroma vessels.
Comment. As no other cause could be found for the thrombosis, it strongly suggests the rapid onset of acute bronchitis with pneumonia and central congestion, probably due to some exposure to cold when under the influence of drink. The extremely congested state of the vessels would lead to stagnation and thrombosis, and then to extravasation. This last occurring in the pons or other important parts leads to the rapid death. In a case such as this one wonders that resuscitation was not freely used. The pulse had been slow—only 60, suggestive of difficulties in its flow, or a congested nervous system denying the cardiac ganglia their proper supply of arterial blood.

Case 54. A case of cerebral hemorrhage following thrombosis of the sinuses. The patient was a boy aged 5, who was ill 10 days before he died. Headache, vomiting, constipation were the chief symptoms. The breathing was stertorous for 30 or 40 hours before death. There was no pus nor discharge from the ears, nor other sign of scarlet fever; also no sign of any injury to account for it.

Post Mortem. The superior longitudinal and both lateral sinuses contained adherent decolourised clots; also the superficial central veins were firm and thrombosed. There were numerous hemorrhages on the surface of both hemispheres; two of them in the left ascending mediastinal path. Dr. J. London by Dr. Balfour—2d Sc., 1832.
panaretal convolution, and in the right temporal sphenoidal lobe - being very extensive and deep.

No cause for the thrombosis could be found; and the hemorrhage was considered to have been due to nervous tension from the thrombosis.

The data here are not sufficient to found any conclusion as to causation; and I am sorry I have not been able to get a view of the transactions of the Pathological Society of about that time, so as to have a fuller account of the case. Still the case is instructive as it stands; showing, as it does, the further danger from hemorrhage due to thrombosis. In this point the case resembles Case 53 though the hemorrhage was in a different part of the cranium. Hemorrhagic extravasations are the greater unless such form very commonly in nearly all cases of marked central thrombosis.

Case 55. Dr. Cripps described a case in Vol. VII of the Transactions of the Pathological Society of London.

April, aged 16. There was sudden seizure with pain in head, vomiting, mental confusion, and other central symptoms; this was followed by right hemiplegia, loss of speech and inability to protrude the tongue. The pain in the head was referred to the left side, to the temporal, post auricular and occipital regions, also down the neck on that side. Perception of external objects existed, expressed by signs. Pulse normal, respirations slow; turn frequent; vomiting morning.

The power of speech and the limbs returned before referred to by Dr. Willks (in his Dis. of Nervous System, p. 180).
death, which happened rather quickly. The symptoms had lasted about 14 days.

Post Mortem. Superior Longitudinal Sinus was filled with coagulated blood mixed with purulent fibrin adherent to the walls. Superior Central Centennial Sinus filled with pale coloured fibrin. The substance of the brain was healthy, with no abnormal vasculosity. There was no anaemia, and no apparent cause for the thrombosis was found.

Note - In this case there does not seem to have been the haemorrhages spoken of in the two former and other cases.

Case 56. Dr. Burrow described a case of thrombosis of the superior longitudinal, transverse and cavernous sinuses and of the ophthalmic vein. The patient had had central symptoms. This case was referred to in the Lancet, 1880, Vol. 2, p. 1924; and unfortunately I have not been able to obtain a copy of the particular volume of Harkness's Abstracts, which contains the description of the case.

Case 57. A case is referred to in a leading article in the Lancet (1885, Vol. II, p. 441) upon 'Pneumocœlia.' The case was one reported by Colliw, in which the axillary vein became plugged accidentally to thrombosis of the superior longitudinal sinuses, and concomitantly with embolism of the pulmonary artery.

Comment. In this case there was no Harkness's Abstracts 1889, Vol. I, p. 9.
Thrombosis of the Subclavian Vein connecting a continuous thrombus from the Superior Longitudinal Sinus with that in the Axillary Vein or there was some occasional venous connection between the Sinus and the Axillary Vein, it can hardly be conceived possible that the Axillary clot was secondary to that in the Crural Sinus. As Keckhiasenau points out, it was almost certainly a case of multiple thrombosis of which none other illustration occurs in this paper. The following is another illustration of it:

Case 58. Under the care of Mr Macbain, of St. Thomas's Hospital, London in 1886. An anemic girl had thrombosis first of the left Subclavian Vein, then of the right limb, which extended into the Inferior Vena Cava. On the 17th day there were symptoms of central thrombosis (fumes), much head ache, vomiting, drowsiness, and some twitching of the arms. 2 days later there was coma.

Remarks: This is probably a case similar to some of those related in this paper as due to hemorrhia. But, not having been able to get a copy of the particular Volume of the Hospital Reports in which it is related, I have not thought it advisable to include it under in that portion of my paper, with so few data before me.

This concludes the relation of cases as

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...on page 114. They have been collected from various English sources whenever obtainable, and not with a view to favor one particular theory. The conclusions to be drawn from these cases related in this paper, from a consideration of some of their details are:

1. That thrombosis of the central arteries is liable to occur in the course of many and various diseases.

2. That it may be caused by anaemia or chlorosis.

3. That the symptoms are not uniform to any marked extent; this want of uniformity being in great measure due to the symptoms of the thrombosis itself being, in most cases, masked by other symptoms also belonging either to the disease giving rise to it, or to secondary effects of the thrombosis (hemorrhagic extravasations, pyemia, &c.) which also vary a great deal.

4. The most frequent symptoms are pain in the head, sometimes referred to the situation or neighborhood of the disease, and losses of the sensorium or less loss of consciousness, memory, or motor power. In cases of inflammatory origin, pyemic symptoms abound. These cases also seem to suggest that loss of central functions is more frequently found in non-inflammatory cases, while excited or exaggerated functions are more frequent in cases due to inflammation.

I should have gone into the consideration of thrombosis of the central arteries as a cause...
of death, firstly as one of a series of steps in a pathological ladder (as in tetanus, followed by phrenia) and secondly as a last step (as in ascemia, charbon in infants &c.). But this paper has become so much longer than originally intended, that I must leave that part of the subject.

It is evident that we have a vast deal more to learn in connection with Central Thrombosis and only patient and careful observations of well reported cases will enable us to diagnose and treat this disease more satisfactorily. No doubt we should be able to form better ideas as to diagnosis if we could collect a few better observed and better reported cases. Clinical observations are frequently poor chiefly because we do not suspect that there is anything new or interesting in the case, and so, in consequence, many valuable points are omitted in the report as unimportant. Whereas, in fact, every single case has its points of interest from which we might learn much if we only took the trouble to observe and note.

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