ProQuest Number: 13906908

All rights reserved

INFORMATION TO ALL USERS
The quality of this reproduction is dependent upon the quality of the copy submitted.

In the unlikely event that the author did not send a complete manuscript
and there are missing pages, these will be noted. Also, if material had to be removed,
a note will indicate the deletion.

ProQuest 13906908

Published by ProQuest LLC (2019). Copyright of the Dissertation is held by the Author.

All rights reserved.
This work is protected against unauthorized copying under Title 17, United States Code
Microform Edition © ProQuest LLC.

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

Puerperal Embolism & Puerperal Thrombosis

with special reference to D Playfair's [illegible]

[Midwifery 2nd ed page 336]

"Without denying that there is considerable force in
"the arguments adduced, I think that the clinical
"history of cases strongly favours the view of
"spontaneous coagulation".

My own case.

Mrs J., aged 28 years, a large, well developed, woman, was delivered by forceps of her first child on Sep. 4th 1893. There was no rupture of the perinæum beyond that which is usual, and no unnatural hæmorrhage followed or preceded delivery.

The nurse thought that throughout the case there was rather less lochial discharge than usual. It was never particularly offensive.

She seemed for some days to be making a

2.

rapid and excellent recovery.

Sep 11th 08. She suffered from headache and felt a little out of sorts.

Was prescribed for under the impression that she ailed from dyspepsia.

She sat up in her bedroom, feeding the baby from the breast, and was seemingly quite well with the exception of a little weakness.

On [illegible] 15th, [illegible] 16th, & [illegible] 17th, she walked downstairs & there spent the day.

However, before she came down stairs, she had complained to her friends of pains in the lower part of the belly.

[illegible] 18th, she complained of being out of sorts, and her friends considered she had "caught cold" in consequence of coming down stairs from her bedroom.

She had, she described, something like neuralgic pains in the head. First on one side of the forehead and then on the other. The pain

lastly particularly affected the right eye, causing dimness of vision. This night, she was "somewhat rambling in her head" whilst sitting up in her chair. And, during the afternoon, a pain had struck in the right side of her chest and gradually got worse. There was also some cough, which caused pain in the chest-wall. I visited her, and considered, from the symptoms, that there was some pleurisy. Her temperature was slightly over 100°F.

11th p.m. On this day, Tuesday, the symptoms led me to diagnose the presence of some pneumonia, the right side. T. 101°F. some dyspnoea and accelerated Respiration. Cough troublesome. The child was weaned. P.V. Examination gave no result.

2 p.m. She was worse. T. 102. There was expectorated a frothy mucous sputum tinged with blood.

There was an increased amount of pain present in the right side.

6.

On Sept 21st, 1[illegible] 23 days after delivery, at night she had gone to sleep; but suddenly awoke, at about 11 pm, in a dreadful state of dyspnœa. She thought every minute would be her last. Syncope seemed imminent. I found her in bed propped up high by many pillows. The surface of her body was cold and clammy. She could only whisper, & that with difficulty; though all her mental faculties were more acute than usual. Her lips and face were cyanotic. Cold sweat poured down her face. Her distress was exceedingly painful to behold. The pulse was exceedingly small, and almost too quick to count. T. 101°F. The expectoration was bloody, stringy, mucus with some froth. There was marked dulness at the base of the right lung.

8.

In the centre of the dulness, & below, there was diminution of breath sounds but- around it there was tubular breathing. Dr. B. saw the patient with me and pronounced that there was some pneumonia, but also some fluid below it. By the aid of strong stimulants the patient gradually rallied, but for several days the prominent symptoms were still present and very gradually disappeared. She was unable to lie down in bed for some days. The cold sweats and the cyanosis of the lips, continued for three or 4 days. Ever since the expectoration had been tinged blood (Sep 20th), this had been a very marked symptom. There was always more blood, bright red, than it is usual to have in a case of pneumonia. In fact I had darkly shadowed a subsequent phthisis pulmonalis. And on the night of

the critical attack it was mooted whether or not ergot should not be added to her mixture. The stimulants used to her were Ether, Digitalis, Strychnine &c. It was the indefiniteness of the pleuritic and pneumonic symptoms, and the presence of the abnormal amount of blood in the expectoration, that, on this night, compelled me to come to the conclusion that I was dealing with a case of pulmonary Embolism; the critical attack of dyspnoea, of course, strengthening me in my opinion.

There was no trace of tuberculosis in her family history, and she had never had a day's illness in her life.

The dulness at the back refused to clear up for a very long time. But the Tubular

breathing disappered in a weak and the T came down to normal. The tubular breathing had vanished, but there was an almost total absence of breath sounds at the base of the right lung.

Oct. She was well enough to be put on tonics.
Phlegmasia dolens attacked right leg and thigh. The leg swelled up, and there was great pain. The limb was as useless as a log of wood.
Between this date and Oct 11th this leg got almost well. And the Expectoration, a slight cough being still present, was nearly, if not quite, free from blood stain.
Oct 12th She was removed in a cab to another house, about 100 yards distant.
Oct 13th She sat up in a chair in her bedroom for the first time, since the relapse, and immediately a pain shot into the left-

calf.

She was unable to rise from her bed, the leg being very painful and swollen tense.

The leg is still oedematous, but not very painful. The expectoration is quite free from blood. She has plenty of flesh on her.

In fact, there has been, from the first, comparatively little loss of flesh.

129 There is still some dulness at the base of the right lung with somewhat diminished breath sounds. The left lower limb is not quite well.

She is downstairs and walks about without much difficulty, tho, by night both legs are rather swollen. She is much benefited now by wearing elastic bandages. Her general health is good. The lung dulness has gone,

16.

and the breathing is little if any, inferior in power on the right to the left side.

She walked down to our Surgery and is seemingly quite well in every respect.

Science and Practice of Medicine by Dr Aitken vol ii. p 105.

× Dr Aitken gives the following as symptoms × of Pulmonary apoplexy. ×

(1) a sudden difficulty of breathing threatening suffocation.
(2.) Expectoration of blood.
(3) Mucous rhonci.
(4) Cough
(5) inability to lie down.
(6) Dulness of the lung – a sign of circumscribed condensation of the lung, not infrequently followed by signs of pneumonia or of pleurisy. If the patient recover there is always some pneumonia.

7. Cyanosis
8. pulse small and frequent.

Dr Aitken says – apoplexy of the lung may be distinguished from hæmoptysis by dulness on percussion, by tubular breathing and by subsequent fever and pneumonia.

Dr Taylor says "their (infarcts) occurrence often gives rise to hæmoptysis, and if of large size there may be dulness and deficient respiratory murmur at the surface of the chest corresponding to them (the infarctions).

Now, in my case we first notice that she complained (p.3 l 12) of pains in the lower part of the bowells. This might have been caused by a thrombic condition of the uterine veins and those in connection with them. Cruveilhier in the Journal of Anatomy and Pathology records a case of death from Pulmonary Embolism

" in which pm. it was found that "the
" uterine, ovarian, and hypogastric veins were
" like hard cords with compact adherent clots,

On the 15th, 16th, and 17th she came downstairs and her subsequent malaise is attributed by the friends to "catching cold".
Then we notice that on the 17th, after the disturbance caused by the exertion and jolting movement of descending a steep set of cottage stairs, she suffered from neuralgic pains in the head, and that there was pain, very severe, over the right eye with loss of vision. Then there is the pleuritic kind of pain in the right side, with slight rise of temperature. "The rambling in the head" as the friends described it.
Now is it too much to presume that these symptoms were caused by a small shower of minute emboli perhaps caused

by the canalisation of soft pappy central parts of a thrombosed vein in the uterine regions! And might we not take it that the pleuritic and pneumonic symptoms set up were due to the irritation of these minute emboli? On the 19th and 20th I thought there were signs of pneumonia; and there was the blood stained sputum of the 20th, gradually increasing in intensity. As I said before, the abnormal amount of blood expectorated first set me thinking that there was something more present in the case than an ordinary pneumonia; and the very fact that an infarction evidenced itself by the dulness and the sanguineous sputum, indicating considerable diapedesis, would point rather in favour of the idea of this shower of minute emboli. Of course it might be said that this was only a case of

pleurisy and pneumonia and that the
subsequent slower attack of pulmonary apoplexy
was largely brought about by the obstruction
caused to the circulation by this condition
of the lungs. But the pleurisy would
be also an argument in favour of an
early infarction, for we know that this
is more liable to occur near the surface
of the lung where one side of the tissue
is covered by pleura

Prof Coats' Pathology p. 37

Now we come to the sudden attack
in force, on the 26th.
In this case the patient was in bed and
awoke suddenly to find her life in seeming
imminent peril. We notice the absence
of sudden exertion, unless, as is probable,
she awoke in a fright and sat up
suddenly in bed. Still there is no history
of exertion. We have to account for
some sudden interference with the
respiration.

Dr Finlayson in his Clinical Manual writes
" this element of suddenness is of great
" importance in leading to dyspnoea, for
" if the breathing surface be cut off
" gradually (e.g. pleuritic effusion) the
" respiration & the system may have
" time to adopt themselves to the altered
" condition, hence there may be extensive
" thrombosis of the pulmonary artery without
" any alarming dyspnoea until perhaps
" a fatal displacement of some clots
" takes place".

Therefore we must come to the conclusion that a large embolism suddenly was floated into the pulmonary arteries causing this critical attack.

It may have come from the process of canalisation of the thrombosed vein in continuation of the process before suggested.

Dr Playfair [illegible] says
in 7 out of these 25 cases there was distinct
evidence of true embolism, & in these the
death occurred at a remote period after
delivery; in none before the 19th day.
In my case this attack occurred on the 21st
day after delivery.
I read in a Lancet 2/67 that a Dr Ball
advocated the view that the spontaneous
thrombosis (of which he was an upholder)
could only begin in the peripheral vessels
and creep backwards.
But in my case supposing there had
been a primary shower of small emboli,
each of these would be a centre for subsequent
thrombosis, and this going on steadily for
some length of time then the patient might
wake from sleep and assuming the erect
position, discover that some great
impediment to the oxygenation of her blood

had taken place, and that the balance of Respiration and circulation was critically disturbed.

Now suppose we take it that the cause of the dyspnoea was

either (1) Embolism.

(2) Thrombosis.

1-A.

Embolism from a central thrombus i.e either in the heart or larger divisions of the pulmonary artery

1.B Embolism from a peripheral thrombus i.e in the uterine or other peripheral veins.

2-A. Thrombosis from alterations in the blood itself e.g. in the leucocytes

2.B. Thrombosis from alterations in the vessel wall, i.e destruction of the vital force of the endothelium.

B.

how the symptoms in my case threatened death from asphyxia.

In some cases, especially in the very sudden, death is said to occur from inhibition of the Respiratory centre in the Medulla due to a lack of blood being sent to the brain. In the details of all the post mortems on such cases the left side of the heart is found empty of blood.

British and Foreign Med chir Rev. 1/66 p 459

Dr Ogston found the cause of death in 16 cases to be asphyxia, and in 4 to be from syncope, in each of these latter there was fatty degeneration of the heart.

Clinical Soc. Transactions vol xxiv

Dr Rempfry gives a case in which is shewn the wonderful effect of the administration of oxygen gas in these attacks. After 4 hours of other treatment without much avail oxygen gas was administered and "she rapidly improved. The pulse became perceptible", and she went on steadily to recovery".

Ed. Med. Jour. 1892. vol 38 p. 18

Dr Church in reporting a case writes "if one could only have got the "blood to the oxygen or oxygen in some "concentrated form to a stream, even a "small stream, of the blood what a relief "to the patient."

Now let us consider the hæmorrhage. An early and abiding symptom in my case, and one which riveted my attention by its persistency and abundance.

Lancet 2/72 p 409.

Dr Atkinson reports a case in which this symptom developed on the 3rd day. and continued some time.

In Dr Rempy's case it came on the next day after the attack.

Leishman's Midwifery p. 826.

~~From~~ a case of Dr & Mc Millan I quote "15 days after her confinement she had "pain in the lower part of the left lung "posteriorly, this became very acute in "a few hours and resembled a pleuritic

"stitch, subsequently rusty expectoration
" and dulness on percussion supervened,
" and the diagnosis of pleuropneumonia was
" made. She was pretty well on Jan 1st. 75
" On this day severe pain began in the left
" leg & swelling supervened as well
" marked phlegmasia dolens.
Compare this with my case and see the great resemblance.

Of course in my case a pulmonary infarction occurred, which was rendered more probable by the previous obstruction caused by numerous small emboli.
The hemorrhagic sputum is of course due to diapedesis through deteriorated capillary walls.

Theory & Practice of Medicine p. 472. Dr Bristowe

Dr Bristowe says the hemorrhage takes place into the interalveolar texture of the lungs and aircells, and the extravasated blood tends partly to be

expectorated, partly to accumulate in the tissues."

From Cohnheim's experiments it was considered that the haemorrhagic infarction of the lungs was caused by plugging of the pulmonary end arteries removing the blood pressure in the part beyond the plugs, while, there still being pressure in the veins, a backward flow set in from these latter. But it has since been shown in relation to infarcts of the kidney that the engorgement was due rather to the action of the capsular arteries. Therefore, remembering that in the lungs these infarcts are generally peripheral, ~~and that the bronchial arteries supply in part the pleura, as well as the connective tissue of the lungs, the bronchi, and the bronchi~~, may we not suppose that in this case also the ~~capsular~~ vessels of the pleura pulmonalis

Green's Pathology p. 240

have a similar influence in producing a hæmorrhagic infarction!

In Dr Rempy's case, before quoted, we find that the attack was upon Aug 17th. on Aug 18th there was bloody expectoration, and on 21st tubular breathing is heard for the first time. Dr Coats says that "in the lung alveoli the blood acting as a foreign body irritates the tissue and sets up a catarrhal inflammation."

Next we notice in my case that nine days after the chief attack on the 26 Sept phlegmasia dolens attacked the right leg, and after removal in a cab, and making an attempt to sit up on the 13th Oct a similar attack occurred in the left leg. Perhaps, in my case, it would be more accurate to say that painful œdema of the leg occurred with phlebitis. Anyhow there was in each case an evident obstruction

of the veins of the lower Extremities. Here we must recollect that the woman had gone through a severe and debilitating attack in bed, and that there had been great obstruction to the circulation in the lungs. Hence the blood in the veins of the lower extremities would be flowing in a very languid condition, and that thrombosis round the valves of the veins would be an extremely likely occurrence. But moreover plenty of time would have been given for a thrombus of the uterine veins to have grown by accretions down into the femoral veins.

Science & Practice of Midwifery Dr Playfair vol ii p 338

" In one or two cases distinct signs of
" pulmonary obstruction have shewn themselves
" without proving immediately fatal, and
" shortly afterwards, peripheral thrombosis,
" as evidenced by phlegmasia dolens of one
" Extremity, has commenced. Here the

"peripheral thrombosis obviously followed
"the central, both being produced by identical
"causes, and the order of events, necessary
"to uphold the purely embolic theory, was
"reversed."

The assertion of the facts of the events is not to be quarelled with, for they are undeniable, but surely it is quite a different affair to make a dogmatic assertion that they are both produced by identical causes. That statement presupposes, that both are due to "spontaneous coagulation" of the blood which is his pet theory. Now I do not suppose, for one moment, that if he had been merely discussing peripheral thrombosis of the lower extremities that, he would have propounded the theory that it was due to coagulation of the blood from changes in its own nature or composition. He would not then have wilfully shut his

46

eyes to the well known effects that injured vessel walls have upon the blood in contact with them.

Moreover, might he not have asked himself whether the anatomical structure of the veins of the lower extremities and the pulmonary arteries was identical. If the thrombosis of the femoral veins always took place at a branching, then his reference, quoted below, might have been apposite in reference to the pulmonary arteries.

p 336 vol ii "the artery breaks up at once into a number
" of branches, which radiate from it, at different
" angles, to the several parts of the lungs.
" Consequently, a large extent of surface is
" presented to the blood, & there are numerous
" angular projections into the currents,
" both which conditions are calculated to
" induce the spontaneous coagulation of the fibrine"

Had he been able to point out that there were valves in the pulmonary arteries then he would have had greater justification in making his assertion as to identical causation. In one case the vessels are farthest removed from the heart-impulse; in the other they are in closest proximity. In one case, we have the effects of gravity; in the other the heart as a vis a tergo, and the respiratory movements as a vis a fronte.
Moreover, it is very customary to call these cases in general practice "phlegmasia dolens", but it is very questionable whether they have all been accompanied by the peculiar complication in the capillary circulation. Not only so, but these cases seem to frequently to occur at a time after labour, when the peculiar blood dyscrasia, that Dr Kay Jan relies

50

Lancet 2/64 p. 67

CASE 1. *Natural delivery; puerperal mania; peritonitis; death from pulmonary obstruction on the thirteenth day.*—Sarah D——, aged forty-five, married, was confined in the Nightingale ward at King's College Hospital of her first child on the 31st of January, 1865. The labour was rather tedious, but in all other respects perfectly natural. No unfavourable symptoms were observed until the fifth day after delivery, when she became eccentric in manner, and on the seventh she was decidedly maniacal. She did fairly well until the thirteenth day, the pulse being always rapid; but there was no tenderness over the abdomen, nor any œdema of the lower extremities. On the morning of that day she was suddenly attacked with well-marked symptoms of pulmonary obstruction, rapid and gasping respiration, coldness of the face and extremities, and intense syncope. She rallied somewhat on the administration of stimulants; but soon again fell into a semi-unconscious state, and died the same evening.

Shortly *after* she was attacked with chest-symptoms the left leg was found to be swollen, and tender to touch. This was evidently of very recent occurrence, the matron being certain that there was neither pain nor swelling on the previous day.

A post-mortem examination was made twenty-four hours after death. It was found that there had been recent and intense peritonitis. The femoral and other large veins in both lower extremities were filled with dark-red, soft, non-adherent clots, those in the left leg being, if anything, rather larger than in the right, but the difference was not well marked. The vena cava and iliac veins were empty. The left ventricle of the heart contained fluid blood, and in the aortic arch there was a very small pale clot. The pulmonary artery contained an extremely large firm clot, which extended into the right ventricle on the one hand, and into the minute ramifications of both pulmonary arteries on the other. The coagulum adhered firmly to the curtains and tendinous cords of the tricuspid valve, but not to the arterial wall. It was quite solid, and nearly colourless in the larger trunks. It was perfectly uniform in texture through its entire length, and no indication of any impacted embolus could be found, although carefully searched for.†

The history in this case seems to me clearly to show that the pulmonary coagulum was formed before those in the veins of the lower extremities; for not only were the symptoms of pulmonary obstruction observed before the tenderness and

œdema of the leg, but the anatomical peculiarities of the clots point to the same conclusion. Those in the legs were soft, dark, and unadherent; while that in the pulmonary artery was dense, firm, partially adherent, and entirely decolorised, as if the fibrin had been deposited for a considerable time. In this respect the ordinary post-mortem signs met with in undoubted examples of embolism were exactly reversed. In typical cases of the kind, several of which will be found carefully described in Dr. Ball's work, the pulmonary coagula are described as being soft and recent, containing here and there portions of older and firmer clots, generally colourless, exactly corresponding to similar clots in some of the peripheral veins, from which they have been broken off and carried through the right side of the heart, until they were arrested in the pulmonary artery, and served as nuclei round which the more recent coagula were deposited.

upon as the cause of spontaneous coagulation, would be hardly likely to be ~~present~~ still operative. And it is worthy of enquiry why this spontaneous coagulation does not occur in other localities, anatomically far more suitable than the pulmonary arteries, e.g in the venous sinuses of the brain.

Lancet 2/67 page 67.

Sr Playfair says "in almost every case that
" can fairly be ascribed to Embolism, there
" have been well marked symptoms of phlegmasia
" Dolens for a considerable period before the
" fatal attack, seldom less than 10 or 14 days;
" while in this instance the oedema of the leg
" showed itself for the first time after the
" chest symptoms had occurred."

My case is evidently an exception. The blood must have been pretty nearly normal at the time of the accident, more especially as there was no hemorrhage post partum.

52

lancet 2/64
p93

CASE 2. *Placenta prævia, complicated with bronchitis and emphysema; pulmonary thrombosis; death on the fourteenth day.*—On the 8th of October, 1866, a woman twenty-nine years of age, and the mother of five children, was admitted into the Nightingale ward in King's College Hospital. She had been long afflicted with emphysema, and was suffering from an acute exacerbation of her usual chest symptoms. She had lost blood in considerable quantities at intervals for the last month, and at the time of her admission had been in labour for several hours, and was flooding profusely. On examination, the os was found fully dilated, and three-fourths of its diameter was occupied by the placenta. She was losing much blood, and was in so exhausted and debilitated a state that Mr. Dabbs, the resident accoucheur, thought it advisable to terminate the labour at once, and delivered her of a dead child by turning. The uterus contracted firmly, and there was no further hæmorrhage.

For some days she did pretty well, although she was in a very feeble condition, and the bronchitis diminished under appropriate treatment.

On the evening of the fifth day after her delivery the nurse left her feeling comfortable, but on returning to her bedside after a short absence found her in a state of collapse, and breathing with great difficulty. I was sent for, and found her suffering from all the symptoms of pulmonary obstruction. She was gasping for breath, and was almost unconscious, with a pale face and cold extremities. Diffusible stimulants were administered freely, but she never rallied, and died in the course of the night.

On post-mortem examination, the lungs were found highly congested and emphysematous, especially the right lung, in which there were numerous very large bullæ, some as big as a walnut. Both pulmonary arteries, up to the smallest ramifications, contained dark-red, soft, unadherent clots. Both cavities of the heart were empty. No clots were found in the uterine, ovarian, or femoral veins. The uterus itself presented nothing unusual.

Now this case on page 50 seems to me to clearly shew that there was a previous thrombus of the tricuspid valves and that the pulmonary apoplexy was secondary to it. It was an adherent thrombus at the tricuspid shewing that there had been a diseased condition of the arterial wall. Now Dr Humphry B M J 1869 p 582. in backing up the spontaneous coagulation theory endeavours to minimise as much as possible in his own interests any affection of the vessel wall as being the cause + not the effect of the clot. Now, I quote the following so as to render it quite clear what Dr Playfair is arguing for.

Lancet 2/64 p 94

"When spontaneous thrombosis occurs, on the
"other hand, it must arise from the state of
"the blood rendering it peculiarly liable to
"coagulate, & this is more likely to be met with
"at a much nearer period to delivery"

Case 2. on the opposite page certainly does not

54

Lancet 2/67 p154 ✳

CASE 5. *Symptoms of pulmonary obstruction eleven days after labour; phlegmasia dolens; recovery.*—On Aug. 20th, 1866, I was consulted by a lady forty-four years of age, the mother of twelve children. She complained of general weakness and debility, and she had phlegmasia dolens of the left lower extremity, which was hard and swollen up to middle of thigh. She informed me that she had had an easy labour on the 6th of July. She made a good recovery, and on the eleventh night went to bed feeling very well. There was no swelling or discomfort of any kind about the lower extremities at this time. About half-past three A.M. she was sitting up in bed, when she was suddenly attacked with an indescribable sense of oppression in the chest, and fell back in a semi-unconscious state, gasping for breath. Her husband, greatly alarmed, went for the nearest medical man, who administered stimulants freely. She remained in this condition for about three hours, when she commenced to rally. The secretion of milk, previously abundant, was suspended. Two days *after* the attack, symptoms of phlegmasia dolens came on; the left leg swelled, and has since remained in the same condition as it was when I saw her. She has since been very feeble and out of health, but there has been no recurrence of syncope.

support Dr Playfair. It rather supports Dr Ball in his contention that the thrombosis begins in the peripheral vessels and creeps backwards. But, we must remember that these peripheral vessels were, in this case, in a very damaged condition from Emphysema. Even if I seem to reiterate, I will quote this sentence from the same paper at the foot of column 1, p 154." I believe therefore, * " that, as in Case I, this is an instance " of pulmonary and peripheral thrombosis " occurring simultaneously from some " condition of the blood tending to coagulation"

It seems to me that Dr Playfair does not seem to fully appreciate the dangers of canalisation of thrombosed veins, more especially when he demands that the pulmonary clots should be of the same consistence as the primary.

> Clots in the peripheral veins are certainly frequently absorbed, and the circulation in the occluded vessels becomes as perfect as ever. So strong is the tendency to this that Humphry observes with regard to it: "It appears that the blood is almost sure to revert to its natural channel in process of time, unless the vein be completely destroyed."* If this, then, be so frequent an occurrence in the venous system generally, surely it is more than probable that it may now and then occur in the pulmonary artery also.

Lancet p 153

86

Lancet
2/67 p 154

Addendum.—The following case has come under my observation since the above was written, and, although not connected with the puerperal state, it is in itself so instructive and interesting that I venture to relate it. It would be impossible to bring forward a more striking example of the insidious way in which this affection sometimes comes on, or of the terrible suddenness with which it may carry off a patient whose general condition shows no ground for alarm.

Rheumatism of the knee of three days' duration; sudden death; obstruction of the pulmonary arteries.—On Friday, the 15th March, 1867, I was requested to prescribe for one of the nurses employed in the children's ward at King's College Hospital. She was an apparently healthy girl, nineteen years of age. She complained of pain in the right knee, which was, however, neither swollen nor tender on pressure. The pain had been felt on the previous day for the first time. The bowels were constipated, and the tongue was foul; but there was no general feverishness, the skin was cool and moist, and the pulse was under 80. She had never suffered previously from rheumatism. Altogether, she gave me the idea of having a very slight indisposition. I prescribed an alkaline mixture containing bicarbonate of potass, with a Dover's powder at bedtime if the pain rendered it necessary. She had already taken some aperient medicine, which had not as yet operated. The knee was wrapped in cotton wadding and oiled silk. On the next day I did not see her, but it appears that she remained in much the same condition. There was no pain in any other joint. She took her food well, and was cheerful and happy as usual. She merely stayed in bed to rest her knee. On the morning of Sunday, the 17th, she awoke at six A.M., and entered into conversation with one of the other nurses who slept in the same room. She said that she had passed a good night, and that she felt altogether better. Shortly after this she complained of feeling weak, and asked one of her companions to give her a bed-pan, as she wished to pass water, and felt disinclined to get out of bed. The nurse gave it to her, and retired to the other end of the room to dress herself, but soon returned to ——'s bedside, being attracted by her rapid and gasping breath. Her appearance was so alarming that she called out for assistance. Dr. Fenn, the house-physician, was immediately summoned, but she was dead before he could reach her.

An autopsy was made by Dr. Kelly, Pathological Registrar to the Hospital, twenty-six hours after death. There was no effusion in the pericardium, and so sign whatever of either pericarditis or endocarditis. The structure and valves of the heart were healthy. The left side of the heart and the aorta were empty. In the right ventricle there was a large, firm coagulum of decolorised fibrin, adherent to the valves and walls of the ventricle. A similar clot extended into the pulmonary arteries on both sides for a considerable distance, but did not reach the smaller ramifications. It was of the same firm structure as that in the ventricle, of a pale-yellow colour, and cylindrical in shape. It was quite solid, nothing like a central channel existing. It was not attached to the coats of the arteries, which were themselves healthy in appearance. Both the cardiac and arterial clots were perfectly uniform in structure, there being no trace of an impacted embolus. The vena cava and iliac veins contained dark black clots, evidently of post-mortem origin, and entirely different in structure and appearance from those in the pulmonary arteries. All the other organs in the body were healthy.

Here we have a blood dyscrasia, in so far, at least, resembling the condition after delivery that there was an increased amount of fibrin present. However great the alteration of the blood may have been, it is certain that there were no general symptoms calculated to give rise to the least anxiety. There were four nurses sleeping in the same room as this girl. They had all been chatting and laughing together the previous night, and none of them saw anything to make them think that their companion was even seriously ill. One of them, who had been wakeful during the night, had noticed how calmly and quietly she slept. It appears to me that there had been deposition of fibrin going on for some time before death, probably during the whole night. On waking, before making any exertion, she felt pretty well, although weak. The attempt to micturate, however, necessitated an exertion which acted as the immediate cause of the fatal syncope, as happens in the majority of fatal cases. Before this sufficient blood reached the lungs to carry on respiration without much inconvenience, but enough could not pass when respiration was hurried by movement.

I see no reason to doubt this being a true case of thrombosis, not of embolism. There were no peripheral clots from which an embolus could come, nor was there any appearance of an impacted portion in the clot itself. If so, it goes far to disprove Dr. Ball's theory, that spontaneous thrombosis can only form in the smaller divisions of the pulmonary arteries, gradually creeping up to the larger ones, since in this case the smaller ramifications were collapsed and empty.

I give the case on the opposite side to shew how very much out of his way Dr Playfair can go to find an analogous case to bolster up his dyscrasia of the blood theory.
We know that there is nothing more liable than Rheumatic fever to damage the endothelial lining of bloodvessels. We have the firm adherent clot in the right heart. This case certainly does not prove that the blood can coagulate from some alteration in its nature, without any primary alteration of the vessel wall. Thrombi can grow as this case shews, but more could it does not prove that there could be a deposition of clot without some breaking down of the vital resisting power of healthy Endothelium. Dr Playfair relies on the the similarity of the blood dyscrasia, but in Rheumatism we generally get Cerebral Embolism as a sequence to Cardiac

58

endocarditis. I copy the following from Dr Ogston's paper above referred to (p. 33)

"Rokitansky says "This form of apoplexy "(pulmonary) is very frequently found to be associated "with active dilatation of the right side of "the heart, and seems to bear the same "pathogenic relation to this cardiac affection "as cerebral apoplexy bears to active "dilatation of the left side of the heart."

Brit & For. Med Chir. Rev. 1/66 p459

Dr Ogston examined post mortem 20 cases of sudden death by pulmonary apoplexy.

15 males and 5 females.

Average age of males = 55·3 | One male = 24 yrs
" " " females = 54·2 | " female = 16 yrs

All had been of intemperate habits or of enfeebled constitution.

The apoplexy was in the upper lobe of the Right lung in 4
middle 6
inf^{r} 11
upper Left 6
lower " 18

60

Braithwaite's Retrospect: 2/85: p109. A case by Mr Shield.

A single woman, at 30, suffered from varicose veins of the lower extremities. She was well nourished and healthy looking. On the inner aspect of the left leg below the knee there were 2 clusters of enlarged, hard, painful veins with redness and superficial edema. She was 7 months pregnant. Lotions and rest were prescribed. One day the patient sat up in bed and commenced to shake her pillow, when the nurse heard a moan in the ward, and turning round, saw the patient deeply cyanosed, and gasping in all the agony of impending death. She lived after this probably about 5 minutes Previous to the attack she had been cheerful and free from any serious respiratory symptoms. P. M. Face cyanosed. Lungs larger than natural and were bright red in color, and engorged with frothy blood. The smaller branches of the pulmonary artery were filled with dark red cylinders of clot which could easily be expressed. This was more marked in those vessels the size of a small quill, though by the aid of a lens, the minute arterial twigs were seen to be in the same condition reminding one of the appearance of a lung injected from the pulmonary artery with lead chrome or red wax. The right ventricle was occupied by a large clot which fixed off into the pulmonary artery as far as the bifurcation of the great vessel, where it was in contact with but not joined to another clot which extended into the smaller divisions of the pulmonary artery. Neither did it extend into all the divisions of the pulmonary artery with equal regularity for some remained patent a little way, and on being traced up were found more or less completely blocked at intervals with friable masses of fibrinous clot, dull [illegible] coloured, here and there suffused with blotches of dark-blood color. The Right Auricle

There was dilatation of the Aorta	in	5
Fatty degeneration of the heart	in	5
Heart-loaded with fat	in	3
General enlargement of the heart	..	2
Attenuation of walls of R heart		2
both sides		1
hypertrophy of walls of left Heart		1
left Ventricle		1
Dilatation & Attenuation of R heart		1
Disease of Mitral valve		3
Tricuspid		1
aortic		2.

In the case reported on p 66 we notice that the "in the right ventricle there was a large firm coagulum of decolorised fibrine, adherent to the valves and walls of the ventricle." Compare with this M'Coat's Pathology p 28 on Heart-Thrombi. "These thrombi are frequently of a pearly white colour, and are never composed of the whole blood. As dilatation occurs more frequently

and the pulmonary veins and the left cavity of the heart were empty. The clusters of veins on the inner aspect of the leg contained red masses of clot, here & there slightly mottled on the surface. The process seemed one of passive coagulation in tortuous veins and not due to any active inflammatory mischief.

"in the right heart than the left, these thrombi (
" which were called by Laennec, globular vegetations)
" are most frequent in the right cavities than in
" the left, although by no means rare in the left ventricle,

It is noticeable that in Mr Shield's case reported on page 61 both the peripheral and pulmonary clots were soft. Now clots have, as it were, a regular life history and I cannot see myself anything to prevent embolism at any part of this. Why should not a soft clot not yet organised, give rise to a fatal pulmonary embolism. It would be washed to the right heart and might be broken up there by the columnae carneae and valves and give rise to thrombosis, or be washed against the acute angles of the pulmonary arteries and separate into many portions, as a soft mass of weeds will break up when floated against the buttress of a bridge, and so ~~multiply~~ most numerous

centres for thrombic processes would be furnished. Such a state of things might give rise to the appearance of a spontaneous coagulation of the blood in the lungs and right heart. Then clots which are becoming organised might break down, just as callus may soften, owing to some depressing influence. Not to mention the effects of septicaemia. In this thesis I am only treating of simple clots. Then we may have later on embolism produced during the natural processes for the reopening of the calibre of a thrombosed vessel.

Green's pathology

"A thrombus softens often due simply to the "chemical changes which the constituents of a "clot undergo when dead, but a-septic, and results "in the formation of a more or less pappy "substance which has a red or white color "according as it originates in a red or white "thrombus — debris of corpuscles and fibrin,

66

Lancet 2/72 p409.

Woman was aged 35. Up to the eighth month she was all right. Then she had a violent attack of vomiting on March 24th at 4 am. Suddenly afterwards syncope came on. Respirations 45. Pulse too rapid and small to register. Great anxiety of countenance, lividity, extremities and surface cold. Intellect clear. Next day, 28th, she was confined of a still born child. On this day P. 130. Great dyspnoea. 29th a cough which was troublesome had come on with expectoration of sanguineous sputa. There was pain and oppression about the heart. 30th Consultation with Dr Giles of Oxford. R. 75 P 130. Cough troublesome. Dr Giles found dulness & crepitation at the base of the left lung. Ap. 2nd right lung became involved, cough very troublesome. Expectoration viscid and sanguineous. The inflammatory mischief was spreading. She recovered slowly.

" albuminous fatty and pigmentary granules.
" Canalisation may take place. The larger particles
" will give rise to Embolisms, probably too
" minute to cause symptoms."
And if they caused ~~pulmonary~~ coagulation in the pulmonary arteries, they, as sources could not be discovered, and then the case would be one to support the case for the spontaneous coagulation theory.
And in these puerperal cases it should be remembered that ante partum puerperal embolism may be cause by clots formed from detachment of the placenta in portions. There is an excellent case of this reported in the Lancet 2/72 p409 by D^r J P Atkinson. D^r Playfair forgets this when he writes p337 " ~~and in all of them, with no exception, death occurred before the 19th day~~" The reason of this (viz that the Embolic cases all occurred after the nineteenth day, and all the earlier cases were instances of spontaneous coagulation) seems to be that in the former.

"time is required to admit of degenerative
"changes taking place in the deposited fibrine
"leading to separation of an embolus."
The clots may be in some of the uterine
sinuses undergoing these changes for some
time before labour. There is no doubt at
all that this question of the nature of
the clots found in the lungs is still in a
most uncertain condition. We want more
careful work done on this subject in the
post mortem room. The work is surrounded
with difficulties. The distinguishing for certain
between ante mortem and post mortem clots.
Clots formed in the death agony, and clots
giving rise to the death agony. How exceedingly
difficult it is to examine all the sources of
a peripheral thrombosis. This has once been
discovered in the hemorrhoidal veins.
How very hard to make absolutely certain
that the mass of pulmonary clot does not

include minute embolic centres for secondary thrombosis (or rather tertiary thrombosis). Dr Playfair says p. 337. "surely the mechanical "causes which are sufficient to prevent the "spontaneous deposition of fibrine, would also "suffice to prevent its gathering round an "embolus". But the embolus is a foreign body acting as a direct cause of coagulation. Normally in the vessels "blood never is, but always to be clotted". The blood is always ready to clot given the requisite conditions. One always feels a desire to remind some writers that fibrine as such has no existence in the normal blood.

Now I think we will turn our attention to blood dyscrasia and coagulation.

At the ninth month in a pregnant woman the proportion of corpuscles is greatly diminished.

The proportion of fibrin increases from the

6th month to delivery, and continues for a certain time after delivery.
At the 9th month there is a decrease of albumen.
The proportion of water in the blood increases sensibly towards the end of the ninth month.
The serum is more abundant relatively to the fibrin and corpuscles, and contains less solid matter, which of course helps to increase the total amt of water in the blood.
The above facts are taken from Cazeaux and Tarnier's Midwifery.

	Average in woman.	in 7 cases Average at 9 months
Corpuscles	125	100·45
Fibrin	3	4 to 4·8
water	791·1	818·64

The quantity of white corpuscles is increased in fact the condition approaches to Chlorosis

This alteration in the composition of the

woman's blood is doubtless due to the drain upon her by the nutriment required for the growth of the child. But it should be recollected that after labour there is a large diminution of the blood sent to the uterus and the blood of the woman tends to become more & more natural towards the normal.

Although the blood of the woman in the 9th month is thus altered in relative composition yet it is no more liable to spontaneous coagulation in normal bloodvessels than in the ~~woman~~ non-pregnant female. All that one can say is that if clotting takes place there will be a larger clot. The rapidity of clotting depends upon the amount of ferment present.

As to the influence of involution upon rendering the blood liable to spontaneous coagulation, it must be recollected that the Lochia and the streams by which those effete matters, which are incapable of being so altered as to be rendered again

* "Lymph and chyle contain essentially the same constituents that are found in blood. Their composition differs from that of blood in degree rather than in kind. They do not, however, unless by accident, contain colored corpuscles". Baker's Physiology p 368.
Sir M Foster vol ii 498 says "Broadly speaking we may say that all the substances present in blood plasma are present also in lymph, but are accompanied by a larger quantity of water".

fit to circulate in the blood, are removed from the body. And supposing that the amount of fats is increased, yet there is the milk which draws upon this and other products e.g water that compose the blood. ~~-/~~

Lister has advocated the view that "the blood has no natural tendency to clot, but that its coagulation out of the body is due to the action of foreign matter with which it happens to be brought in contact, and in the body to conditions of the tissues, which cause them to act towards it like foreign matter. Brücke on the other hand, supposes that there is a natural tendency on the part of the blood to coagulate, but that this is restrained in the living body by some inhibitory power resident in the walls of the containing vessels". It will not be disputed that living tissue has a wonderful preventive influence

Baker's Handbook of Physiology p. 101.

47

against the clotting of blood. Lister has shewn that if an artery be ligatured in two places, and cut out while full of blood, it may be hung up, and the blood will remain fluid for some days. Lister says also blood remain fluid for a long time in the angle between an amputated sheep's foot and the skin raised in a flap from it, and we know that extravasations about simple fractures, and into body cavities are long in coagulating. In Green's pathology p 233 it says "the time before coagulation occurs in the jugular vein of a mammal is longer in proportion to the care with which it is laid bare and the ligatures are applied, and if this operation be done antiseptically coagulation does not occur at all". Now, in ligaturing arteries we note this fact that the coagulation begins nearest the ligature by thrombosis

at the point where the ligature has damaged the vessel wall and spreads from there by additional deposition.

It is interesting to observe how long after death the blood remains uncoagulated in the vessels and that it coagulates sooner in the heart and larger vessels. This has been said to be due to the fact that in the smallest vessels there is more comparative contact between the surface of the vessel wall and the volume of contained blood. But in this connection it is worthy of note what was said in the Croonian lecture (Lancet 1863) by Sr Lister. "I believe that we must accept it simply as an ultimate fact that just as the brain loses its vital properties earlier than the ganglia of the heart, so the heart & the principal vascular trunks lose their power sooner than the small vessels of the viscera, or than more superficial vessels be they large or small."

Our knowledge of coagulation comes to this. It has been shewn that a normal clot outside the body can be formed by the action of fibrin ferment upon fibrinogen. There seems to be only the minutest quantity, if any, of this ferment in the blood as it leaves the body. Very active ferment has been injected into the blood current of a living animal, according to Dr M. Foster, without producing any clotting. There must be some inhibitory influence then on this ferment in living bloodvessels. Such experiments are however rarely satisfactory. Now Schmidt asserts that the injection of leucocytes into the circulation of an animal, leads to their disintegration and the consequent local or even general formation of thrombi. Many believe that the fibrin ferment

is contained in the leucocytes, and is set free by their disintegration. So that anything which might lead to the destruction of leucocytes in the living tissues, might if the living tissues do not inhibit the ferment action, cause Spontaneous coagulation of blood in the tissues. And how far Micro-organisms might be successful in accomplishing this, it would be interesting to know.

Dr Foster writes "absence of motion, which
" in shed blood is unfavourable to clotting,
" is apt within the body to lead to clotting.
" This, perhaps, may be explained by
" the fact that the walls of the tract
" suffer in their nutrition by the
" stagnation of the blood, & that consequently
" the normal relation between them
" and the contained blood is disturbed.

85

Whether clotting within and without the body is in reality the same process might by some be questioned.

Now, I think we might look at the best cases of thrombosis we know of viz peripheral thrombosis, and see if it can throw any light on the general question.

1. An extreme retardation of the blood current is the most important cause.

a. The muscular action of the heart may be weak. Thus we find femoral thrombosis in the last stage of phthisis, cancer, and other exhausting diseases, in convalescence from Typhoid and after confinements.

b. There may be some obstruction in the lungs which lessens the force of the circulation.

c. The force of gravity acting on a column of venous blood.

Then there is the anatomical feature

of the valves of the veins predisposing to thrombosis. Dr Coats says p28 "In the "sluggish state of the circulation the "valves lie half closed, the force of the "blood being insufficient to press them "back against the vessel wall. Hence "the blood will stagnate in the pouches "of the valves and coagulation will "begin there. Now this stagnation is only a predisposing cause and not a determining cause. We must first have damage of the endothelium. If the endothelium is nourished by the blood passing over it, then the stagnation of the blood may well cause deterioration of the endothelium. But it has always seemed to me that as there is considerable dilatation of the vessel wall at this part there must be considerable thinning of it, and we may compare what takes

89

vesicular
place with what happens in emphysema
in the lungs. The capillary bloodvessels become
obliterated, and if the endothelium owes
its blood supply & consequent nourishment
to very minute vasa vasorum (as is
asserted in Gray's anatomy) then the
~~destruction~~ necrosis of the endothelium in
the pouches of the valves of veins may be due
to ~~the same~~ a similar cause. A thrombus
once started continues to grow. By the
way a septic phlebitis might cause
considerable damage to the endothelium
by obliterating the vasa vasorum
A thrombus of the uterine sinuses
has been known to grow right down into
the femoral veins.

How much the same state of things
occurs in the heart. We have the valves,
and not only these but the columnæ
carneæ, and many nooks and crevices.

Eq1

Dilatations of the right heart are common enough, and clots easily tend to form for similar reasons as in femoral thrombus in the apices of the ventricle and in the auricular appendage.

Fatty degeneration must be a very fertile cause of clots in the heart, causing dilatation and weak muscular contraction by which the heart is never able properly to empty itself.

Fatty degeneration is not uncommon in pregnant women and after labour In Gray's Hospital Reports 3rd Series xviii Dr J. J Phillips shews from the post mortems in several cases of sudden death after labour that the cause was syncope due to fatty degeneration of the heart.

The anæmic condition of the patient acts, Dr Fancy, must strongly in causing thrombus by its deteriorating effect on the vessel wall

Thus we see that we have two sources of embolism. (1) the peripheral veins
(2) the right side of the heart.
In connection with the latter I may quote an interesting case from Galabin's Midwifery p 794. (a case recorded by Dr Playfair Obst Trans vol xxvi)

"Labour had been severe and protracted, and
" the patient greatly exhausted. After delivery
" the T had fluctuated considerably, often
" reaching a high point, and, rarely, or never,
" becoming normal. There had been one or two
" paroxysms of dyspnoea. 3 weeks after delivery
" Dr Broadbent found a bruit indicating
" thrombosis of the right auricle. Phlebitis was
" just beginning in the left post.r tibial vein.
" A few days later there were symptoms of
" embolism of a branch of the pulmonary artery
" leading to the lower part of the left lung,
" viz impaired resonance, imperfect entry

96

" of air + loud harsh friction. The auricular
" murmur was no longer audible, but there
" was a systolic murmur over the pulmonary
" artery and obscuration of the pulmonary
" second sound, the coagulum interfering
" with the proper closing of the valves. Just
" at this time thrombosis of the right iliac
" vein appeared, + Extensive phlegmasia Dolens
" of the right leg was developed. The patient
" Eventually completely recovered." Cp also
the case on page 56.

Rankin's Abstracts 1/61 pp 76-86.

In Mr Barnes remarks on 14 cases of "Thrombosis and Embolia" of lying-in women, he says
"In the cases suddenly fatal it was generally
" found that not only were the main branches of
" the pulmonary artery filled with coagula as
" well as the smaller ramifications obstructed but
" that clots existed in the right heart".

how as to thrombosis beginning in the pulmonary artery itself. I think it very probable that in some cases of pulmonary disease in which the smallest arteries are damaged that thrombosis takes place in them and that then (aided by the obstructive effect in retarding the blood current in that side) it spreads back towards the heart.

Dr Barnes says in the paper above mentioned

"As far as careful dissection can shew
"there seems good reason to conclude that
"obstruction of the pulmonary arteries
"may arise from primary sudden or
"gradual formation of clots in these vessels
"themselves"

Impaired circulation in a part means damage to the tissues - to its vascular endothelium among others. Retardation and stagnation are at most indirect causes of thrombosis.

99

Atheromatous ulcers, and changes due to extension to veins and arteries of spreading inflammatory processes, may give rise to thrombosis.

It has been noted that thrombosis is an occasional complication of some of the specific fevers, and it is now believed that this is due to a desquammation of the endothelium which is comparable with that ~~desquam~~ of the Epidermis in E.S scarlatina.

In the B.M.J 1869 there are some cases by G. M. Humphry M.D. and I quote the following under the belief that tubercles may produce thrombosis of the pulmonary artery."

Case iv "An emaciated young man had abdominal
" & pulmonary phthisis. Reduced reflexes.
" There were tubercles in the peritoneum &
" in both lungs. In the pulmonary artery

" of one lung was a large firm fibrinous clot,
" nearly filling the vessel, with prolongations
" extending into its 3rd & 4th divisions. It was
" slightly adherent about the point where the
" artery first divides, and the interior of the
" vessel, at this part was rough & reddish when
" the clot had been removed. At the corresponding
" situation in the other pulmonary artery
" was a mottled clot of darker color, & apparently
" more recent. It was slightly adherent to the
" vessel & its prolongations extended into the
" larger branches."

I cannot but accept the possibility of thrombosis originating in the pulmonary arteries themselves, although I think that cases of such must be very rare, but I feel sure that there is no evidence at all that this is due to any changes in the blood but entirely due to damage of the vessel wall which destroys its power of relative resistance to coagulation of the blood.

Dr Humphery noted "that, as a general
"rule, the formation of the clot commences
"at the outside, that is near to the coats of
"the vein — and the first stage in the
"process is the settling of a patch or layer
"of fibrine upon the inner surface of the
"vein". And Dr Lister in his Croonian
lecture (Lancet/62) says
"It is an interesting circumstance with
"reference to the question which we are
"now considering that the coagulation always
"begins in contact with the vein, indicating
"that it is not the wall of the vessel that
"keeps the blood fluid, but that, on the
"contrary, the wall of the vessel when
"deprived of vital properties makes the
"blood coagulate."

Thrombosis of the cerebral sinuses and veins
may occur in infancy and childhood

in cases of extreme anæmia after exhausting diseases as acute diarrhœa, where the action of the heart is weakened and a stasis of the blood takes place. It may also occur in the neighbourhood of some inflammation as otitis. Therefore in these cases we have

(1) extreme debility of the patient.
(2) very weak circulation.
(3) blood in a distant part tending to stagnation in tissues consequently ill supplied with blood.
(4) due to acute phlebitis.

As regards bed sores we know they occur in situations subjected to pressure, and depend in great part on the congestion and lowered vitality of the tissues induced by the impairment of the nerve influence. It might be argued that coagulation of the blood took place in these veins due to the stagnation as a predisposing cause, and the loss of

vitality in the vessels as the exciting cause. But I rather think myself that these bedsores are due to

either (1) anaemia of a part caused by continual pressure of the flesh over a bony prominence

or (2) ulceration of the skin and tissues by continued irritation of acid discharges as urine and faeces.

What one asks very particularly is, why, if these cases we have been discussing are due to pulmonary thrombosis due to the peculiar constitution, why does it not coagulate in every part of the system where from position or anatomical construction there is any approach to stasis of the blood. Surely if Sir Playfair were right intravenous injection would be a peculiarly risky operation. It is the sudden deaths very soon after labour that present the difficulty.

We have seen how very difficult and unsatisfactory are the results of post mortem examination.
The suddenness of the attacks, the previous easy and natural condition of the patient, the frequency with which the attacks are consequent upon exertion, all strongly favour the causation as being Embolism either from a peripheral venous thrombus, or from a central or cardiac thrombus, or perhaps rarely from Embolism from a thrombus on larger trunks of the pulmonary artery.
That otherwise, cases of thrombosis of the pulmonary arteries are those of obstruction of the minute divisions by thrombosis (cp Dr Ball's theory) and consequent spreading back of this towards the heart.
That the constitution of the blood has no other than an accelerating influence

on the production of clot after that the vessel wall is prepared to excite the thrombosis. It is not the blood, but the loss of the vital power in the vessel wall.

As regards treatment.

When the condition of the woman after labour is such as would predispose her to be a likely subject of these accidents, then rest, complete rest, is the best prophylactic treatment, along with all means that are useful for strengthening the patient and improving the condition of her blood, more especially iron tonics. During the attack – stimulants, of which strychnia and Ether are the most valuable. And plenty of fresh air. The administration of Oxygen gas is undoubtedly the most powerful & effective treatment.

After the attack — stimulants, iron tonics, and complete and prolonged rest.

Finis.