

of the bowels, are unsuited for cases of intussusception or other varieties of acute obstruction.

V. In cases of acute intestinal obstruction from other causes than intussusception, should milder measures fail to give relief in the course of three or at most four days, laparotomy should be absolutely recommended, and may under such circumstances be resorted to with a reasonable hope of success.

ART. V.—On *Thrombosis of the Cerebral Veins, and Sinuses of the Dura Mater*. (Second paper.) By JOHN A. LIDELL, M.D., of New York.

III.—THE traumatic and inflammatory forms of this disease, or rather the clinical history of these forms, were minutely described in the number of this Journal for January, 1874. There is, however, still another variety of thrombosis of the cerebral veins and sinuses of the dura mater, which possesses a great deal of interest and importance. It is met with only in subjects who are much weakened from want of food, or loss of blood, or profuse discharges, or broken down by the weight of years, or worn-out by some exhausting disease, that is, in marasmic subjects, without the concurrence of any local traumatic or inflammatory lesion to which the origin of the thrombus can be ascribed, and hence we have, in the *third* place, a *marasmic* variety of thrombosis that affects these veins and sinuses. It is strictly analogous to the marasmic variety of thrombosis, which sometimes presents itself in the veins of the extremities. It is of pretty frequent occurrence. It embraces rather more than one-fourth of the cases collected by Prof. Von Dusch; and almost one-fourth of those collected by the writer. It is important not only from the frequency of its occurrence, but when we closely examine the individual instances of it, and especially when we contrast them with the cases which belong to the other varieties of cerebro-venous thrombosis, we find important differences as well as numerous other points of interest. In the writer's opinion the *marasmic*, as well as the traumatic and inflammatory varieties of cerebro-venous thrombosis, has not yet received that degree of attention which it really deserves. He therefore proposes to describe it also, as fully as he can, within reasonable limits; and this description will be chiefly drawn from the history of thirty-eight cases, of which one was observed, and twenty-one were collected by Von Dusch, and an additional sixteen were observed or collected by the writer. It may here be remarked that Von Dusch styles this variety of cerebro-venous thrombosis "*thrombosis of the sinuses from debilitating influences*." The term *marasmic* thrombosis of the above-named vessels, however, is preferable in a vast majority of instances.

1. Perhaps the most striking of all the cases belonging to this category, are those in which young children having diarrhœa, or dysentery, or cholera infantum, but especially those who are badly nourished, or have become much debilitated and shrunk in appearance from the profuseness or long-continuance of alvine discharges, rapidly sink into a state of unconsciousness, with or without convulsions, and thus after a time expire. Dr. Gerhardt (*Von Dusch's Memoirs*, New Sydenham Soc. Ed., pp. 111-114) relates four cases of this sort. They all occurred in very young, artificially fed children, and agree very closely with each other. Take the following as a fair example of the clinical phenomena and post-mortem appearances.

A well-fed boy, three months old, was seized with profuse diarrhœa; greater fontanelle flat, and pulsating strongly; temporal and frontal veins very prominent; both jugulars much and equally distended. He lies quietly, with an unconscious stare, and occasional strabismus; both pupils equally dilated. Then there followed complete unconsciousness, opisthotonos with rigidity, sinking of the fontanelles, overlapping of the skull bones, convergent strabismus, etc.; the left external jugular became more distended; then the right external jugular enormously so, while the left one appeared almost empty; left side of face slightly paralyzed; left pupil more dilated; after some transient improvement, death on the eleventh day. *Autopsy*.—Superior longitudinal sinus, filled with fluid blood and recent coagula, anteriorly. A knobby, discolored, firm thrombus in posterior part; it also extends into both lateral sinuses, and caases them, especially the left, to appear externally like thick, roandish hard cords; in the left it is partially adherent, and completely fills the sinus; in the right not. Hyperæmia of pia mater, and gray substance; extravasation beneath scalp, at back part of head; bones of skull very hyperæmic in some situation; pneumonin patches in both lungs; usual condition of intestine. (*Ibid.*, p. 112.)

Another case was that of a little girl, aged eleven months, who sank into stupor and died, after only three days' vomiting and diarrhœa. The skull bones overlapped, and the nape muscles were contracted. On *autopsy* the straight and both lateral sinuses were found plugged up with a crumbling, adherent, symmetrically formed thrombus, while the brain, pia mater, and plexuses were hyperæmic and œdematous. No other lesion was revealed that would account for the head symptoms and the fatal issue. It is not improbable that the following case, which occurred in the writer's practice, belongs to the same category as the above:—

CASE LV.—The patient, a pale-looking little boy, aged about two and a half years, had dysenteric diarrhœa. The disease appeared to readily yield to the treatment employed, which consisted mainly in a carefully regulated diet, confinement to bed, porticing the bowels, and administering calomel with Dover's powder in small doses, alternated occasionally with ol. ricini as a laxative. At the end of about the third day he was free from fever, his pulse and skin had become natural, his countenance bright though pale, his stools much less frequent, and more natural in appearance, and I expected he would make a speedy recovery. The next morning, however, I found him lying in a state of profound stupor, with dilated pupils, and a very pale face, I learned that the diarrhœa had given no trouble through the night, and that he was discovered to be insensible on trying to waken him in the morning. He sank rapidly, and died comatose, in the evening. No *autopsy*. His appearance was quite anæmic at the onset of his attack. It should also be stated that he had recently crossed the ocean from Europe with his parents, who were trades-people in moderate circumstances. This voyage, with the insufficient feeding for an infant likely

to attend it, may have had something to do in producing the anæmic state which was already present when the diarrhœa appeared. Now, although no autopsy was allowed in this case, I infer from the nature of his disease, from the morbid state of his blood, from the sudden appearance of stupor and coma, and from the post-mortem lesions which have been found in strictly analogous cases, that this little patient too had thrombosis of the sinuses of the dura mater which caused the coma and the fatal result.

With regard to the causation of the thrombi, and the interpretation of the clinical phenomena in cases such as the above, Dr. Gerhardt has offered a very reasonable, and upon the whole a very satisfactory theory, which is in substance as follows:—

The profuse discharge of fluids which not infrequently attends the diarrhœa, as well as the cholera of infants, necessarily lessens the quantity of the blood, and produces inspissation of it. At the same time absorption of the parenchymatous fluid takes place throughout the substance of the organs in general, but especially in that of the very watery brain of infants. In consequence of this the contents of the skull become correspondingly diminished in volume, whereby the atmospheric pressure causes, first, depression of the fontanelles, and afterwards, when this no longer suffices, overlapping at the sutures. If this compensation also prove insufficient, a distension of the vessels of the brain and its meninges occurs; and the diminution of the general mass of the blood, the lowering of the power of the heart, and the inspissation of the blood itself, all tend to retard the current, that is, to produce stagnation, and lead to the formation of coagula in a locality so favorable as the sinuses of the dura mater. The unequal prominence of the jugular veins is a natural consequence of the obstruction of one of the lateral sinuses with coagulum, inasmuch as the vein corresponding to the obstructed sinus will be less full than the other. This phenomenon, which must be most strongly marked in the deep lying internal jugular vein, also shows itself by a circuitous route in the external jugular of the same side.

The observations of Dr. Gerhardt and others, render it pretty certain that in cases of cholera infantum and other diarrhœal diseases of children, where loss of consciousness, and other head symptoms suddenly and unexpectedly present themselves, the cerebral phenomena are due to the obstruction of the sinuses of the dura mater with coagula, much oftener than is generally supposed.

2. But thrombosis of the cerebral veins and sinuses of the dura mater not unfrequently occurs in children suffering from other diseases, especially those of a chronic and exhausting character. Thus, Prof Von Dusch relates the case of an infant, aged nine months, which came under his own observation.

The little patient was teething, and much debilitated by the discharge from a large abscess of the thigh. Death was preceded by great collapse, without convulsions. The superior longitudinal sinus was found completely filled anteriorly by a firm, three-cornered, pale clot of blood, that adhered to its walls, and was laminated in structure. The veins emptying into this sinus also contained tough, firm, colourless clots. Besides, the same writer has collected five other juvenile cases, in which the marasmic variety of cerebro-venous thrombosis appears to have been the immediate cause of death. One of them was a child, aged two years, "which had long been very weakly;" another was a little girl, aged four years, who had strumous ophthalmia, with swelling of the glands, and pneumonia; the third a boy, aged fourteen years, who suffered from ague-cachexia, with œdema of the limbs, enlargement of the liver and spleen, diar-

rhœa, etc.; the fourth a boy, aged fifteen years, who had for several months suffered from plenrisy, with œdema of the feet, and dyspœnœ; and the last, a girl, aged twelve years, suffering from typhoid fever, one month advanced in its course.

Dr. West (*Lectures*, etc., pp. 108, 109, 4th Am. ed.) relates a case in which the cerebral veins and sinuses of the dura mater were extensively obstructed with thrombi, and death appeared to have resulted therefrom.

CASE LVI.—The patient was a little girl, aged about thirteen months, who had sunk into a state of marasmus after an attack of scarlet fever. "No new symptoms came on till she was suddenly seized with extreme faintness, amounting to almost perfect syncope. She rallied, however, under the use of stimulants; but, forty-eight hours afterwards, the faintness returned, and terminated in death, without any convulsion having preceded it." On *autopsy*, the posterior half of the longitudinal sinus, the torcular, the left lateral, and left occipital sinuses were found blocked up with fibrinous conglum. and their walls were thickened, etc. The ventricles contained a considerable quantity of fluid. There was great venous congestion beneath the middle lobe of the left hemisphere; the cerebral veins in that situation were distended with conglum. and their coats were thickened. In the left middle lobe, towards its anterior part, were also four recent extravasations of blood, each of which was connected with an obstructed and distended vein. The largest clot extended an inch into the cerebral substance; the others were of smaller extent.

The cerebral hæmorrhage constitutes an important feature of this case. It was obviously due to the occlusion of certain of the cerebral veins with thrombi. The blood brought into the brain by the arteries escaped into the cerebral substance, because it could not find its way out again through the obstructed veins. Moreover, this hæmorrhage was, in all probability, the immediate cause of the faintness or syncope which terminated the patient's life.

Concerning the diagnosis of cerebro-venous thrombosis Dr. West justly observes that we are not acquainted with any symptoms which are pathognomonic of this affection; but when head-symptoms suddenly present themselves in debilitated or cachectic children and do not run the course of any ordinary form of cerebral disease, such symptoms will probably be found to be due to the formation of blood-clot in the cerebral veins or sinuses.

Dr. Boeckb, an eminent authority on the subject of thrombosis, relates (*Journ. f. Kindk.*, 1868) the two following cases, which serve to still further illustrate the clinical relations and phenomena of the morasmic variety of cerebro-venous thrombosis when it occurs in children:—

CASE LVII.—A little girl had suffered for several months from whooping-cough. Broncho-pneumonia ensued; incomplete anæsthesia attended, which quickly deepened into a condition resembling asphyxia. Then convulsions, lasting four hours, set in, were repeated twice on the same day, and led to death. The autopsy revealed considerable brain-congestion, and a small quantity of serous effusion in the pia mater under the arachnoid; besides, old conglum. were found in the sinuses of the dura mater; these thrombi were hard and colourless, and one was fifteen centimetres in length; it extended along the lateral sinus, and reached the mouth of the jugular vein. Another thrombus, still harder and paler, lay in the superior longitudinal sinus. It had grown fast to the walls and completely obliterated the sinus.

CASE LVIII.—Another case was that of a phthisical child, in whom thrombosis of the cerebral sinuses induced delirium twelve hours before death. The ophthalmoscope revealed a gray papilla, surrounded apparently by œdema. The boundaries were not recognizable, and were rather surmised from the puncta around which the veins radiated. The vessels were large and pale; the choroid also was very pale, and, like the retina, looked as if covered with fine white sand. The cause of the delirium was sought for in the sinuses of the dura mater, and the associated atrophy. The accuracy of this diagnosis was confirmed by autopsy. Tubercles and cavities were found in the lungs. The longitudinal and transverse sinuses of the dura mater were completely closed by blood-plugs, of which some were old, whitish, firm, and more or less adherent to the walls, whilst others were still fresh, blackish, soft, and had clearly been formed in the last moments of life. The brain and its membranes were sound. On examining the eyes there was seen, through the retina, cluse around the papilla, a rosy areola beset with red puncta. After removing the retina it was easily seen that the vessels of the choroid were filled with blood. The inner layer of this membrane had quite vanished, a few cells only were seen, and these had degenerated into fat. The pigment-cells of the lamina were quite gone, or were atrophic. The sand-like appearance seen with the ophthalmoscope before death was only an optical illusion, and was plainly due to the want of pigment in the choroid. (*New Sydenham Soc. Retrospect*, 1867-1868, pp. 426, 427.)

The latest clinical observations of Bochat teach that, in cases where, after long chronic diseases, children die in convulsions or delirium, we should look for thrombi in the great veins of the brain and its membranes. He also recalls to mind how many chronic diseases of children terminate in convulsions. As a rule, they are the forerunners of death. Many children thus die in the course of mumps brought on by lung-phthisis, simple, chronic, and tubercular enteritis, vertebral caries, white swelling of the joints, hooping-cough complicated with broncho-pneumonia, etc. It was long believed that the delirium and convulsions which appear at the end of such chronic diseases were simply the result of impoverishment of the blood. Most physicians think that inanition and chlorosis are the consequences of the cachectic state, and that the cachexia itself is developed through the protracted disease of the infant organism. This is generally true, and Bochat recognizes the correctness of Marshall Hall's explanation of hydropcephaloid.

But there are cases, he says, such, for example, as the above, in which the loss of consciousness and convulsions result from a quite different cause. In them the formation of thrombi in the sinuses of the dura mater, which obstruct the flow of blood from the brain, produces passive congestion of the brain-substance, and impaired nutrition of the nerve-fibres and ganglion-cells. For when the venous blood does not flow out of the cerebral capillaries the fresh arterial blood which is necessary to the performance of the function of nutrition cannot pass into the cerebral capillaries so as to reach the histological elements of the cerebral substance and maintain their functional activity and integrity. Whenever this form of passive congestion of the cerebrum is excessive and not compensated for, the ganglion-cells and nerve-filaments of the cerebrum lose their functional activity, and loss of consciousness ensues; and the loss of consciousness may be partial or complete; it may be stupor on the one hand, or coma on the other, according to the degree of the passive con-

gestion, or the completeness with which the escape of venous blood from the brain-substance is prevented by the presence of coagula in the cerebral veins and sinuses of the dura mater. Finally, in some cases where at first the passive congestion of the brain-substance is not severe enough to produce stupor or convulsions, it may so modify the nutrition of the brain-substance as to induce delirium.

Dr. M. E. Fritz (*Bulletins de la Société Anatomique de Paris*, 1860, pp. 70-73) relates a very instructive case of marasmic thrombosis of the cerebral veins and sinuses of the dura mater, of which the following is an abstract:—

CASE LIX.—A boy, aged 11, an inmate of the Hôpital des Enfants, had Pott's disease of spine, with profuse purulent discharge, and was becoming much debilitated, when he was suddenly seized, August 28th, with vomiting, headache, and drowsiness. On August 29th he was found in bed lying on his back, with shrunk features, moaning, and unconscious; eyes convulsed, axes divergent, pupils dilated; lower extremities contracted; pulse slow and irregular.

August 31. Profound coma; afterwards profuse sweats limited to face, neck, and upper part of chest.

September 4. He had convulsions in the morning, and died comatose at 8 in the evening.

Autopsy.—Brain very soft, almost diffuent; arachnoid raised up by a serous fluid, containing some gelatinous flocculi. Four or five cerebral veins (superior and anterior, on both sides) were distended and plugged up to the extent of several centimetres, next to the sinus, with blackish-brown, tolerably dry, and elastic coagula; cerebral veins generally much gorged with blood. The vein-clots were continuous with a thrombus which occupied the superior longitudinal sinus through its whole length. In anterior half of sinus the thrombus was dark-brown; back of that, grayish-white, spotted with red externally, and of the colour of wine-lees at the centre, adhering pretty closely by its outer layers to the fibrous bands of the sinus; still further back it became thin, no longer filling up the calibre of the sinus, and its extremity was prolonged a little into the torcular Herophili. The walls of the sinus nowhere presented an inflamed appearance, being smooth and not injected. The left lateral sinus also contained a small coagulum, similar to the above.

The light-coloured part of the thrombus, under the microscope, was found to consist of granular fibrin, mingled with a great number of leucocytes, some oil-drops, and red globules more or less altered. These facts showed that the conglutination was not very recent.

Dr. M. E. Fritz (*Ibid.*, pp. 75-77) relates another very interesting case of infantile marasmic thrombosis of the cerebral veins and sinuses of the dura mater. The following is a condensed account of it:—

CASE LX.—A little boy, aged 3½ years, entered the Hôpital des Enfants January 5, 1859, for prolapsus of rectum of six months' standing. Badly nourished and living in misery he had had measles and smallpox in the last six weeks; and a few days before admission he was seized with whooping-cough. He was very pale and feeble, but without fever; the fits of coughing were pretty frequent, and terminated by vomiting. January 7. Symptoms of capillary bronchitis. January 8th and 9th. He was extremely prostrated and unable to sit up; face extremely pale. On the evening of the 9th he became delirious. On the morning of the 10th he was much sicker, and did not reply to any question. His general sensibility was blunted, but not extinguished; breathing difficult and oppressed. During the day strabismus appeared. In the evening pulse 180 and very small. He died on the 11th.

Autopsy.—Visceral arachnoid considerably elevated by serum. Cerebral veins gorged with black blood; superior longitudinal sinus contained, besides some liquid blood and soft black coagula, a fibrinous concretion which extended

its whole length. It adhered to the walls, which were entirely normal, by some prolongations that entwined themselves in the trabeculae of the angles. It nowhere completely obliterated the calibre of the sinus. At the posterior extremity it was cylindrical and had a diameter of four millimetres; at the middle it was ribbon-shaped or flattened; at the anterior extremity it terminated in a point. It sent prolongations into many veins along the sinus. The calibre of these veins was thus almost completely obliterated. The right lateral sinus was occupied by a similar concretion, nine centimetres long by about four millimetres thick. The middle lobe of right lung presented numerous spots of atelectasis, accompanied by capillary bronchitis with thick purulent secretion.

These two cases differ from each other very much in respect to the symptoms referable to cerebro-venous thrombosis which presented themselves during life. In the first of them the attack came on suddenly with vomiting, headache, and drowsiness. There followed convulsive movements of the eyes, with dilated pupils and divergent strabismus, profound coma, general convulsions, and death. In the other, the earliest symptom was delirium. Towards the end, however, strabismus appeared, but there were no general convulsions. These cases also differ from each other equally much in respect to the coagula which were found in the cerebral veins and sinuses of the dura mater, on autopsy; for in the first case the thrombus was large enough to fill up the calibre or completely obstruct certain of these vessels, while in the other the thrombus was not large enough to entirely occlude any of these canals. Dr. Fritz's cases, therefore, strongly support the views of Bouchut, expressed above.

These thrombi also differed from each other a good deal in other particulars besides their size, which will readily suggest themselves to the reader, and thus render it unnecessary to spend much time in their discussion in this place. One point, however, requires special mention, namely, in the first of these cases the great thrombus which occupied the superior longitudinal sinus was unmistakably formed at two distinct epochs. In the anterior half of this sinus it was dark-brown in colour, and resembled a recent blood-clot in appearance. In the posterior half it was grayish-white in hue, and much older in appearance, having existed long enough to become decolourized, as was shown by the broken-down and disintegrated condition of the red corpuscles belonging thereto, which was revealed by the microscopical examination. Besides, it is worthy of remark, that the walls of the thrombosed vessels did not show any signs of inflammatory action in either case.

Dr. Crisp (*Transact. Patholog. Soc. of London*, vol. x. pp. 117, 118) has related the following remarkable instance of what appears to be idiopathic thrombosis of the cerebral veins and sinuses of the dura mater:—

CASE LXI.—The patient was a girl, aged 16, who died after about fourteen days' illness. The symptoms were headache, confusion of intellect, and vomiting, followed by hemiplegia of right side, loss of speech and of power to protrude the tongue, and inability to pass water. Death was preceded by coma. No cause far her attack could be assigned.

Autopsy.—The superior longitudinal sinus was filled with coagulated blood,

interspersed with portions of fibrin closely adhering to the walls thereof. The superior cerebral and cerebellar veins were also extensively thrombosed. The superficial cerebellar veins were nearly all plugged with fibrinous coagula, and all the cerebral veins entering the superior longitudinal sinus were firm and cord-like from the same cause. The upper and lateral portions of the arachnoid were opaque on both sides. The pia mater was rather vascular. The lateral ventricles contained about two teaspoonfuls of reddish serum, and about the same quantity was found under the arachnoid at the upper part of the spinal cord.

The autopsy does not appear to have revealed any cause to which the death of this young girl could be ascribed, other than spontaneous coagulation of the blood in the veins and sinuses belonging to her brain. How does cerebro-venous thrombosis arise in such cases? First of all, the blood itself must be in a condition to readily coagulate, that is, the blood must be more coagulable than natural, or in a morbid state whereby its coagulability is greatly increased. Besides, a protracted stasis of the blood in these vessels, such as might readily attend a passive congestion of the brain, and perhaps also an expanded condition of the cerebral veins and sinuses, such as might be induced by a vaso-motor paralysis of these vessels, would, if present, determine the occurrence of the coagulation. It is not impossible that all these causes combined to produce the thrombosis in the above case.

Dr. Andrew (*Transact. Patholog. Soc. of London*, vol. xvi. pp. 27, 28) has reported an exceedingly interesting case of cerebro-venous thrombosis, which in many particulars resembles the one last related. It illustrates in a striking manner the clinical history and post-mortem appearances of the morasmic variety of this disorder when it occurs in oemic young women.

CASE LXII.—Eliza S., aged 20, admitted to St. Bartholomew's Hospital March 29, 1865; had enjoyed good health up to twelve months before, when, without apparent cause, her catamenia suddenly ceased, and she began to suffer from anæmia. On admission she was very feeble and anæmic; ankles cedematous; pain in left side; anæmic murmurs in neck, etc. She complained of slight headache, at first frontal, which gradually became more intense, so that on the night of April 3d it kept her awake. On April 4th she vomited several times, and was delirious during the night. On the 5th she gradually became insensible and comatose; pulse 90–120, and slightly irregular. On the 6th, at 10½ A. M., she died.

Autopsy.—Cerebral convolutions flattened, particularly on right side, and marked by impress of the fibres of dura mater. A dark clot, the size of a pea, in posterior part of each hemisphere about half an inch from upper surface. Considerable blood-stained fluid in each lateral ventricle, also a long thin dark clot in right one, lying upon the corpus striatum and optic thalamus. Septum lucidum entire, but soft. Optic thalami unusually prominent; on section, they are found to be cedematous, and studded with numerous small dark clots, by which their substance is broken down, but these changes are more marked in the right than in the left one. The veins of the choroid plexus and velum interpositum, also the venæ Galeni, are distended with firm, partly yellow, fibrinous coagulum. It extends continuously along the straight sinus, and about an inch into the lateral sinuses, but rather further down the right than the left one. It does not completely fill up the calibre of the lateral sinuses, whilst the straight sinus and the tributary veins are greatly distended thereby. The oldest part of the thrombus was found at the junction of the

straight with the lateral sinuses. Here it was of a dull pinkish tint, and somewhat soft. In the other sinuses the blood was almost entirely fluid. Cerebral arteries not diseased. Cerebral substance throughout rather soft and watery. No tubercle in any organ. In each lobe several branches of the pulmonary artery were obstructed by old clots, some of which were breaking down at the centre. Beside the endocardium of left ventricle (which was firmly contracted) were numerous large ecchymoses.

The symptoms referable to thrombosis of the cerebral veins and sinuses of the dura mater, which this patient exhibited, were headache, gradually increasing in severity, vomiting, delirium, stupor, coma, and death. Moreover, this disorder appears to have run its whole course in three or four days.

The autopsy revealed cerebral hemorrhage, cerebral œdema, ventricular effusion, and obstruction of the veins of Galen, velum interpositum, and choroid plexus, as well as the straight and lateral sinuses with fibrinous coagulum. But the oldest part of the thrombus was found at the junction of these sinuses. This circumstance shows that the coagulation began in a portion of the cerebral sinuses where the calibre is widely expanded, where the blood-stream is usually sluggish, and where the blood itself would be likely to stagnate on lessening much the power of the heart and arteries to carry on the circulation. The extravasations of blood, which, by the way, were very numerous, the œdema of the cerebral substance, and the effusion into the ventricles, were clearly due to the blocking up of the straight sinus and the tributary veins with thrombus.

It is also worthy of special mention that this young woman was very pale, feeble, and anæmic; that her ankles were œdematous; and that she had anæmic murmur in the neck ere she was attacked with cerebro-venous thrombosis. In other words, the occurrence of this disorder was preceded by undoubted evidence of blood-disease. In all probability this blood-disease was the principal cause of the thrombosis; for the anæmia or chlorosis doubtless was attended with much diminution in the quantity of red corpuscles and much increase in the white ones, and from this change in the composition of the blood there resulted a considerable increase in its coagulability.

Again, anæmia and debility the result of *excessive bloodletting* sometimes lead to coagulation of the blood in the cerebral veins and sinuses of the dura mater, especially in persons who are predisposed to this disorder. It is scarcely necessary to say that these cases are very important in a practical point of view. Von Dösch presents two rather striking instances, of which the following is a brief account:—

A young woman, aged 23, was attacked by peritonitis twice during the first week after delivery, for the cure of which copious local bleeding was repeatedly employed during nine days. Twice, forty leeches were applied, and, on two other occasions, twenty, making in all one hundred and twenty leeches. A fortnight after delivery headache and vomiting appeared, followed by hemiplegia. Great restlessness and screaming supervened, then coma, and finally, death three weeks after delivery. It must be added that, in consequence of the new symp-

toms, she was bled once in the arm, and had fifteen more leeches applied on various occasions.

Autopsy.—The superior longitudinal sinus was seen to be very much distended, and of a glittering blackish appearance. It was filled up by a thrombus, in the centre of which there was a puriform fluid, resembling wine-lees. The right lateral sinus also was blacked up, in the direction of the jugular vein, by a firm coagulum which contained a similar fluid. The other sinuses were quite normal. There were ecchymoses in the gray matter on the surface of the brain, especially in the course of the thrombosed veins on the convex surface and at the base. In the true pelvis two collections of pus were found, and, in the veins of the uterus, corresponding to the place where the placenta was attached, very firm, small, black plugs. In the chest there was nothing abnormal (*op. cit.*, 108).

In this case thrombosis of the cerebral veins was the cause of death. It occurred because the coagulability of the blood was greatly increased on the one hand, and the heart-power was greatly diminished on the other. The abnormal coagulability of the blood, however, was due in part to the puerperal condition of the patient, and in part to the excessive losses of blood by leeches and venesection to which she was subjected, for it is well known that the puerperal state is usually attended with a considerable increase in the coagulability of the blood as a consequence of hyperinosis, and that copious bloodletting generally causes a great increase in the coagulability of the same fluid. But excessive bloodletting also weakens the action of the heart, and thus assists more or less strongly in producing cerebro-venous thrombosis in such cases as the foregoing.

The other case claims affinity to the last in one important respect, namely, in it the formation of thrombus in the sinuses of the dura mater was also due, to great extent, to colossal bloodletting.

A soldier received a gunshot wound of the left parietal bone, which produced fracture with depression. The symptoms which supervened rendered trephining necessary. The patient was also bled five times in a short period. Death occurred on the thirteenth day.

Autopsy.—Fleshy coagula were found in the left lateral sinus, and smaller ones of similar appearance in the superior longitudinal and right lateral sinuses. There were also fracture of the inner plate and purulent osteitis (*op. cit.*, pp. 109, 110).

If in this case the formation of the thrombus had been induced by the arachnitis, one or more of the contiguous veins of the pia mater would pretty certainly have been found thrombosed, and, in all probability, the thrombus itself would have been discovered to be broken down, and undergoing puriform softening, as usually obtains in venous thrombosis connected with suppurating parts. It is therefore highly probable that the arachnitis had but little, if anything, to do with the production of the thrombosis. However, the injury itself, and the operation of trephining, doubtless were concerned in producing it; but this circumstance should not lead us to overlook the influence which five venesections, all performed within a short period, must exert in the way of increasing the coagulability of the blood, thereby promoting the occurrence of venous thrombosis in the neighbourhood of the injured part; besides, these venesections most

have weakened the heart-power considerably. We must therefore look upon excessive loss of blood by venesection as an important, if not the chief, cause of the thrombosis of the cerebral sinuses which occurred in this case, and its importance, in a practical point of view, is not diminished by the fact that it was entirely preventable.

The *syphilitic cachexia*, and syphilitic diseases of the brain or its membranes, are sometimes attended by the formation of conglua in the cerebral veins and sinuses of the dura mater, whereby these vessels become more or less completely obliterated. Von Dusch does not mention this form of cerebro-venous thrombosis. It is, therefore, incumbent upon us to give particular attention to this point. Dr. Marchison (*Transact. Patholog. Soc. of London*, vol. xiii. pp. 250-253) has reported two cases, one of which certainly, and the other probably, comes under this head. The following is a much condensed account of them :—

CASE LXIII.—The subject was a woman, aged 27, who died of syphilitic cachexia, at the Middlesex Hospital. After complaining of vertigo, dimness of sight, and headache for a time, she got epileptic fits, but her consciousness remained clear almost to the last. On *autopsy* there were found extensive syphilitic deposits on the dura mater, moderate injection of the pia mater, numerous anastomosing vessels, and thrombosis of the sinuses of the dura mater, in addition to other lesions. The sinuses were full of dark soft coagulam.

The writer infers from the symptoms which presented themselves during life, and from the appearances which the thrombus exhibited at the autopsy, that it was formed during the closing hours of life. Nevertheless, it may have played an important part in producing the fatal result.

CASE LXIV.—The other patient was also a woman, aged 36, who, after having had repeated attacks of copper-coloured eruptions, periostic nodes on cranium, intense pain in forehead and occiput aggravated at night, was attacked with "fits" and died a fortnight later at the Middlesex Hospital. The "fits" commenced with a scream, and were attended with loss of consciousness, but not by foaming at the mouth, nor by convulsive movements. The "fits" increased in frequency and severity until she died, while, in the intervals, she had intense pain in the forehead and occiput.

Autopsy.—Pericranium thickened in spots. Bones of skull generally thicker and denser than natural, etc. While removing the brain the left hemisphere of the cerebellum was found so firmly adherent to the dura mater, that a portion was torn off and left behind. The dura mater in rear of the petrous portion of temporal bone, and on the occipital as far as the mesial line, was greatly thickened by the deposit on its inner surface of flattened masses of a firm yellowish-white substance, exhibiting a smooth surface on section, and yielding no juice. This deposit extended at some places fully half an inch under the tentorium at its attached margin. The left lateral sinus passed through the diseased mass, and its canal was quite obliterated. There was an evident connection by continuity between the disease of the dura mater and the periostic nodes external to the bone.

It seems highly probable to the writer that in this woman's case the obliteration of the left lateral sinus was effected through the agency of a thrombus, and that the formation of the thrombus itself was due in part to syphilitic pachymeningitis and in part to syphilitic cachexia.

Analogous in some respects to the last group are those cases wherein a *fungous tumour of the dura mater* causes the blood to coagulate in one or more of the cerebral sinuses.

Mr. Forster (*Transact. Patholog. Soc. of London*, vol. ii. pp. 162, 163) has recorded the following case in which thrombosis of the right lateral sinus resulted from the above-mentioned affection of the dura mater:—

CASE LXV.—A strumous lad, aged 18, was knocked down by a cub, and struck on the right side of his head sixteen months before death. Soon afterwards he became deaf and suffered severe pain. The part became slightly swollen and excessively tender, especially over the mastoid process; paralysis of the right facial nerve also took place. No great change occurred until the last six months, when from another blow on the same spot, the disease grew more active. The side of his head from above the temporal ridge to two inches below the ear, became enormously enlarged and tender; the external ear appeared to be pushed away from the side of his head. He experienced great difficulty in swallowing solid food, and was also unable to speak. About two months before death the swelling began to fester and slough. Profuse hemorrhage occurred at intervals. The sloughing extended very rapidly, and at last laid the pharynx bare. No brain symptoms appeared. The hemorrhage and the suppuration, which also was profuse, quickly destroyed him.

Autopsy.—The tumour which caused this swelling was developed from the dura mater. The temporal bone was curious and extensively destroyed in consequence of the pressure exerted by the tumour. No vestige of meningitis or mastoid cells could be found. The lateral sinus was filled with a coagulum.

No head-symptoms resulted from the thrombosis in this case because it was limited to one lateral sinus, and, therefore, the blood could for the most part, readily find its way out of the brain through the other lateral sinus, etc.

In cases such as this it has been customary to ascribe the formation of thrombus to compression of the affected sinus resulting from the growth of the tumour. Von Dusch (*op. cit.*, p. 103) quotes a case from Virchow, in which there was a large cholesteatoma in the left petrous bone, and at the same time thrombosis of the left lateral sinus, etc.; but the patient had also had otorrhœa for twelve months, and when we consider the intimate relationship which has in another place been shown to exist between disease of the internal ear and thrombosis of the corresponding lateral sinus, we are led to refer the causation of the thrombosis in Virchow's case to the ear-disease quite as much as to the tumour. Indeed, Von Dusch perceived this point, for his remarks that although the formation of thrombus in the cerebral sinuses as a consequence of compression, etc., seems *a priori* very probable, "the few cases to be met with in the literature of the subject for the most part indicate other sources." So too in the case related above, the writer thinks that the formation of the thrombus should be attributed to the morbid state of the patient which resulted from the hemorrhages and the suppuration, rather than to any diminution in the calibre of the sinuses which may have been produced by the fungous growth.

Anæmia and debility, the result of chronic disease of the rectum, sometimes proves fatal by inducing cerebro-venous thrombosis. Dr. Ogilvie (*Transact. Patholog. Soc. of London*, vol. vi. pp. 31, 32) has reported a case of this sort, in which several cerebral veins and sinuses of the dura mater were occluded by dense fibrinous clots that were traceable to a spon-

taneous tendency in the blood to the deposition of fibrin. The following is a brief abstract thereof:—

CASE LXVI.—The patient was a woman, who gradually sank in St. George's Hospital, whither she had been brought for long-continued disease of the rectum. The symptoms exhibited towards the close of life were those of asthenia, and nothing pointed to disease of the brain, excepting that a short period before death, she completely lost the faculty of speech, the mind being unaffected.

The *autopsy* showed the presence of much blood-stained fibrin which blocked up the left lateral sinuses, the inferior longitudinal sinuses to a certain extent, the straight sinuses, most of the venæ Galeni, the left petrosal sinus, and several veins which pass into the above-mentioned sinuses from the sides and base of the cerebral and cerebellar hemispheres. The coagulum did not extend beyond the posterior jugular foramen. The subarachnoid tissue and the ventricles contained much fluid, but the substance of the brain and its membranes were natural. No marked lesion of any organ excepting the rectum was found, and no collection of purulent matter existed in the body.

Other exceedingly chronic diseases may in adults, as well as in children, as we have elsewhere shown, so far as the latter are concerned, bring about a state of marasmus with increased coagulability of the blood and the formation of thrombi in the cerebral veins and sinuses of the dura mater. Von Dusch (*op. cit.*, pp. 107, 108) presents a case in point, wherein a man, aged 53, who had old hepatic and renal disease, with cough and copious expectoration, ascites, anorexia, and loss of strength, got diarrhoea with involuntary evacuations, accessions of unconsciousness, and thus died. On *autopsy* there were found cirrhosis of the liver, degeneration of the kidneys with cysts and concretions, thrombosis of the superior longitudinal sinus and of some of the corresponding veins of the pia mater, etc. The lungs were œdematous, and the pulmonary arteries contained obstructive plugs. Again we remark that any chronic disease which produces a cachectic state, and increases the coagulability of the blood as well as weakens the action of the heart and arteries, may in like manner lead to cerebro-venous thrombosis in adults as well as in children.

But diseases which run a comparatively short course—diseases which are by no means chronic—such for example as pneumonia, pleurisy, typhoid fever, etc., may also cause the blood to become abnormally coagulable and clots to form in the cerebral veins and sinuses of the dura mater in both adults and children. The following case reported by Dr. Ogle (*Transact. Patholog. Soc. of London*, vol. x. pp. 30, 31) comes under this head:—

CASE LXVII.—A man, aged 26, entered St. George's Hospital for pneumonia of left side (a relapse?). His pulse was very feeble and debility great. Afterwards he complained of intense pain in the head, especially in the back part thereof, and subsequently in the left temple. He was treated with wine and other stimulants. About five weeks after admission, he was found one morning in bed totally unconscious, and deprived of all power of moving the left arm and the left leg. He lay three days in a state of half-stupor and then died.

Autopsy.—The superior longitudinal, left lateral, and left petrosal sinuses were found plugged up by firm, and for the most part reddish-brown coagulum, which generally adhered pretty firmly to their walls; but in one or two places

was diffident and broken down into a grayish-brown grumous fluid. Several veins of the pia mater, both small and large, which empty into the sinuses, contained brown adherent clots, but none of them were softened. The veins on the right cerebral hemisphere were very much engorged. The left arachnoid sac contained a considerable amount of yellowish purulent fluid mixed with soft fibrinous material. Brain-substance slightly softened at the posterior-inferior part of middle lobe of left cerebral hemisphere. An abscess about the size of a hazel-nut, existed about one-third of an inch from the surface at this spot. A large amount of turbid fluid in the lateral ventricles. The lungs showed evidences of recent pneumonia. In one or two places the pulmonary tissue had given way, accumulations of purulent matter having formed.

Von Dasch mentions an analogous case:—

The patient was a servant girl, aged 20, who was attacked with pleuro-pneumonia while suffering from typhoid fever. Contracture of the cervical muscles, convulsions of right side, and coma supervened; death ensued. The cerebral veins, superior longitudinal and lateral sinuses were found extensively obstructed with firm adherent coagula.

In both of these cases the cerebro-venous thrombosis which was developed after the occurrence of pneumonia may fairly be regarded as a result of marasmus, unless we consider the cerebral affection to be metastatic, an assumption, however, which appears much more forced.

The case of a young girl, aged 12 (already referred to), who died of a cerebro-venous thrombosis which came on at an advanced stage of typhoid fever, belongs to the same category as the above. In this case clonic and tonic convulsions, with loss of consciousness, but not of sensibility, suddenly supervened. These phenomena lasted an hour. A fresh accession soon occurred, with trembling of the muscles which lasted till death. The convulsions were followed by coma and contracted pupils. On *autopsy* the whole of the superior longitudinal sinus was found obstructed by a conglum, everywhere adherent and partially decolorized, as also the veins of the pia mater communicating with it, etc.

Again, thrombosis of the cerebral veins may occur in connection with thrombosis of the veins of the extremities and pyæmia, as it did in the case which Dr. Jneway brought before the New York Pathological Society, April 8, 1868 (*Medical Record*, July 15, 1868).

CASE LXVIII.—The patient was a news-boy, aged 16, who entered Bellevue Hospital on the morning of April 4th, giving an imperfect history to the effect that three days before he observed on waking in the morning an "injury" to his right forearm, near the wrist, and saw a physician that day who applied splints. On entering the hospital these were removed and evaporating lotions applied. At that time he exhibited no cerebral symptoms, but in the evening he was slightly delirious, though he still walked about the ward. The next morning his condition was about the same; towards night, however, he became comatose, and so remained until death occurred at 10 P.M. the following day, April 6th. While in this state his pulse was 120 to 130, and respiration about 40.

Autopsy.—Forearm and hand œdematous. A fluctuating tumour at the wrist contained a quantity of sanious pus, that had burrowed among the tendons down to the bone; periosteum thickened and vascular, but not detached. The outer one of the radial venæ comites contained, in the middle of its course, a firm clot about two inches in length; the inner one, a whitish clot of soft consistency from commencing disintegration. The basilic and axillary veins, with the

brachial venæ comites, were distended throughout their course with soft coagulum. On the brain, chiefly on its convexity, many small opaque spots were seen in the arachnoid and pia mater, the result of thickening. Each of them was surrounded by a red border, due sometimes to congestion, sometimes to extravasation. The brain-substance showed several points of hemorrhage. *Three of its larger veins contained attached fibrinous clots*, evidently formed some time before death. In the lungs were hemorrhagic infarctions and so-called metastatic pneumonia and pleurisy. In the left ventricle were many fresh signs of ulcerative endocarditis. Numerous small white points surrounded by red areolæ, similar to those found on the arachnoid, were also found on the exterior of the heart, on the liver, in the kidneys, and small intestines. Prostate, bladder, etc., normal.

This boy's illness was of less than six days' duration. It ended fatally by coma, that is, death began at the brain. The case itself is an unusual one, and possesses three features of intense interest and great importance. They are, *firstly*, the abscess at the wrist with the accompanying thrombosis of the veins of the forearm, etc., and the pyæmic lesions of the lungs; *secondly*, the ulcerations and other changes in the left ventricle of the heart, and the multitude of white points surrounded by inflamed areolæ which were found on the surface of the brain, liver, kidneys, small intestines, and heart itself, that probably had been produced by the lodgment in the capillary arteries of these parts of minute emboli that had been washed out of the left ventricle by the blood-stream; and, *thirdly*, the obstruction of several cerebral veins of large size, with thrombus, and the occurrence of coma and death in consequence of this obstruction. It is probable that the patient would have lasted some time longer if thrombosis of the cerebral veins had not taken place. Moreover, the plugging up of these vessels was attended with the extravasation of blood at several points in the brain-substance. This case, then, serves an admirable purpose for illustrating thrombosis of the cerebral veins, thrombosis of the veins of the extremities, and capillary embolism, the result of ulcerative endocarditis.

Lastly, we have to point out that thrombosis of the cerebral veins and sinuses is an occasional consequence of *senile marasmus*. Cruveilhier (*Anat. Patholog. du Corps Humain*, liv. 36, pp. 2, 4, 5) relates two cases of this sort. One of them was that of an old woman of weak intellect, who died after twenty-four hours' coma. The superior longitudinal sinus and the veins communicating therewith were filled with a brilliant, black, adherent thrombus, and the gray substance of the cerebral hemispheres contained numerous capillary hemorrhages, etc. The other case was that of a woman, aged 80, who after having paralysis of the left half of the body, excepting the tongue and face, for some time, got paralysis of the right side, and thus died. The superior longitudinal and lateral sinuses were blocked up by thrombus, and the superior cerebral veins were also filled with plugs. In the arachnoid sac a fresh extravasation of blood was spread out over the convex surface of both hemispheres, etc.

A case related by Dr. Charcot (*Boucharde on the Pathology of Cerebral Hemorrhage*, pp. 16, 17, London, 1872) is in point. In it, however, the cerebro-venous thrombosis was due in part to senile marasmus, and in part to the depressing influence of disease.

CASE LXIX.—A female, aged 65, had pneumonia in 1858, and again in 1863. In the summer of 1865 she was under treatment for cirrhosis and ascites. On August 21st, she suddenly became paralyzed on the right side. On the 23d, at 2 A. M., she became comatose; at time of visit her breathing was stertorous, pulse small and frequent, skin warm. The limbs were all flaccid; reflex movements on both sides abolished. At 4 P. M. she died.

Autopsy.—Extensive subarachnoid hemorrhage on both cerebral hemispheres, more abundant, however, on the right than on the left one. Dark-colored very friable clots filled up each lateral ventricle. The floor of the left one was the seat of yellow softening throughout. The left optic thalamus presented a reddish pulp on its surface, consisting of blood mixed with softened cerebral tissue. A patch of red softening was also found on the left hemisphere behind the convolution which bounds the fissure of Rolando. The gray matter had the color of wine-lees, and the white substance beneath was softened and slightly yellowish. Another patch of red softening was found on the right hemisphere behind the fissure of Rolando, but it was smaller than the preceding. The arteries of the base were not atheromatous. On the surface of the clots which covered the inner aspect of both cerebral hemispheres two veins that emptied into the superior longitudinal sinuses were seen to be stretched; they were yellowish in color, and contained old clots. Liver cirrhotic; peritoneal cavity distended with yellowish serum.

The cerebral hemorrhage and cerebral softening were doubtless occasioned by thrombosis of the cerebral veins. It is also worthy of remark that cerebral or meningo-cerebral hemorrhage occurred in both of the other cases of senile thrombosis of the cerebral veins and sinuses of the dura mater mentioned above. From this circumstance we infer that when cerebro-venous thrombosis occurs in old persons, it is especially prone to occasion cerebral hemorrhage, and this relationship may be accounted for by the fact that the walls of the bloodvessels in general, usually become weakened by atheroma, fatty degeneration, etc., with advancing age.

But, in the last case, the longitudinal and other sinuses of the dura mater did not contain any coagulum; and thus it is conclusively shown that thrombosis of the cerebral veins, attended with very striking morbid changes and consequences, may occur without there being any thrombosis of the corresponding sinuses of the dura mater. In Dr. Joneway's case LXVIII, also, certain of the cerebral veins were obstructed with thrombus while the sinuses remained free, and still cerebral hemorrhage was present. The reader's attention is specially called to this point, because it is a new one, and possesses considerable interest, at least for pathologists.

Etiology.—The clinical histories related above, and in the preceding paper, show pretty clearly that in one set of cases the blood congeals in the sinuses of the dura mater as an immediate consequence of cranial injury, especially when the sinus-walls themselves are implicated, that in another set of cases the blood congeals in these vessels as a result of various inflammatory affections of the head, internal ear, eye-socket, nose-cavity, and

face, and that in still another set of cases the cerebro-venous thrombosis is occasioned by wasting diseases or debilitating influences. Thus, we find that this disorder presents three important varieties, in respect to causation, namely, the *traumatic*, the *inflammatory*, and the *marasmic*. Some of them, however, occur much less frequently than others. For example, in looking over the 58 cases presented by Von Dusch, I find but 2 in which the origin was directly traumatic, while in 34 the causation was inflammatory, and in 22 marasmic. Besides, out of 72 additional cases collected by myself the thrombosis had a traumatic origin in only 4 instances, an inflammatory starting point in 52, and a marasmic origin in 16 patients. Finally, in a grand total of 130 cases, thus obtained, we have but 6 in which the coagulation resulted directly from traumatic causes, 86 from inflammatory lesions, and 38 from marasmic conditions, or debilitating influences. From this it appears that the inflammatory is by far the most common form of cerebro-venous thrombosis, inasmuch as it has been met with considerably more than twice as often as the marasmic, and rather more than fourteen times as often as the traumatic. Dr. Lancereaux, however, states that of 74 cases collected by himself 39 were inflammatory and 35 non-inflammatory in respect to origin; but he has doubtless overlooked many of the inflammatory cases which have rewarded my researches, and therefore his statement does not disprove the conclusion arrived at above.

1. *Causes of the Traumatic Variety.*—Concerning the formation of the coagulum which are occasionally met with in the sinuses of the dura mater after injuries of the skull, Virchow has already pointed out that in some cases the coagulation probably begins in the wounded veins of the diploë as a consequence of the atmospheric air coming into contact with the blood at the gaping orifices in these vessels; for the nature of the veins of the diploë is such, and their walls are so firmly attached to the surrounding bone, that they cannot collapse, and, therefore, in cases of compound fracture of the cranium, with depression, gaping apertures may readily present themselves in the veins of the diploë through which a hemorrhagic thrombus formed externally to them may easily extend into their calibre, and thence by further prolongation may penetrate the corresponding sinus of the dura mater. In other cases, however, the formation of coagulum in the sinus is due to the fact that some foreign body, such, for example, as a fragment of the inner table of the skull, has been driven into the calibre of the sinus, and that the blood, on coming into contact with this foreign body, coagulates around it, as crystals form around foreign bodies when suspended in crystallizable liquids. This is precisely what happened in a case quoted from Schmucker by Von Dusch, wherein a splinter from the vitreous plate half an inch long penetrated the superior longitudinal sinus, and that vessel soon became plugged up with a firm

thrombus, which afterwards, to a considerable extent, underwent puriform disintegration or softening.

But thrombosis of the cerebral sinuses does not occur in all the cases wherein the part of the cranium which overlies the vessels in question is fractured in such a manner as to expose the lacerated veins of the diploë to atmospheric action, nor in all the cases wherein a foreign body is driven into the calibers of a sinus, nor in all the cases wherein the walls of a sinus happen to be wounded. On the contrary, it occurs in only a small portion of these cases, as is clearly shown by the records of clinical experience, such, for example, as M. Lassus's *Memoir on Wounds of the Superior Longitudinal Sinus* (already referred to), various of the treatises on cranial fracture, the current annals of clinical surgery, etc.

Now, why does thrombosis occur in some cases where the sinuses of the dura mater, or the parts covering them, are wounded, and not in other cases where the lesions are strictly analogous? This difference must be ascribed to an abnormal tendency on the part of the blood itself to coagulate, which is present in one set of cases, and not in the other. Indeed, it is possible that traumatic thrombosis of the cerebral sinuses never occurs unless the coagulability of the blood itself happens to be considerably increased, or the condition of the blood in respect of readiness to coagulate is analogous to that which various crystallizable solutions must possess in order for crystals to form.

If this view is correct, of which there seems to be but little if any doubt, abnormal coagulability on the part of the blood itself must be looked upon as the predisposing cause, and fractures which lay open the veins of the diploë or the walls of the sinuses, together with the curdling action of the atmosphere on the blood at the gaping apertures in these vessels, as well as the presence of foreign bodies in the calibre of the sinuses, must be considered as the exciting causes in most instances of *traumatic cerebro-venous thrombosis*. I may here remark that I am acquainted with but one or two forms of local injury which lead the blood to clot in the larger veins during life when its coagulability is not increased, and they produced by the application of ligatures, and by amputation. In such cases the blood coagulates because all motion in it has been arrested over a certain space, and the coagulum usually extends from the place of ligation as far as the nearest collateral tributary having some considerable size. This is the simplest of all the forms of thrombosis.

In like manner coagula form in certain of the veins when they have been divided, as, for example, in the operation of amputation. I remember, however, one thigh-stump in which I found on dissection that the femoral vein was occluded with blood-clot, not beginning at its cut extremity, but at the first valve above the cut extremity and extending therefrom up to the next tributary, the cut end being empty and contracted for about three-fourths of an inch. In like manner also coagula would form in the cerebral veins.

and sinuses of the dura mater should the blood-stream therein be arrested by mechanical violence or traumatic agencies in any way analogous to the above. Under such circumstances it is only the portion of the blood which is completely stagnant, that coagulates, unless perchance the coagulability of the blood itself, at the same time, happens to be considerably increased.

2. *Causes of the Inflammatory variety of Cerebro-venous Thrombosis.*

—A perusal of the cases belonging to this class which are related or referred to in the first part of this essay shows very clearly that some local inflammatory disorders of the head, or of the parts contained therein, much oftener give rise to thrombosis of the cerebral veins and sinuses of the dura mater than others. For example otitis interna was the starting point of this affection in 33 instances; facial anthrax, in about 30; facial erysipelas, in 3 (including 1 to be related hereafter); ozena, in 2 (including 1 to be related hereafter); traumatic caries of the skull, in 3; traumatic inflammation of the skull and neighbouring parts without caries, in 2; suppurating wounds of the scalp, the skull being uninjured, in 1; suppurative diseases of the hairy scalp, such as chronic eczema, etc., in 2; idiopathic abscess of the eye-socket, in 3; extensive purulent infiltration following suppurative otitis, in 1; purulent meningitis, in 4 cases, and this disorder was probably the starting point of the thrombo-formation in several other instances. Thus, it appears that although a considerable variety of inflammatory affections may produce thrombosis of the cerebral sinuses, suppurative inflammation of the ear and carbuncle or furunculoid inflammation of the face occasion this disorder much more frequently than all the others put together, namely, in about three-fourths of all the cases. Now let us inquire more closely into the method by which internal otitis, on the one hand, and facial anthrax, on the other, bring about the formation of coagula in the sinuses of the dura mater; and, after that, ascertain, if we can, the reason why these diseases are so much more apt to be followed by cerebro-venous thrombosis than the other suppurative inflammations which so frequently attack the parts entering into the composition of the head.

With regard to suppurative otitis, it was found that in 29 out of the 33 cases above mentioned, disease of the temporal bone in the shape of caries or necrosis was the connecting link between the affection of the ear and the affection of the sinuses. It is probable that in these cases the disease of the bone caused the blood to coagulate in the minute veins of the diploë in the first instance, and that these small coagula afterwards extended themselves by prolongation until they entered and occluded the corresponding veins of the dura mater. In giving the rationale of this occurrence Von Dusch justly observes: "In thrombosis of the sinuses, the result of caries of the bones of the skull, the peculiar conditions of the veins of the diploë [that is, their incontractile state and gaping apertures

above-described], also appear to me to play an important part. The necrosis of the individual layers of bone, resulting from the process of ulceration in the bone, must necessarily diminish the supply of blood from the bone to the large venous trunks, and it will even happen in many instances that the supply of blood from the bone to certain of these is completely arrested. But as a shrinking of the calibre of these vessels is impossible, as already remarked, the diminished or arrested supply of blood leads to stagnation, and the formation of thrombus in them, which may extend into the sinus. We should thus have to regard the thrombus in the sinuses in such cases as propagated, that in the small veins of the diploë as primary and dependent upon the comparative excess in their calibre" (*op. cit.*, pp. 100, 101).

But caries (or necrosis) of the petrous portion of the temporal bone is not always the connecting link between internal otitis and thrombosis of the cerebral sinuses, for among the cases related above are four in which internal otitis produced thrombosis of these vessels without the intervention of either caries or necrosis. In these four cases the starting-point of the clot-formation appears to have been the inflamed and suppurating part of the ear, or rather some of the venous radicles belonging to this part. One of Dr. Dickinson's cases, also related above (see Case XIV.), clearly illustrates this point. At the autopsy of this case a small vein which passed directly from the diseased tympanum into the right lateral sinus was found filled with coagulum and converted into a solid cord thereby. Thus, it appears that in this case a thrombus whose formation commenced at the suppurating tympanum extended itself along the canal of a small vein, in a direct manner from the tympanum into the corresponding lateral sinus, which in the end it completely filled, occasioning also a secondary phlebitis therein.

In a manner closely analogous to this does facial anthrax, as well as facial erysipelas, ozæna, etc., produce thrombosis of the cavernous and other sinuses. In such cases the process of coagulation commences in some minute branch or branches of the facial or frontal vein which are in relation with the suppurating parts, and travels along the facial or frontal vein and its anastomoses into the ophthalmic vein, and so on, into the cavernous sinus.

The correctness of this statement is clearly proved by the post-mortem records of the cases of facial anthrax, etc., related above. For example, the autopsy in Case XXI. revealed an abscess as large as a small nut in left part of upper lip; surrounding tissues infiltrated with matter; a branch of the facial vein extending from the abscess to the inner canthus of the left eye contained this matter throughout; it anastomosed with the ophthalmic vein. The latter vein and the cavernous sinus were filled with a thick, chocolate-coloured liquid, containing streaks of puriform matter. The walls of these vessels were inflamed. The affection extended

through the circular sinus to the right cavernous. The autopsies in Casen XXIII., XXV., XXVI., XXXVIII., XL., and L. strongly support the same view; and the autopsy of no case supports a different view of the method by which facial anthrax and facial erysipelas produce thrombosis of the cerebral sinuses. In the account of Dr. Blachez's case of oœna (Case XLIX.) also, it is distinctly stated that the thrombosis and secondary phlebitis were traced, at the autopsy, to the nasal ulceration as the starting point.

With regard to the other, or the remaining forms of inflammation and abscess of the head and face which have occasionally led to the occurrence of thrombosis of the cerebral veins and sinuses of the dura mater, Von Dusch aptly says: "A similar view may be taken of extensive sanious inflammations of the cellular tissue, inflammations, which, as is well known, easily lead to phlebitis of larger venous trunks and to the phenomena of pyæmia and metastatic deposits. In such phlegmonous inflammations a greater or less extent of venous radicles is, for the most part, soon destroyed by the inflammatory process, which rapidly spreads and leads to necrosis of the tissues, whereby the supply of blood to the smaller venous trunks is diminished or completely arrested, and stagnation of the blood in them takes place from the absence of the *vis à tergo*. It may, indeed, more readily occur here than in the veins of the diploë that these small vessels collapse and propel their contents into the next branch, by which means the formation of a thrombus in them would be prevented. This may also be the reason why phlebitis does not always ensue in such cases. But if the inflammatory process produces solid infiltration and thickening of the tissues around these small veins, it will happen here, as in the veins of the diploë, that, the supply of blood being diminished, the unyielding nature of the vessels will furnish conditions favourable to the formation of thrombus. These small thrombi grow, and finally reach the larger trunks, and phlebitis ensues in the latter.

The reason why this consequent [or secondary] phlebitis mostly leads to suppurative and destruction of the coats of the vein, and but seldom assumes the so-called adhesive form, lies in the deleterious nature of the thrombus, which, arising in a deposit of various products of decomposition, conveys these by imbibition into the larger vessels" (p. 101). According to this view the formation of coagula in the small veins during life must be regarded as a consequence of stagnation of the blood therein, while those in the larger veins may be represented as occasioned by propagation therefrom; and the accompanying inflammation of the vein-walls may also be looked upon as secondary. Inflammation of the outer coat of the small veins could only be assumed to be a determining cause for the formation of thrombus in so far as the infiltration or induration of the connective tissue around them might prevent the collapse of their walls.

This view is by no means a new one, and allusions to it may be found in

the very suggestive *Rational Pathology* of Henle (ii. 516, 517), who did not overlook the favourable conditions for the formation of thrombus which exist in the non-contractile veins as well as in the sinuses of the dura mater, in the veins of bone, and in veins whose coats are paralyzed, or thickened, or indurated. Henle also remarks that in cases where the blood stagnates in the capillaries from diminished *vis à tergo*, the blood may also lose its motion and coagulate in the veins which spring from these capillaries, and thus phlebitis may result from stagnation in the capillaries. In brief, the method by which various inflammatory disorders of the head and face cause the blood to coagulate in the sinuses of the dura mater and the walls of these vessels to become inflamed, is as follows: In the cases which belong to this category the process of blood-clotting usually commences in some small vein or veins connected with the seat of inflammatory change, as a consequence of blood-stasis therein, and afterwards the coagula extend themselves by prolongation until they reach and occlude the sinuses; and these consecutive thrombi generally undergo puriform softening or disintegration instead of organization, because they have their origin in the decomposing products of the primary inflammatory lesion, the more fluid portions of which are conveyed into the larger vessels by imbibition. The disintegrating thrombi act upon the sinus walls like other deleterious substances, and like foreign bodies in general, and thus cause them to become inflamed.

But the starting-point of the thrombosis was found to be either internal otitis or facial anthrax in about three-fourths of all the cases having an inflammatory origin which I have collected, as stated above. Now why are internal otitis and facial anthrax so much more liable to be followed by thrombosis and phlebitis of the cerebral sinuses than the other suppurative inflammations which attack the parts entering into the composition of the head? These are questions of great practical moment, since the indications for the preventive treatment hang thereon, and we shall endeavour to answer them as fully as we can.

Firstly, with regard to internal otitis. The first cause which we are likely to assign is increased coagulability on the part of the blood itself. No doubt this liquid is abnormally coagulable in all the cases where thrombus forms in the sinuses of the dura mater in consequence of local inflammatory processes; for it is difficult to understand why a few patients having suppurating sores on the scalp, or orbital abscess, or facial erysipelas, or œzœna should get cerebro-venous thrombosis, while the many who suffer from these affections entirely escape, unless the blood is more disposed to clot in the veins of the former than it is in those of the latter. But granting this, the question then arises why otitis interna so much more frequently causes the coagulability of the blood to be increased than the above-named affections, although they are vastly more common than otitis interna?

The last-mentioned circumstance naturally leads us to suspect that the

discrepancy is due to some peculiar features of the morbid process which are generally present in the cases of otitis interna, and are, for the most part, not present in the other cases, such as the occurrence of caries or necrosis on the one hand, and the production of a highly deleterious pus on the other. Now, although caries or necrosis of the skull was present in twenty-nine of the thirty-three cases related or referred to above, in which otitis was attended with thrombosis of the cerebral sinuses, the occurrence of the last-named disorder cannot justly be ascribed to this caries or necrosis, *per se*, because these affections of bone are very often produced in the walls of the cranium by other causes, and when they are so produced they very seldom give rise to cerebro-venous thrombosis; for example, caries or necrosis of the skull was found to be connected with thrombosis of the cerebral sinuses, in all, in thirty-six cases that are related or referred to above, but out of these the caries or necrosis was due to internal otitis in twenty-nine, and to all other causes in only seven instances, while caries or necrosis of the skull is much less frequently produced by internal otitis than by other causes, such as struma, syphilis, injury, etc. In other words, cranial caries or necrosis occurs much oftener unconnected with inflammation of the ear than it does as a result of this disease, but is, at the same time, four times more liable to be attended with thrombosis of the cerebral sinuses when produced by internal otitis than when it results from all other causes combined.

The same question then presents itself in another form, namely, why is caries or necrosis of the skull so much oftener attended with cerebro-venous thrombosis when produced by internal otitis than when produced by other causes? To this some may reply that in cases of ear-disease the caries or necrosis occurs in the pars petrosa of the temporal bone, that is, in a portion of the skull which is closely connected with the lateral sinus; but this answer is not satisfactory, because caries or necrosis is folly as often, perhaps oftener, met with in parts of the skull which bear no equally close relationship to the superior longitudinal and other sinuses of the dura mater, and that, too, without leading to the formation of thrombus in these vessels, except in occasional instances, as is already proven by statistics given above as well as by the records of clinical experience and the observations of practising surgeons in general.

We are, therefore, compelled to attribute this remarkable proclivity on the part of otitis interna to occasion thrombosis of the cerebral sinuses, not to the caries or necrosis of the temporal bone with which it is so often attended, but to something peculiarly deleterious in the purulent matter which is formed by this disease. Furthermore, it is well known to every one acquainted with the subject that, in cases of inflammation of the ear, the discharge is oftentimes dreadfully offensive. Finally, we infer that the remarkable proclivity on the part of internal otitis, above mentioned, is due to the presence of some peculiar ichor or acrid exudate in the purulent

secretion, a result perhaps of potrefactive changes, which causes the blood to congregate first in some of the small veins connected with the seat of inflammatory change, and afterwards in the sinuses of the dura mater, wherein they empty, which also permeates the thrombus by imbibition, causing it to undergo disorganization instead of organization, and the vein or sinus walls to become correspondingly inflamed. And it is highly probable that thrombosis of the cerebral sinuses was produced in the same way in some at least of the four cases of internal otitis related in the first part of this essay, wherein thrombosis of these vessels occurred without the intervention of caries or necrosis.

Again, with regard to facial anthrax. It was found, as already stated, that in about 30 of the cases having an inflammatory origin, the thrombosis of the cerebral sinuses resulted from furunculoid or carbunculoid disease (anthrax) of the face; that in every one of these cases where the veins proceeding from the anthrax were subjected to examination at the autopsy, their calibres were filled with softening thrombi and their walls were inflamed; and that the progress of the thrombosis and phlebitis was traceable through these vessels from the anthrax to the ophthalmic vein and cavernous sinuses, and in occasional instances also downwards into the veins of the neck. We have likewise shown that furunculoid and carbunculoid inflammation of the face is a very fatal disease. Out of 23 cases mentioned in the first part of this essay, but 2 recovered. In 15 cases Mr. Puget saw, but 1 got well; and in a grand total of 45 cases we can find but 5 recoveries. Furthermore, we have shown that this enormous fatality is mainly due to the fact that facial anthrax excites thrombosis and secondary phlebitis in the veins connected therewith, which are rapidly propagated into the ophthalmic vein and cavernous sinuses, etc., on the one hand, and into the external jugular vein, etc., on the other. Now, why does it happen that facial anthrax is so much more liable to produce thrombosis and phlebitis than any other suppurative inflammation with which we are acquainted?

Prof. Güntner holds that the short stiff connective tissue of the lips, nose, septum oriom, etc., especially favours the occurrence of thrombosis in the veins belonging to these parts. This explanation, however, does not appear to me satisfactory, since suppurating wounds and ulcers of the lips and face in general, are not particularly liable, nor even apt, to be followed by thrombosis, phlebitis, and pyæmia, or either of them, which could hardly obtain if the connective tissue of these parts exerted so baleful an influence upon the veins. The truth is, this form of facial inflammation is entirely unlike any other disease known in this country. It has been called "malignant pustule," but improperly so, because it runs its course entirely without the history of contagion which belongs to that disorder. It has also been called, and with less impropriety we think, "malignant carbuncle" of the face. No doubt its peculiar malignancy is due to some

special condition of the anthrax itself, to some peculiarly acrid sanies of a septic origin and character which is developed in the foci of suppuration. We must suppose that a peculiarly intense poison is formed in these carbuncles, under circumstances that we do not exactly understand, which, on entering the veins by absorption, causes the blood in them to coagulate and to undergo putrefactive changes with great rapidity, and their walls to become destructively inflamed, in order to explain the phenomena of this disease. Moreover, this conclusion affords an important indication for the treatment of this disease.

But it may be asked whether in these rapidly fatal cases of facial anthrax the inflammation of the vein-walls does not precede the formation of coagulum in the vein-calibres—whether the phlebitis does not precede and induce the thrombosis. We reply that, on autopsy of cases belonging to this category, the vein-walls are usually found to be not at all, or but slightly, inflamed in parts where the thrombus is newest, while they are considerably inflamed only in parts where the coagulum is of a considerably older date—a circumstance which would not obtain if the thrombosis followed and depended upon the phlebitis for its production. We may, however, with propriety here remark, that a slight glance at the history of the formation of coagula in the veins during life shows that it is intimately connected with the history of phlebitis, and that widely different views concerning their origin and relationship have prevailed at different times. At first the clots were regarded as inflammatory exudations which were thrown out upon the inner surface of inflamed veins (*Hunter*). Afterwards it was ascertained that the clots were genuine coagula that had been formed from the blood itself, and then the opinion that the blood coagulated in these cases as an immediate consequence of the phlebitis, which was supported by the authority of Cruveilhier, generally prevailed. But it was reserved for Virchow, in his classical work on thrombosis, to clear up the matter and to show that, in a large majority of instances, the coagulation of blood in the veins precedes the inflammation of their walls, and that primary phlebitis with subsequent thrombosis much more rarely occurs.

3. *Causes of the Marasmic Variety of Cerebro-venous Thrombosis.*—There are four parts of the venous system in which the blood is especially liable to coagulate in consequence of debilitating influences and marasmus, during life, namely, the right chambers of the heart, the veins of the lower extremity, the veins of the true pelvis, and the sinuses of the dura mater; and of the last-mentioned, more particularly the superior longitudinal and lateral sinuses. The circumstances which specially favour the formation of coagula in these sinuses, in marasmic subjects, are the dilatations of calibre, the triangular shape, and the transverse bands (*chordæ Willisii*) projecting inwards from the walls, whereby the blood-stream is retarded, and more or less of blood-stasis (stagnation) is produced. Be-

sides, the cerebral veins, for the most part, empty into the superior longitudinal sinus in such a manner that the streams coming from them flow in directions which are either at right angles with the course of the blood in the sinus, or even oblique from behind forwards, that is, in directions more or less considerably opposed to the natural course of the blood in the sinus; and this circumstance favours the retardation of the blood-stream, not only in the sinus, but likewise in the cerebral veins themselves. Thus, we perceive that even under normal conditions the blood-current in the cerebral sinuses is comparatively slow, especially in the superior longitudinal, and the veins connected therewith; and when certain pathological conditions supervene which tend to more or less considerably diminish the force and rapidity of the general circulation, the retardation of the blood-flow, or the approach to blood-stasis, in the cerebral veins and sinuses is proportionally augmented, and a state of things correspondingly favourable to the formation of thrombus is produced. Also, we should, in this connection, again call attention to a circumstance of considerable importance which Heule was the first to point out, namely, that when the blood stagnates in the capillaries from diminished *vis à tergo*, as may readily happen in advanced cases of marasmus and debility, the blood may also lose its motion in the veins which spring from these capillaries, and thus thrombosis of these veins may result from stagnation in the capillaries, especially if, at the same time, the blood itself should be unusually disposed or inclined to coagulate.

But a perusal of the cases related above shows that other causes besides retardation of the blood-flow are concerned in the production of marasmic thrombosis of the cerebral veins and sinuses of the dura mater. And, first of all, the blood itself must be in a condition to readily coagulate, that is, it must be more coagulable than natural, or in a morbid state, whereby its coagulability is at least considerably increased. In perusing the marasmic cases of cerebro-venous thrombosis quoted above from the reports of Gerhardt, Von Dusch, West, Bouchut, Fritz, Crisp, Andrew, Murchison, Forster, Ogle, Janeway, and Charcot, I have been particularly struck by one circumstance, namely, in almost every instance the subject was obviously labouring under some blood-disorder of importance when the thrombosis occurred, and had been so affected, in most instances, for some time prior to its occurrence.

Moreover, on scrutinizing these cases we find that, although the nature of the blood-disorder differed considerably in different instances, yet all of them agreed in one particular, to wit, in exhibiting an abnormal tendency on the part of the blood to coagulate. For example, in Dr. Gerhardt's cases of diarrhœa and cholera infantum, the coagulability of the blood in the vessels became increased because its volume was rapidly diminished, and substance inspissated in consequence of the withdrawal therefrom of a great quantity of watery liquid to support the abdominal discharges. In

some cases mentioned by Von Dasch, the coagulability of the blood also became increased because its volume was suddenly diminished by excessive bloodletting, and in a somewhat analogous case related by Mr. Forster the coagulability of the blood was increased, in part, by a succession of hemorrhages to which the sloughing of a fungous tumour of the dura mater subjected the patient; in another set of these cases the increased coagulability of the blood was due to *hyperinosis*, which resulted from pneumonia in Dr. Ogle's case (No. LXVII.), from typhoid fever and pleuro-pneumonia in a case mentioned by Von Dasch, and from the puerperal state in a case also mentioned by Von Dasch; finally, in the cases of marasms, anæmia, and debility, the result of various wasting diseases of a chronic character, such as abscess, vertebral caries, scrofulosis, lung-phthisis, chlorosis, syphilitic cachexia, albuminuria, ague cachexia, chronic pleurisy, etc., that are related by Fritz, Von Dasch, West, Bouchat, Andrew, Murchison, Ogle, Janeway, etc., the increase of coagulability of the blood was probably due to the increase of its white corpuscular element, or the state of leucocythæmia which usually attends these disorders.

This abnormal tendency on the part of the blood to coagulate is doubtless one of the most important of the agencies which are concerned in producing the marasmic variety of cerebro-venous thrombosis. There is, however, another cause which is almost always concerned in producing this disorder, that possesses nearly equal importance. It is the enfeebled action of the heart, or the diminished force of the circulation which is usually present in the cases belonging to this category as an immediate consequence of the anæmia, debility, and marasmus. There results from this weakening of the cardiac contractions a corresponding tendency on the part of the blood to stagnate in the capillaries and to cease to move in the veins beyond them from want of the *vis à tergo*. This point in the etiology of this affection is also important because the vigour of the heart's contractions can be increased by remedies.

Another cause which probably exerts some influence, in occasional instances, in producing the marasmic variety of cerebro-venous thrombosis, is vaso-motor paralysis of the cerebral bloodvessels, which, by destroying the contractility of these vessels, would allow them to become expanded by the force of the blood-stream, and would favour to a corresponding degree the occurrence of stasis and coagulation, especially in the capillaries and veins.

Again, age exerts an important influence in producing the marasmic variety of this affection. Its victims are mostly found among the very young and the aged. Infants and young children are especially liable to be attacked by it because they are exposed to the occurrence, not only of the diseases which produce it in the more advanced periods of life, but also of special diseases, such as the colliquative diarrhoea and cholera infantum, which, as we have shown above, may rapidly increase the coagu-

lability of the circulating blood by diminishing its volume or inspissating it, on the one hand, and, at the same time, may induce it to stagnate in favorable localities by weakening the cardiac contractions and diminishing the vascular tension, on the other. Furthermore, during infancy and childhood the brain constitutes a much larger proportional part of the whole body, amounting then to one-tenth or one-fifteenth thereof, than it does in adult years, when it becomes reduced (comparatively) to one-fortieth or one-fiftieth part of the whole body. Besides, the watery brains of infants are especially prone to shrink and thus cause the cerebral veins and sinuses to expand their calibres in order to fill up the resulting vacuum, on the occurrence of any disease which rapidly withdraws the serum from the blood and compels it in turn to withdraw the serum from the substance of the brain, as well as from the parenchyma of other organs, by the process of absorption. Aged people, also, are especially liable to be attacked by cerebro-venous thrombosis, because the coagulability of their blood is prone to become increased from senile miasmata, while the cardiac contractions grow weaker from the decay incident to advancing years, and the cerebral veins and sinuses become expanded and the blood-stream sluggish in them in consequence of the shrinkage to which their brains are exposed from senile atrophy. Moreover, in aged people the force of the blood-stream is apt to be weakened in the capillaries, veins, and sinuses of the brain, by the occurrence of atheroma and fatty degeneration in the coats of the cerebral arteries, which renders them more or less stiff, non-elastic, and non-contractional, and thus still further lessens the *vis à tergo* of the senile heart. But, although marasmic thrombosis of the cerebral veins and sinuses of the dura mater is most frequently met with in aged persons and young children, it may also occur at any other period of life when some disease is present which induces a cachectic state of the system with feeble heart-action, and, at the same time, increases the coagulability of the blood.

Furthermore, we should remark that the causation of cerebro-venous thrombosis is usually a very complex affair, and that not a solitary one but several of the factors above mentioned are jointly concerned in the production of almost every individual case. We should also state in this place that we have discussed the etiology of this deadly disease as thoroughly as we could, with a hope that we might discover in this way what the causal indications are for its treatment, and thus acquire some means to prevent its occurrence and to obviate its mortality. These indications, however, will be taken up and only considered in the appropriate place, that is, under the head of treatment.

Anatomical Appearances or Changes.—On examining post-mortem the veins and sinuses affected with thrombosis they usually present externally a more or less swollen or distended appearance, and are more or less stained with a reddish or a brownish hue. On opening them the distension

is found to be due to the thrombus which fills up the calibre and dyes the walls with its colouring-matter. The thrombus itself, if recent, is usually dark-red or brown in colour, pretty firm in consistence, rather dry, somewhat elastic, laminated, and more or less strongly attached to the vessel which contains it. As it grows older, however, the red corpuscles gradually disappear, and at the end of ten days or a fortnight it may be so completely decolorized as to resemble a plug of pure fibrin. But in the cases of inflammatory origin it almost always undergoes softening or disintegration, and sometimes putrefaction. In such cases, also, the walls of the affected veins and sinuses are exceedingly apt to be inflamed. When the thrombus softens the disintegrating process usually commences at its centre, and the liquefied material varies in colour and consistence from that of wine-lees to that of cream-like pus. More frequently, however, it has a dirty reddish-brown appearance, and the consistence of purulent matter. Examined with the microscope the disintegrating thrombus, even when puriform in appearance, is found to consist of granular fibrin, shreds, some red corpuscles more or less altered or broken down, and many leucocytes. When the walls of the affected veins and sinuses are inflamed they become thickened and more or less softened, their capillary vessels injected, their inner surface tomentose, roughened, and sometimes eroded or ulcerated, as well as dark-red in colour. When erosions or ulcerations are present, genuine purulent matter can also be found intermingled with the disintegrating thrombus. Furthermore, the veins which lie on the distal side of the thrombosed sinus are generally gorged with blood, the pia mater and cerebral substance oedematous, with more or less serous effusion in the ventricles, and in many instances blood also is extravasated into the meninges or into the cerebral substance.

In nineteen of the cases related or referred to above, cerebral or meningeal cerebral hemorrhage occurred. This complication, however, is met with very much oftener in the marasmic than any other variety. Of these nineteen hemorrhagic cases sixteen had a marasmic origin, and an examination of them shows that cerebral hemorrhage is most liable to occur in cases where the superior longitudinal or straight sinuses, or the cerebral veins themselves, are thrombosed. But secondary phlebitis is met with very much oftener in cases having an inflammatory than in those having a marasmic origin. Besides, in these cases the inflammatory process is exceedingly apt to spread from the sinus-walls to the contiguous membranes of the brain, thereby inducing a meningitis which is often purulent in character; and sometimes the inflammatory process spreads also to the brain-substance and induces inflammatory softening and abscess therein. Finally, the cases having an inflammatory origin are frequently, and the marasmic cases occasionally, complicated with the occurrence of hemorrhagic infarctions in the lungs, of so-called metastatic pneumonia and

plenistry, of so-called secondary abscesses in the lungs, liver, etc., and with the phenomena of pyæmia or septicæmia.

Symptoms and Course.—Different observers give somewhat different views of the phenomena produced by thrombosis of the cerebral veins and sinuses of the dura mater. We shall, however, present only a brief sketch of the symptoms which the leading varieties of this disease present, and illustrate it with condensed reports of some additional cases. Dr. Hubner (*Archives der Heilkunde*, ix. 5, p. 417, 1868) gives the following instance of mural thrombosis of the cerebral sinuses, wherein, as usually obtains, the diagnosis was not made during life, together with a critical analysis and interpretation of the symptoms:—

CASE LXX.—The patient presented at first general head-symptoms, but soon afterwards neuralgia of the right supra-orbital nerve, blepharoptosis on the same side, œdema of the corresponding eyelids, loss of hearing, and finally facial paralysis on the right side with blood-stases in the corresponding frontal veins. These phenomena were all very transient. On *autopsy* there were found thrombosis of the superior longitudinal and transverse sinuses, thrombosis of the right cavernous sinus, and thrombosis of the right ophthalmic vein.

Dr. Hübner thinks the formation of the thrombus in this case commenced in the superior longitudinal sinus and extended therefrom into the transverse sinuses; the obstruction of these vessels with coagulum brought on an intense collateral stasis of blood in all the sinuses at the base of the brain. The stasis thus produced in the cavernous sinus led to dilatation of the collateral veins of the face, and to compression of certain nerves, including the great sympathetic; hence to dilatation of the cerebral arteries. When the thrombosis had extended as far as the cavernous sinus, the phenomenon of stasis and compression diminished in intensity. It is easy in this way to explain the mutability of the symptoms, which was indicated by Lebert as one of the characteristic phenomena of thrombosis involving the cerebral sinuses.

Dr. Hubner remarks concerning the phenomena which result from thrombosis of the cerebral sinuses: The stasis in the cerebral veins generally gives rise to characteristic cerebral symptoms. Venous dilatation may also exist in the collateral vessels of the walls of the cranium, or rather it may show itself in the veins which unite the vessels of the face to those of the cranium. In the latter case vascular dilatation has considerable diagnostic importance. In obstructions of the transverse sinuses one may observe dilatation of the emissary veins of Santorini and of the mastoid vein. The parietal veins also are dilated in cases where the longitudinal or ethmoidal sinuses are obstructed. When the ophthalmic vein which carries to the cavernous sinus the blood of the frontal vein, of the ophthalmic bulb, of the eyelids, and of the ocular muscles, is obstructed with thrombus, one may observe a venous injection in the skin of the forehead, with œdema of the eyelids, of the globe of the eye, and even of the ocular muscles; hence doubtless arises the prominence of the eye often noticed in cases of this kind (*Archiv. Gén. de Méd.*, 1869, vol. i. pp. 357-359).

Dr. Hermann Weber (*Medico-Chirurg. Transact.*, vol. xliii. pp. 177-180) relates the following case in which erysipelas of right side of face, etc., was followed by thrombosis of right ophthalmic vein and of right cavernous and circular sinuses, meningitis, and death. The account of the symptoms and *post-mortem* appearances, although much abridged, is very interesting and useful in this connection:—

CASE LXXI.—F. M., aged 25, a pale and delicate-looking shoemaker, was attacked by erysipelas of right cheek and right eye on December 14th. At the end of a week the erysipelas had almost disappeared, and the patient was considered convalescent, when, on December 23d, he began to complain of dull headache; pulse 105; body temperature increased. During the next three days the headache increased, there was occasional delirium, grinding of teeth in sleep, twitching of muscles; pupils rather contracted, almost immovable; sickness and constipation with a pulse slow and irregular (60-85). On December 27th, he became drowsy; pupils wide and sluggish; left arm slightly paralyzed. On December 30th, drowsiness much increased; pupils dilated and insensible; paralysis of left side extending now to leg as well as arm. The degree of this paralysis varied at different times, but a remarkable, although transient improvement of this symptom and all the others occurred on December 31st, when the drowsiness almost disappeared, the patient became conscious, and was able to speak to his friends; in the following night, however, the coma returned, the pulse became very weak and frequent, the breathing irregular, until death ensued on January 2d.

Autopsy 26 hours after death.—Upper part of nose, lids of right eye, and surrounding tissue slightly swollen; a moist light-brown scab from an almost dried blister is seen on right side of nose; a similar scab on upper lid of right eye, slight œdem of pia mater on both sides; purulent meningitis on lower surface of anterior lobe of right hemisphere; lateral ventricles contain about two ounces of turbid fluid; their walls slightly softened, but the rest of the brain-substance normal. The right cavernous sinus feels hard and enlarged; its walls are thickened and its calibre filled by a grayish-red conglum strongly adherent to the lining membrane, which appears not quite smooth. The clot is composed of several layers; the exterior being grayish-red, the middle purer red, the centre cherry-red, and softer than the others. The internal carotid and the nerves in the walls of the sinus appear unchanged. The ophthalmic vein, in connection with the sinus, contained a dark conglum, rather firmly adherent to the thickened lining membrane, but the rest of this vein, as well as the veins of the face, etc., were not examined, because it was not permitted to open the orbit and the tissues of the cheek. Blood in circular sinus coagulated; that in left cavernous sinus not quite coagulated; their walls normal; the other sinuses exhibited nothing pathological. Heart hypertrophied; lungs and abdominal organs normal; no secondary abscesses.

The brain-symptoms in the above case resemble in many respects those caused by acute hydrocephalus, which was, in fact, diagnosed by two physicians who examined the patient without being acquainted with his history. The changing character of the hemiplegia was remarkable, and not less so the striking improvement of all the symptoms two days before death; but the latter phenomenon has occasionally been seen to occur in the most deceptive manner in other diseases of the brain (*Weber*).

The fact that the appearance of head-symptoms was preceded by erysipelas of the face, especially of the right eyelids and side of the nose, that the right ophthalmic vein and right cavernous sinus were distended with thrombus and their walls inflamed, and that there was circumscribed purulent meningitis confined to the under surface of the anterior lobe of the

right cerebral hemisphere, renders it pretty certain that the endocranial affection was caused by the facial erysipelas, and that the ophthalmic vein was the medium of communication. It is not improbable that the coagulation of the blood commenced in the capillaries and venous plexus of the right side of nose, cheek, and eyelids, and proceeded thence to the right ophthalmic vein and cavernous sinns, as it did in Cases L. and LI., related in a previous paper, which are strictly analogous to the above.

The inflammatory thickening that was found in the coats of the ophthalmic vein and cavernous sinns might lead to the view that phlebitis (properly so called) was the first link in the chain of pathological processes, and that the coagulation of the blood and the alteration of the clot in these vessels were only the consequences of inflammation of their walls. As we know, however, that the presence of blood-clot in veins, and the changes in composition which it is very liable to undergo, are in themselves sufficient to cause inflammation of the vein-walls, on the one hand, and that inflammation of the vein-walls alone is not sufficient to cause thrombosis on the other, the inflammatory thickening above mentioned cannot be considered to possess any value as evidence that the erysipelatous process was propagated from the face to the membranes of the brain by idiopathic phlebitis, instead of thrombosis and secondary phlebitis.

Dr. Hermann Weber (*Medico-Chirurg. Transact.*, vol. xliii. pp. 182-186) has also reported the following case of cerebro-venous thrombosis resulting from syphilitic *ozæna*, in which the symptoms of thrombosis were complicated with those of pyæmia. The following brief account of it will likewise prove useful in this connection:—

CASE LXXII.—A waiter, aged 24, of scrupulous parentage, had been afflicted with constitutional syphilis for about six years, and with syphilitic *ozæna* for over one year.

February 4. He has headache, especially in frontal region; rather increased by shaking the head; not increased by tapping on the forehead. Nose flattened and slightly swollen; nostrils blocked up by dry brownish crusts; fetor moderate; pressure on nose not painful; pupils symmetrical and responding well to light; pulse regular, about ninety; bowels costive.

7th. Appearances of nose unchanged; headache constant, but increased at night; yesterday he was often delirious; to-day he is drowsy, but can be roused, and then answers reasonably; pupils rather dilated and sluggish. Has had repeated rigors. Spleen yesterday found perceptibly enlarged; to-day there is in addition, tenderness of lower part of left side, and a soft friction-sound is occasionally heard.

9th. Somnolency increased; carphology; muttering delirium; muscular twitching, especially in thighs; ptosis of left upper eyelid; motions of left eye much restricted from paralysis of external rectus; pupils (both) wide and sluggish; tongue dry; pulse 120-125, very small; friction-sound over lower part of left chest again noticed; *dalaess* increased.

10th. Complete coma; right-sided hemiplegia; left eye almost closed; its conjunctiva slightly injected. Death on the 11th.

Autopsy 22 hours after death.—Examination of eye-socket not allowed. Circumscribed purulent meningitis limited to the inferior surface of anterior lobe of left cerebral hemisphere; veins of pia mater distended and filled with dark coagula. Lateral ventricles contain about three drachms of not quite transparent fluid.

Superior longitudinal sinus contains a dark-red coagulum, rather firmly adherent to its walls, which, however, are not perceptibly altered. Left cavernous sinus filled by a crumbling slightly cohesive thrombus of reddish-gray colour. The external part thereof adheres closely to the walls. The central part is softened to the consistence of thick pus, and has a brownish-red colour. The walls of this sinus are considerably thickened, as are also those of the ophthalmic vein (as far as examined) which is likewise filled by a dry, rather dark coagulum. The internal carotid artery and the nerves in relation with the left cavernous sinus appear not materially changed, excepting increased thickness of the surrounding areolar tissue. The left inferior petrosal, the circular, and the right cavernous sinuses contain a dark-red coagulum, which adheres but slightly to the uncalcified lining membrane. The other sinuses of the dura mater contain a loose coagulum which does not adhere at all. Cranial bones not carious.

The softened part of the thrombus of left cavernous sinus, examined with the microscope, contains: 1st. Small granules, which are dissolved by acetic acid; 2d. Aggregations of such granules into irregular-shaped bodies (round, oval, crescentic), which are likewise dissolved by acetic acid, nowhere exhibiting the characters of pus globules; 3d. Other granules and aggregations of granules, dissolvable by ether and not by acetic acid; 4th. Oil-globules of various size; 5th. Blood-globules of different shapes, some being much shrunk, some exhibiting a tendency to the star form, others being granular on the surface.

A pint of purulent effusion in left pleural cavity; secondary abscesses in lungs and liver. Spleen enlarged and much softened.

The dropping of the upper lid of left eye, as well as the paralysis of the left external rectus, in this case, are easily accounted for by considering the relation which existed between the third and sixth nerves and the thrombosed cavernous sinuses, together with the thickened state of the surrounding connective tissue, since these nerves may have been compressed thereby.

The symptoms referable to the cerebro-venous thrombosis were frontal headache, delirium, stupor, dilatation of pupils, swelling of left eyelids, injection of conjunctiva, ptosis of left upper eyelid, paralysis of left external rectus oculi, right-sided hemiplegia, and coma. The symptoms referable to pyæmia were rigors occurring at irregular intervals, marked increase of body-heat, enlargement of spleen, soft friction sound and dulness over lower part of left chest, muttering carphology and other muscular twitchings, dryness of tongue, and great frequency of pulse. With regard to the diagnosis, an attentive consideration of the history of the patient, and of his symptoms as a whole, employing at the same time the process of exclusion in their interpretation, could hardly fail to lead any one familiar with the subject of thrombosis of the cerebral sinuses, together with the symptoms of pulmonary infarction and pyæmia, to a correct understanding of the case.

The thrombosis probably commenced in the veins directly connected with the nasal ulceration, and proceeded thence by prolongation through the anastomosing branches of the facial vein into the left ophthalmic, and the left cavernous sinuses. The thickening of the walls of these vessels was the result of inflammatory action excited therein by the presence of a disintegrating thrombus; and the purulent meningitis in turn was due to the

extension of the inflammatory process from the walls of the ophthalmic vein and cavernous sinns to the neighbouring membranes of the brain. The thrombosis of certaiu veins of the pia mater, which was found on autopsy, resulted in all probability from the purulent meningitis, and the formation of thrombus in the superior longitudinal sinus was occasioned by the extension of the thrombi from the cerebral veins to the calibre of the sinus.

But a review of the various cases of thrombosis of the cerebral veins and sinuses of the dura mater, heretofore presented, conclusively shows that the symptoms attending this affection when produced by inflammatory causes differ considerably from those which are present when it results from debilitating or marasmic influences. Without descending too much into particulars we may remark that in the cases of inflammatory origin we are much more apt to find phlebitis of the sinuses together with an inflammation of the membranes of the brain which has been lighted up by an extension of the inflammatory process from the sinus-walls to the contiguous membranes of the brain, on the one hand, as well as the phenomena of pulmonary infarction and septicæmia, on the other, than in the cases having a marasmic origin. In fact, the marasmic variety of cerebro-venous thrombosis is but seldom attended with secondary phlebitis, septicæmia, and the so-called metastatic pneumonia, while the inflammatory variety is often attended with these consequences. In the marasmic cases, then, we but seldom or never find the symptoms complicated with, or masked by, the phenomena of secondary meningitis, of septicæmia, or metastatic pneumonia.

What are the consequences, so far as the brain is concerned, of plugging up the cerebral veins and sinuses of the dura mater with conglulum? They are the following: venous congestion, œdematous infiltration of the pia mater, and of the brain-substances itself, together with cerebral hemorrhage in a large proportion of the cases. In other words, the substance of the brain becomes compressed by the engorged veins, the exuded serum, and the extravasated blood; or, speaking more correctly, the cerebral capillaries become compressed in this way, and the nerve-filamenta and ganglion-cells of the cerebral substance deprived of nutrient blood. The symptoms resulting from thrombosis of the cerebral sinuses are, therefore, the symptoms of cerebral anæmia, namely, sopor with or without convulsions, pain, paralysis, coma, death. It will be observed that the symptoms are much the same as when the anæmia of the brain-substance is produced by some other cause.

The question then arises whether it is possible to make a differential diagnosis between cerebral anæmia due to thrombosis of the cerebral sinuses and that due to other causes. Von Dusch thinks that when infants are the subjects such a diagnosis can sometimes be made. He points out that in such cases as those related by Dr. Gerhardt the symptoms taken to-

gether indicate with tolerable certainty the presence of cerebro-venous thrombosis. When profuse diarrhoeas in weakly children are followed by the above-mentioned cerebral symptoms, with depression of the fontanelles and overlapping of the cranial bones, accompanied by an unequal distension of the two external jugular veins, the diagnosis of cerebral-venous thrombosis is highly probable, the disease being located on the side where the vein is least distended; but the absence of the last-named phenomenon does not exclude the possibility of thrombosis of the cerebral sinuses, because if the superior longitudinal sinus alone, or both lateral sinuses simultaneously, happen to be the seat of the disease, no inequality with respect to distension can be observed in the two external jugular veins. In most of the marasmic cases, however, the diagnosis is very uncertain and can only be based on a general consideration of all the symptoms, or be arrived at by the exclusion of other affections.

In the cases which have an inflammatory origin the symptoms that present themselves are usually due not so much to cerebral anæmia as to certain morbid changes in the membranes and substance of the brain, and to septicæmia with pulmonary infarctions or so-called metastatic pneumonia. When, however, in cases of chronic otitis, or facial farinicle, or facial erysipelas the above-mentioned symptoms of cerebral anæmia supervene, and cannot otherwise be accounted for, they should lead us to strongly suspect that thrombosis has occurred in the sinus of the dura mater which lies next to the inflamed part; and in cases of facial anthrax and of facial erysipelas this diagnosis will be still more probable if there are also œdematous tumefaction with incomplete closure of the eyelids and protrusion of the eyeball on the affected side of the head.¹

The mutability or transient character of the symptoms which obtains in many cases belonging to each variety of this affection is probably due to the fact that the degree of cerebral anæmia is not constant but varies according to the growth of the thrombi themselves, as well as in accordance with the freedom with which the blood escapes from the brain through the collateral channels.

Ophthalmoscopic Symptoms of Cerebro-venous Thrombosis.—But few observations on this point have been placed on record. Dr. Bouchut, however, (*L'Union Médicale*, 1869) concludes, from his own experience with the ophthalmoscope in cases of cerebral disease, which appears to have been extensive, that hyperæmia of the retinal veins, when attended with stasis and the formation of coagulum, denotes repletion of the sinuses and thrombosis of the meningeal veins; that œdema of the papilla indicates thrombosis of the sinuses or of the cerebral veins; and generally

¹ In facial anthrax such symptoms as the following, namely, headache, delirium, stupor, dilatation of the pupil, difficulty in swallowing, incontinence of urine, and coma, pretty uniformly occur.

that in cases where the cerebral circulation is greatly disturbed (as it is in cerebro-venous thrombosis) one may see something analogous in the eye (*Brit. and For. Med. Chirurg. Review*, Oct. 1871, pp. 511, 512). Should these opinions be confirmed by other observers, they will furnish diagnostic signs of great importance.

Treatment.—In this disease art is powerless to dissolve the thrombus or to reopeo in any other way the obstructed veins and sinuses of the brain, after the coagulum is formed. In treating this affection, therefore, the physician should mainly direct his attention to the prophylaxis; that is, while treating the various accidents and disorders which, as shown above, are liable to prove fatal by inducing cerebro-venous thrombosis, he should endeavour to prevent the formation of the thrombus itself, and in doing this he must be guided by the causal indications. We will briefly present them for each variety of the disease.

1. With regard to the traumatic variety: the indications for treatment which are derived from a consideration of the etiology of this variety are few and simple. They consist in the removal by surgical means of any foreign bodies which penetrate the sinuses so as to come directly into contact with the blood contained in them, or press upon them externally, so as to narrow their calibre, or obstruct the movement of the blood through them in any other way. They consist also in rendering negative the operation of any cause which by increasing the coagulability of the blood promotes the formation of thrombus at the seat of injury, in avoiding excessive blood-letting, in arresting excessive hemorrhages, and in restraining excessive alvicol discharges in cases of cranial injury.

2. With regard to the inflammatory variety: inasmuch as the occurrence of thrombosis and phlebitis in the cases belonging to this variety is due to the presence of some deleterious agent of peculiar character, some special ichor, in the inflamed part or suppurating focus, as we have shown above, which by virtue of its own destructive properties causes the blood to coagulate in the veins proceeding from the inflamed part, and in the corresponding sinuses of the dura mater, which likewise causes the thrombus itself to disintegrate, and sometimes to putrefy, and the walls of the affected vessels to become inflamed, the causal indications for the treatment of these cases are also very plain. They consist in preventing the development of this poison in the inflamed or suppurating part on the one hand, and in reducing it to a state of inertness, that is, in destroying it, on the other.

Experience has shown that internal otitis, facial anthrax, facial erysipelas, and eczema, but especially the first two, are the inflammatory disorders which are most liable to be attended with thrombosis of the cerebral sinuses. In cases of suppurative otitis we can do much to ward off thrombosis by securing a free outlet for the matter, by extracting the fragments of dead bone as soon as they become detached, and by the persistent use of antiseptic or disinfectant washes, such as chlorine water, liquor sodii chlorinat. suitably

diluted, weak solutions of carbolic acid, pretty strong solutions of sulphite of soda, etc., and the "girondin" disinfectant fluid. An antral surgeon of eminence informs me that the last-named has yielded very satisfactory results in his hands. By employing these means we may keep the purulent matter in the internal ear and neighboring parts from putrefying, and the ichor above mentioned from forming, or we may destroy it if already formed.

Generally, while treating the local inflammatory affections of the head we can best diminish the risk of cerebro-venous thrombosis by the early evacuation of all purulent collections with free incisions, by cleanliness, and by the unstinted use of antiseptic fomentations and lotions, combined with appropriate internal medication. But when carbuncloid or furunculoid inflammation (anthrax) of the face presents itself, something more is required in order to prevent the development of thrombosis, phlebitis, and septicæmia. The terrible mortality recorded against it above (40 deaths in 45 cases) shows the insufficiency of the plans of treatment heretofore employed. The medication must be more radical and thorough in order to prove successful. The pimple, boil, or pustule which ushers in the disease, that is, the inflammatory focus, wherein the special septic poison which induces the thrombosis, etc., is formed, must be destroyed with an unsparing hand, at no early stage, if we would save the patient. The plan of treatment which has proved so successful in a strictly analogous disease, the malignant pustule of Europe, should be tried in this affection also. Dr. Belles (*Gaz. Méd. de Paris*), a physician practising in the districts of Béja and Faro, in Portugal, where malignant pustule is very frequent in consequence of the number of cattle raised there, says he has treated hundreds of cases of this disease without having noted a single death. The treatment employed by Dr. Belles consists in crucial incision of the boil throughout its whole extent, in depth as well as breadth, and canterization with chloride of antimony, repeated until bleeding ceases. The gangrenous tissue is completely insensible to both cutting and canterization (*Med. Record*, Aug. 1873, p. 372). The separation of the eschar should be favoured by the application of antiseptic poultices made of charcoal, yeast, bark, etc., or, better still, by antiseptic fomentations, consisting of carbolic acid (part 1 to 200), or liquor sodii chlorinat. (part 1 to 12), or zinci chlorid. (part 1 to 200), or of the "girondin" disinfectant fluid. Prof. Güntner, of Salzburg, who saved two out of five cases of facial anthrax, recommends especially the constant application of warm fomentations. He found that cold applications were positively hurtful. Mr. Thomas Smith (*Clin. Soc. Transact.*, iii. p. 63) also proposes that the sulphite of soda or magnesia should be systematically administered from the very first in facial anthrax as a preventive of blood-poisoning. At the same time, if the body-temperature ranges high, it may be advantageously abated by giving quinine in full doses, or in such doses as to produce cinchonism.

3. With regard to the marasmic variety: in the cases belonging to this

sort of cerebro-venous thrombosis, also, the causal indications for treatment are but few and plain. In managing the various forms of chronic disease which are attended with marasmus, leucocythæmia, and great debility, and which, as we have shown above, are also liable to result in thrombosis of the cerebral veins and sinuses of the dura mater, the indications are first to lessen the abnormal tendency on the part of the blood itself to coagulate, and second, to prevent the occurrence of blood-stasis in the venous system by supporting the failing powers of the heart and arteries. And here we should remark, that the physician's success in saving or prolonging life in cases of pulmonary tuberculosis, chronic bronchitis, chronic pneumonia, chronic pleurisy, chronic abscess, vertebral caries, chlorosis, leukæmia, scrofulous adenitis, ague cachexia, syphilitic cachexia, infantile marasmus, senile marasmus, and many other debilitating diseases, will largely depend upon his ability to prevent the occurrence of thrombosis in some part of the venous system. Moreover, it is a fortunate circumstance that the remedies which are best adapted to fulfil the above-mentioned indications are, for the most part, also the medicines which experience has shown to be most useful, in a general way, in combating these several diseases. These remedies are the various preparations of ammonia, especially the muriate and carbonate, alcoholic stimulants, quinine, and such cardiac excitants as digitalis and atropia.

After much observation and reflection, I have no doubt that certain preparations of ammonia, especially the muriate, carbonate, aromatic spirit, and the liquor ammonii anisatus of the Prussian codex, are capable of diminishing considerably the coagulability of the blood when it is morbidly augmented, and that too, without inducing debility even when their use is long continued. For this and other reasons I hold the muriate of ammonia to be one of the most valuable medicines with which I am acquainted. It is probable that the spt. ammon. aromat and the liquor ammon. anisat. prove so useful in the bowel complaints of children, when great debility is present, not only because they strengthen the cardiac contractions, but also because they diminish the coagulability of the blood itself. It is doubtless for a similar reason, that the administration of carbonate of ammonia proves so serviceable in scarlatina, that it is held by many to be almost a specific for that disease. Finally, it was the aromatic spt. of ammonia taken steadily for many months which saved the patient in the only case of leukæmia that I have found recorded where recovery took place.