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EMBOLIC
DISEASES OF THE EYE.

BY

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(These remarks, in substance, were made before the New York Medical Journal Association, January 22, 1869.)

Our knowledge of *embolic diseases* in general is yet of a recent date, but already it has thrown full light on so many morbid processes, formerly quite inexplicable, that it may with justice be called one of the greatest acquisitions pathology has made in modern times, and it alone would secure to the name of *Virchow* a prominent place in the history of medical discoveries. Soon after the invention of the ophthalmoscope, "which crowns the forehead of *Helmholtz* (now Professor in Heidelberg) with the laurel of immortality," Professor *Virchow* prophesied that it must be possible to *observe directly*, with this instrument, *emboli in the living eye*. Four years later the prophecy was realized by

a third no less illustrious representative of the German school and of medical progress, that of Professor *Von Graefe*. It is not without intention, gentlemen, that I pronounce these glorious names, for only some five or six weeks ago I had the honor to listen, in the New York Academy of Medicine, to a lecture on Medical Progress in the Present Century, and therein not one German investigator was mentioned. Despite these omissions, the lecture was very elaborate and excellent. Had it not been so, and had it been delivered by a lesser man than *Austin Flint, Sen.*, I should not have minded it. But since even the most learned cannot aim at completeness, and we all here form an association for mutual assistance, I thought it might not be an unfit subject for a minor mind to fill up some one or other of the omissions of the great.

Out of the large series of embolic diseases, I can exhibit before you only one branch, *embolism in the eye*, and even this would take the time of several evenings, if I were to describe it fully. Nevertheless, I shall try to give you an uncurtailed picture of it, but executed in such a way, that the known parts are only roughly sketched, while I shall more minutely draw those which are but little elucidated, and point out others that are not yet known at all.

Embolic diseases of the eye may be thus divided:

A. *Thrombosis* (*a*) of the retinal, (*b*) of the choroidal, and (*c*) of the ophthalmic veins.

B. *Capillary embolia* (*a*) of the choroid, and (*b*) of the retina.

C. *Embolia* (*a*) of the choroidal, and (*b*) of the retinal arteries.

In thrombosis we must always distinguish between primary and secondary

A (a). *Primary thrombosis of retinal veins has never been described.* Yet there are conditions for its development in several eye-diseases, where the venous current is checked in a high degree, so that coagulation of blood appears possible, especially under favorable conditions of the general system. In *glaucoma*, the retinal veins are compressed at the point of their exit from the eye; they are swollen and tortuous. A notable retardation of the current must be the consequence. At the same time, the increase of intraocular pressure allows only of smaller supply of arterial blood into the eye. Often even the continuous arterial current is interrupted, and the increase of intravascular pressure, caused by the systole of the heart, throws a certain quantity of blood into the retina, while during the diastole of the heart the retinal artery may be entirely compressed by the tension from the globe. In this way we find, in *glaucoma*, different causes for the retardation of the venous current: 1. Impediment at the outset by compression of the venous trunks. 2. The greater filling of veins and capillaries consequent to the pressure on the trunks, increasing the column of blood to be moved by the *vis a tergo*. 3. Weakening of the latter through compression of the arteries by the increased tension of the eye.

Although we know that, in blood-vessels with healthy walls, normal blood, even when stagnating, will for days remain fluid, the conditions for coagulation under the circumstances just described appear so favorable, that I think it fit for ophthalmoscopists to direct their attention to this point. Quite similar are the conditions in certain forms of *neuro-retinitis*, where an exudation into the ocular end of the ophthalmic nerve compresses the central retinal arteries as well as the

veins, causing swelling of the latter, and not unfrequently hæmorrhages. As we see these hæmorrhages most abundant in that form of retinitis which is dependent on Bright's disease, where the coagulability of the blood is increased, I do not think it quite devoid of sense to take into consideration the question whether the retinal hæmorrhages in this disease may not be in some connection with thrombosis in the retinal veins or capillaries.

Another pathological state which may possibly occasion primary retinal thrombosis is *detachment of the retina*, especially one form of it, which I may be allowed to point out here for the first time; I mean *detachment caused by contraction of tendinous cords* such as are seen in rare cases of plastic (interstitial) retinitis. Although such cords are not so very uncommon after certain forms of retinitis, especially syphilitic, they very rarely cause detachment. One striking example of it, however, I had the opportunity of examining by the favor of my esteemed colleague, Dr. C. R. Agnew. The retina was detached, or, rather, folded up like a frill or ruffle, and through its tissue ran a number of grayish-white cords, lying, in part, beneath the retinal vessels, in part enveloping them. The vessels were evidently compressed by these cords, for they disappeared or became very narrow where they passed through them, but were exquisitely tortuous and engorged in their neighborhood. Some looked so very dark red, that an extreme slackening, or perhaps an arrest of the blood-current within them, might be fairly assumed.

I feel the lack of positiveness in the above statements, but analogy led me to point them out. I do not wish to pass them for more than mere possibilities, apt

perhaps to be the forerunners of some direct and useful observations.

Secondary thrombosis in the retinal veins has not yet been described either. Of this, however, I am able to give an exquisite example occasioned by embolic occlusion of one branch of the retinal artery, and shall describe it hereafter, when I speak of this case in full.

A (b). *Primary thrombosis of the choroidal veins is wholly unknown.* *Secondary thrombosis* may arise either from embolism in the capillaries of the choroid, or be, in a retrograde direction, an extension of thrombosis in the orbital veins. One dreadful case of it I shall never forget. It occurred in the course of erysipelas and phlegmone of the orbit, running under quite the same symptoms as metastatic choroiditis. I enucleated the eyeball on account of extreme painfulness and imminent danger in the other eye. The anatomic examination stated the origin of the suppuration lying in the choroid. The transition of the suppuration from the orbit into the globe can only be accounted for by conveyance of some mischievous material through or along the blood-vessels; and this admitted, the simplest way and that most conforming to analogy is to assume a stagnation and coagulation of the blood in the vasa vorticosa, extending into the globe and causing thrombosis of the choroidal veins and capillaries, and, in consequence thereof, pyæmic abscess. The other eye of the same patient (a healthy man of twenty-four years) became similarly affected; erysipelatous swelling and formation of several abscesses in the orbit, protrusion of the eye, diffuse opacity of the refracting media, but never so intense as to prevent the ophthalmoscopic aspect of the fundus; retinal vessels hyperæmic, diffuse turbidity of the retina, entire loss of sight.

All the symptoms disappeared in the course of four weeks; the fundus only showed tortuous retinal veins; blindness remained. In this eye, too, I think thrombosis was conveyed from the orbit through the vortex veins into the eye, but did not extend in such a fatal degree to the capillaries, and became retrogressive without causing destructive suppuration.

A (c). *Primary thrombosis of the orbital veins* may happen as a result of injuries, orbital phlegmone, erysipelas, etc. Great swelling by hyperæmia and œdema, pulsation in the orbit by collateral fluxion, inflammation and formation of abscess, protrusion of the eyeball, pain and fever, will be the principal symptoms. It mostly heals, but in some cases the thrombosis may extend through the cerebral ophthalmic vein into the cavernous and other sinuses, and cause death, which occurrence is confirmed by two good examples of *post-mortem* examination. The inverse, centrifugal progression of the thrombosis from the orbital veins to those of the globe has just been spoken of.

Secondary thrombosis in the ophthalmic veins may proceed either from thrombosis of the cerebral sinuses toward the eye, or from the latter to the orbital veins and the cerebral sinuses, thus accounting for the occurrence of death after severe inflammation of and operation on the eye-ball. Besides the cerebral symptoms, those of the eye are: protrusion of the globe, hyperæmia and œdema of the orbital, conjunctival and palpebral tissue, photophobia, wide, immovable pupil, amblyopia, and paralysis of the ocular muscles.

I must pass rapidly over this highly interesting subject of the connection between ophthalmic and cerebral thrombosis, having already given a full description of it in vol. xiv., pp. 220-236, of the *Archiv für*

Ophthalmologie, in a paper on the Obstruction of the Blood-vessels of the Eye.

B (a). *Capillary embolia (or perhaps thrombosis) in the choroid is observed in nephritic retinitis.* * It was first noticed and described by H. Müller as sclerosis of the choriocapillaris. I have seen and examined one exquisitely marked specimen of it. Part of the capillaries of the choroid were filled with a uniform, finely-granulated substance, which penetrated to a certain extent into the smaller venous and arterial twigs. As a collecting trunk on both sides of the capillaries was seen choked, the one must have been an artery, the other a vein. This finely-granular mass looked very similar to molecular fat, but did not dissolve in ether, a fact which pleads for its fibrinous character. Neither red nor white blood-corpuscles, nor any other structural elements, were contained in it. The choroid was quite normal, aside from these small islands of plugged capillaries. Here is another promising subject for further studies. I may be allowed to make one suggestion on its symptomatic signification. All physicians know that every variety of diminution of sight is met with in Bright's disease. In the majority of cases this is accounted for by very marked changes in the retina, but in some nothing very conspicuous is found on ophthalmoscopic examination. In such instances, the weakness of sight is ascribed to the presence of a detrimental substance in the blood impairing the functions of the brain, and was called by Professor Frerichs *uræmic amblyopia*. It is possible that a number of such cases fall under the head of capillary embolia of the choroid.

Another group of cases of capillary embolia of the choroid most probably consists of those ocular affec-

tions we see in *cerebro-spinal meningitis* and certain other severe constitutional diseases, typhus, etc. No such eyes have been subjected to a thorough anatomico-pathological investigation, but the symptoms during life are so similar to the cases of metastatic choroiditis we witness in puerperal fever, that I have no doubt both processes are of similar origin. The destructive ophthalmia occurring in severe cases of puerperal fever is sufficiently known in its symptoms. I have myself examined three such eyes during life and microscopically after death. There are regular pyæmic abscesses formed in the choroid, which commonly destroy all the structures of the eye, but, in rare cases, are arrested at a certain stage of their course. Then the pus, investing the inner side of the choroid, becomes more or less inspissated; the eye shrinks, but retains its shape, being incurably blind. Under these conditions, I have seen about thirty eyes perish in the epidemics of cerebro-spinal meningitis which reigned, four years ago, in the upper valley of the Rhine, between Basel and Mannheim. The results of my experience and anatomical research of these diseases I have published already in an article on Metastatic Choroiditis (*Archiv f. Ophthalmol.*, xiii., pp. 72-181). Therefore I shall not dwell on the subject any longer.

B (*b*). *Capillary embolism of the retina* has never been observed, although the retina is the most fitted location to study embolism during life with the ophthalmoscope, and after death under the microscope. Since I do not know any reason why it should not occur in the retina as well as in other structures, I think that due attention paid to it will be rewarded.

C. (*a*). *Embolism of ciliary arteries* is very little studied as yet. There are only a few cases of it on record,

and they all belong to me. (*Arch. f. Ophthalmol.*, xiv., pp. 237-251.) I have seen more instances of it than I have described, but all did not allow of a definite diagnosis. The novelty and general importance of this disease will justify a brief summary of its symptoms. A patient suffering from cardiac disease, especially when combined with acute articular rheumatism, perceives quite unexpectedly, on awaking in the morning, or in the course of one or several hours during the day, a general haziness before one eye. This haziness increases in one part of the visual field very considerably, even to absolute blindness: scotoma, or defect in the visual field. The defect has in most instances a triangular shape, the apex at or near the centre of the field of vision, the basis at the periphery. Both sides of the triangle are mostly straight lines. The extent of the angle varies from 35° to 90° . In cases where the apex of the triangular scotoma does not reach the point of fixation, the patient enjoys good central vision, being able sometimes to read small print. Besides the general cloudiness and the scotoma or defect in the field, the patient complains of photopsy and chromopsy, but no pain or other symptom of distress is felt. The eye looks healthy in its external appearance, sometimes there is some discoloration of the iris and slight circumcorneal injection. Motion and tension of the globe are normal.

In ophthalmoscopic examination we find, in the first days, the background of the eye lightly veiled. Nevertheless, its details can be recognized. There invariably exists a less or higher degree of retinal hyperæmia: the optic disk appears redder, the veins are dilated and tortuous. Besides that, serous infiltration at the optic disk and in its vicinity is manifested by diffuse

grayish turbidity and swelling of the retina. Both the hyperæmia and œdema of the retina are always more marked and extended further toward the periphery, in that direction which corresponds to the scotoma in the visual field. Sometimes there is an increase of swelling and cloudiness in the retina, and a much greater dilatation and tortuosity of its veins, at one certain circumscribed spot in the affected quadrant of the fundus. This spot then is distinctly raised, and quite resembles retinal detachment. On examining the intensity of the scotoma in the visual field, we find it darkest in a place corresponding to this more infiltrated and hyperæmic portion of the retina.

Beside those symptoms, slight floating opacities in the vitreous body may occur, but I never observed ecchymoses, which is rather surprising. Probably they will be observed when more cases come to our notice. After a few days' aggravation, the mild cases begin to improve, and get well in one or some weeks. Others have a slower course, and are apt to relapse in conjunction with exacerbations of the cardiac disease. I observed one such case for eight months, until all symptoms had completely disappeared. First the general cloudiness in the field of vision fades away, then the scotoma gets gradually less dark, until at last it disappears too. The retinal hyperæmia and infiltration diminish proportionately with the improvement of sight; but, whenever there is a circumscribed raised opacity in the retina observed, it leaves very long a slight grayish-white veil on the fundus of the eye. Till now, I have not seen any marked changes in the choroïd after the affection had terminated.

Combined with these symptoms are the general symptoms of the cardiac disease and its consequences.

Nearly always there are cerebral symptoms present leading to the assumption of embolisms in the brain, and once I found the other eye had lost its sight by total embolism of the central retinal artery.

The diagnosis of embolism of a ciliary artery, after such a definite complexity of symptoms during life, has not yet been verified by autopsy. It was derived from the alterations of structure, the functional disturbance of the living eye, and the history of the disease. Cardiac disease, especially endocarditis, sudden appearance of a circumscribed scotoma or triangular defect in the visual field, hyperæmia and œdema of the corresponding part of the background of the eye, which in the retina are due to collateral fluxion, further the simultaneous occurrence of embolic symptoms in the brain, and, in one case, evident embolism of the central retinal artery of the other eye: these symptoms made the diagnosis so probable, that it might almost be called certain. But there are cases in which this series of symptoms is less complete, and the diagnosis must be made probable by exclusion of other possible affections. I think here too is opportunity for further studies, and I would particularly recommend physicians to watch those sudden obscurations in the visual field of patients affected with endocarditis.

C (b). *Embolism of the central retinal artery was first described, in 1858, by Prof. v. Graefe.* Since that time, about twenty-four cases are on record, five from myself. Of a sixth and most remarkable one I shall speak hereafter. The symptoms briefly enumerated are: sudden, almost instantaneous loss of vision without pain or inflammation. Retinal arteries extremely thin, like slender threads. Veins likewise thin, but somewhat thickening toward the periphery.

By pressure upon the globe, no change in the conformation of the retinal vessels, especially no arterial pulsation, can be produced.

After some days the region of the yellow spot becomes grayish opaque; in rare cases, some ecchymoses between yellow spot and optic disk set in. After a fortnight the retinal vessels begin to be refilled to a certain degree, pressure again produces arterial pulsation, the opacity around the yellow spot disappears. The ultimate result is atrophy of the optic nerve and total blindness. The causes are mostly endocarditis; in one case I found an aneurism of the common carotid artery. In some cases, where a cause has not been detected, atheroma of the arteries may be supposed. In one instance I found this disease occasioned by stabbing with a knife into the orbit.

Three cases are on record, in which sight was not lost; in one, from *Steffan*, it reacquired about one-tenth of the normal, in another, from *Schneller*, one half, and in the third, from myself, it was completely restored. In these cases, at least in the latter, the embolic obstruction was incomplete, and a sufficient arterial supply soon reëstablished, partly by maceration or contraction of the embolic mass, partly by collateral circulation.

Embolism of branches of the retinal artery has only twice been observed, by Prof. *Saemisch* and Dr. *Hirschmann*. There was a defect in the field of vision, extending throughout its lower half. In the first case, the principal upper artery was thin from its exit of the papilla, until at some short distance it was abruptly metamorphosed into a white cord, the result of secondary changes in the walls of the artery, common in obstructed vessels. In the second case, no such change

had taken place, but the artery appeared ever afterward as an extremely thin red thread. To these two cases I can add *a third, perhaps the most interesting of all on record.*

A lady, laboring under an acute exacerbation of endocarditis, felt on a sudden, while reading, a mist spreading over the book. On shutting the eyes alternately she found out at once that the left eye only was obscured. Two weeks afterward she came to consult me, her sight not having improved, nor got any worse either. Nothing abnormal in external appearance; motion and tension of the globe normal too. She read ordinary print with that eye, but finest with the other. The field of vision was deficient: the inner lower quadrant totally failing, the apex not exactly reaching the point of fixation. With the ophthalmoscope, I found the lower half of the optic disk and five-sixths of the background of the eye in a healthy condition, but the most remarkable changes in a triangular space, lying with its acute angle in the optic disk, while one side went horizontally outward, the other one diagonally outward and upward, defining an angle of about 50° . The retinal artery emerged in the centre of the optic disk and divided, as usual, in one upper and one lower principal branch. The latter ran its normal course, while the former was covered midway between the centre and the margin of the optic disk by a gray, slightly-elevated speck.

In the direct prolongation of the vessel, a very slender red line emerged out of the speck and ran upward as far as the length of one diameter of the optic disk. There it swelled abruptly to nearly the calibre of the corresponding lower branch, had a double outline, and continued its course toward the periphery in

a normal way. The beginning of the inner branch was marked by a short, oval, dark-red swelling, out of which three small arteries came forth.

The explanation of these conditions is the following: An embolus, from the endocardium, was carried through the arteria centralis retinæ as far as the first division of the upper of its primary branches. There it plugged one secondary branch (the upper) completely, the other (inner one) incompletely. At some distance from the optic disk, an anastomatic vessel from the choroid communicated with the upper branch, and conveyed a considerable amount of blood into the channel beyond the point of its occlusion, thus establishing a collateral circulation.

But, what was most remarkable, was a *hæmorrhagic infarctus of the retina*. I think this is *the first and only instance that a hæmorrhagic infarctus has been directly seen in the living body*. It was triangular in shape, corresponding exactly to the defect in the visual field. All the veins were engorged, dark red, and tortuous, some not to be traced up toward the papilla. Numerous smaller and larger hæmorrhagic patches encompassed and covered the small venous twigs, while the branches of the artery ran through the ecchymoses without giving rise to any extravasation. In the neighborhood, the twigs of the lower retinal veins and arteries were somewhat more swollen and tortuous than usual. The retinal tissue in the whole triangular region of the infarctus was diffusely gray. These changes extended from the optic papilla *as far as* ~~until~~ the periphery of the ophthalmoscopic field of vision. *The explanation is as follows:*

An embolus plugged the artery entirely. The

blood was driven by the elasticity of the arterial tunics into the capillaries and veins, where it stagnated, the vis a tergo being absent. The stagnating blood coagulated. Then a small amount of blood flowed into these same capillaries and veins from the neighboring twigs—collateral fluxion—causing a greater filling of the former, since the coagulated blood offered resistance to the current. This stagnating blood, and the want of supply with arterial blood, brought the walls of the veins and capillaries into unhealthy nutritive conditions; relaxation, dilatation, and rupture of the vessels, extravasation of blood, and transudation of serum, were the consequences. All this was directly ascertained with the ophthalmoscope.

The collateral circulation caused gradual absorption of the ecchymoses. The retina got more transparent, the veins smaller and straighter, and, after about six weeks, a sufficient regular current was re-established. When I saw the patient last, six months after the attack, no ecchymosis was left, the artery was in the same condition as in the commencement of the affection, the veins still somewhat tortuous, and the retina a little misty. The acuteness of vision in the centre was nearly normal; the defect in the visual field had not changed.

In conclusion, I may add yet, *that this highly interesting subject of embolism in the eye may be studied experimentally.* The late O. Weber, our much regretted professor of surgery in Heidelberg, injected serous pus into the crural vein of a cat. Two days later, death. Among embolisms in different parts of the body, there were found some in the ~~visual~~ tract of both eyes, and the retina of the right eye showed *weal*

numerous ecchymoses and exquisitely distinct emboli of the smaller arteries, their peripheral ends ischaemic, the corresponding veins engorged and some of them burst, similar conditions to those observed in the case just above described.