Fat embolism / by Robert Saundby.

FAT EMBOLISM.

By Robert Saundby, M.D. Edin., and Gilbert Barling, M.B. Lond., F.R.C.S. Eng. (Plate XIII.)

Although the subject of fat embolism has been before the profession for twenty years, and has been the subject of many important contributions to periodical literature, it has been slow in obtaining any general degree of acceptance, and is still mentioned exceptionally in our text-books.

In 1862 Zenker noticed that embolism was produced in the lungs by fat introduced into the veins in the case of a man who had been crushed, but the condition was not considered by him as of much importance. In the same year Wagner made a similar discovery, but regarded it as rather the result of a pyæmic condition than as the consequence of the crushing of fat-containing tissue. But in 1865 Wagner and Busch published further independent accounts of fat embolism, showing that it occurs in all cases of fracture of bones to a greater or less extent, that the embolism generally implicates other organs besides the lungs, and that it gives rise to symptoms which are capable of being recognised during life. They suggested that it might explain some cases of death hitherto ascribed to shock. Moreover, they showed that the embolism varied in amount in direct proportion to the extent of injury to the cancellous tissue of the bone, and that the fat passed into the circulation by the great veins and larger lymphatics. Further, they suggested that embolism may occur in cases of bone degeneration without injury. Dr. Busch's paper contained an account of a case in which death had actually resulted from fat embola; yet very little notice seems to have been attracted towards the subject until the publication of Professor Czerny's[1] paper towards the end of 1875. Czerny related the case of a man admitted with a transverse fracture of the thigh. The first abnormal symptom was the rise of temperature to 102°·6 Fahr., which took place on the day after the injury. In the evening his pulse and respirations were rather rapid; and as he was restless and unable to sleep a little morphia was given him at 9 P.M., and as this

[1] *Berlin Klin. Woch.*, 1875, Nos. 44 and 45.

had no effect it was repeated just before midnight. A few hours later his breathing became loud and rattling, and on examination he was found to be quite comatose, cyanosed, and breathing deeply and rapidly. There were loud bubbling râles in the chest, but the percussion note was clear and somewhat tympanitic. The pulse was 100, full and strong; the pupils were contracted, and insensible to light. There was no reaction to external stimuli. He died at 7.30 A.M. on the third day, about thirty-eight hours after the injury. The last symptoms observed were convulsive twitchings of the arms, profuse perspiration, and tracheal râles. At the *post-mortem* examination the lungs were found œdematous, and marked with small bright red spots and streaks, and on microscopical examination the smaller arteries and capillaries were distended with fluid fat, which was equally distributed over both lungs. Branching fat embola were found in the brain, in the pia mater, and cerebral substance, their sites being indicated to the naked eye by numerous punctiform ecchymoses. The liver and kidneys also contained a number of similar embolisms.

In 1877 Dr. Arthur Boettcher[1] reported a case of death from fatty embolism of the pulmonary vessels after a gunshot wound of the knee-joint. There was in this case an unusual abundance of subcutaneous fat, and free oil was found in the iliac veins and the cavities of the heart.

In October of the same year, Dr. D. J. Hamilton[2] published a case of laceration of the liver, followed by fatty embolism. The patient, a lad, was so little hurt that he was able to walk about, but in an hour or two he became much distressed, his breathing became embarrassed, and coma supervened, death occurring in a few hours. At the *post-mortem* examination the liver, which was peculiarly fatty, was found to have sustained a few small lacerations. The embola were discovered accidentally; they were present in the lungs and kidneys, being more numerous in the lungs.

In the following year, M. Déjérine brought before the Société Anatomique an account of two cases of fatty embolism occurring after fractures. In the first case there had been some hæmor-

[1] *Dorpater Med. Zeitschrift*, 1877, p. 326.
[2] *British Med. Journal*, Oct. 1877.

rhage, the pulse was small and frequent, the respirations were quiet, and death occurred in two hours and a half. The blood contained a large proportion of fat, and the vessels of the lungs were crowded with embola. The absence of dyspnœa and coma is noticeable, and may be held to indicate that death was due to some cause other than the embolism of the pulmonary vessels.

In the second case death occurred in thirty-six hours, and embola were found in the lungs, but the report is very brief, and does not state whether any symptoms were present during life.

In March 1879 M. Déjérine brought the subject before the Société de Biologie, and stated that since the publication of these two cases he had met with ten others in which this condition was present.[1] He found that the number of embola varied with the extent of the injury to the bone. He had made experiments on animals—first by simply fracturing the bones when the amount of embolism was very small, and afterwards by introducing a foreign body into the medullary canal, when the embola became very manifest, and the fat could be followed from the veins of the limb to the lungs. But when a substance capable of expansion, such as a tent of sponge or laminaria, instead of a piece of wood or iron, was introduced into the medullary canal, the lungs became literally injected with fat; and he considered that this indicated the probability that in man fat embolism followed the development of acute osteomyelitis, giving rise to increased pressure within the medullary cavity, so as to force the fat into the osseous capillaries.

At the Société Anatomique, a week later, M. Duret[2] reported a case of compound fracture, followed rapidly by death, in which oil was found in the veins of the limb, and free oil globules in the tissues around the wound; and he remarked, not that this was a case of death from fatty embolism, but that it illustrated the starting point of that condition and the manner in which the fat obtains entrance into the circulation.

Wiener,[3] in the course of some experiments on animals, found that the oil may be absorbed from the serous cavities (peritoneum, pleural sacs), or from the subcutaneous tissue. The

[1] *Le Prog. Méd.*, March 1, 1879.
[2] *Le Prog. Méd.*, March 8, 1879.
[3] *Archiv für Exper. Pathologie*, Band xi.

intervention of lymphatic glands does not prevent embolism. A very high degree of embolism is required to produce death, which is caused by general pulmonary œdema. The phenomena were never followed by any general rise of temperature, or inflammation in the embolised organs. The fat was found in the urine and in the urinary tubules and capillary loops of the glomeruli, so that it is excreted by this channel, according to Wiener, by a process of filtration.

Dr. Egli. Sinclair[1] has stated that fatty embolism was found in 10 per cent. of all the bodies examined in the Pathological Institute at Strasburg. He summarises the causes of this condition as follow:—(1) crushing of fleshy parts of the body, which contain much adipose tissue; (2) lesions of the marrow of bones; (3) inflammatory changes (not acute osteomyelitis) taking place in the latter. The following are the symptoms of fatty embolism:—The patient begins suddenly to feel weak; the respiration rises to about 60; the pulse is small and very frequent; the temperature is high; rattling is heard first in the bronchi, then in the trachea; there is dyspnœa, passing frequently into orthopnœa; the lips are covered with a reddish froth; the face is first pale, and later on becomes cyanotic; the extremities are cool, and the pupils contracted; the patient becomes somnolent, then comatose, and finally dies, death being in some cases preceded by vomiting and convulsions.

Jolly[2] has described three cases in which fat embola were found after simple mechanical rupture of the fat cells of the subcutaneous connective tissue. Symptoms were present during life in three cases only; in these the embola in the pulmonary vessels were very numerous.

In 1880 Mr. Southam of Manchester published a case of double amputation of the lower extremities for injury, in which death seemed to be probably attributable to this cause. Twenty-four hours after the operation the patient became restless and excited; countenance cyanosed, pulse and respiration rapid; there was no dyspnœa nor any rigors. Delirium set in, and death ensued seven hours after the commencement of these symptoms. The temperature at the time of death was 105° Fahr., and in

[1] *Correspbl. f. Schweizer Acryte*, No. 6, 1879.
[2] *Arch. für Psych.*, Band xi. p. 201.

spite of Listerian precautions, the wounds showed evidences of putrefaction. The lungs were found to contain numerous fat embola. Mr. Southam inclines to the opinion that the actual cause of death was acute septicæmia.

In 1881 Mr. Mansell Moullin[1] published twelve cases of fractures of bones and lesions of soft parts, in which he had found fat embola in the lungs. In only one of these cases were there any symptoms during life to indicate the existence of this condition, and in that case the number of embola was below the average. In one case of fracture of the cervical vertebræ high up, in which death occurred immediately, there was most abundant embolism. He draws attention to the old observation of the occurrence of an oily pellicle on the surface of the urine of fracture patients, and demonstrates the passage of the fat through the glomeruli and urinary tubules. He refers to some experiments by Scriba, who found that in order to produce death it was necessary to inject a quantity of fat equal to three times the weight of the fat contained in the medulla of the femur. He is inclined to the view that pulmonary embolism is usually free from harm, and that when ill effects ensue they are possibly due to embolism of the medulla oblongata.

The following case occurred in the General Hospital, Birmingham, for the notes of which we have to thank Mr. Howard Lowe, the resident surgical officer :—

H. G., a strong healthy looking man, 37 years of age, was admitted on June 21, 1881, with a compound fracture of the left leg, caused by a kick from a horse on the night before admission. He was a good deal addicted to drinking, and was not sober at the time of the accident. He lay by the roadside two or three hours before he was found; his leg was then bandaged up in side splints, and he was driven seven or eight miles to the hospital. On admission he seemed to be in good general health, and was very talkative; the fracture was comminuted, and the wound was filled with blood clot. The limb was placed on a back splint, and carbolic oil dressing applied. The temperature that evening was 101° Fahr. He slept well during the night.

June 22, 10 A.M., his face was rather flushed and dusky, and had a dull heavy look, but he said he felt very well. Tempera-

[1] *The Lancet*, July 30, 1881.

ture 101·4° Fahr., pulse 113, respiration 40. Worried looking, perfectly quiet, blood clot undisturbed. About an hour later he fell into what appeared to be a deep sleep. At noon he was still sleeping heavily, with rapid and stertorous breathing. Pulse soft and dicrotic, face flushed and cyanosed; could be roused with difficulty into a semiconscious state; pupils were slightly dilated while sleeping, and became more dilated when he was roused. There was abundant coarse crepitation at the bases of both lungs. Towards evening the cyanosis increased, and he could scarcely be roused at all. Temperature 101·8°, respiration 42, pulse 14°.

June 23. Cyanosis more marked. Moans, but does not speak when roused. Yawns sometimes. Makes a grimace when his medicine is poured into his mouth. Passes his urine involuntarily.

	M.	E.
T., . . .	99·8	101·6
P., ...	121	114
R., ...	...	44

June 24. Cyanosis more marked. Stupor deeper. Pupils equal and rather contracted, but dilate if an attempt is made to rouse him.

	M.	E.
T., ...	100·2	104·2
P., ...	143	160
R., ...	46	56

He died at 10.50 P.M.

At the necropsy the only naked eye appearances were that the brain substance was much injected, there were numerous small ecchymoses on the surface of the heart and lungs, and the bases of the latter were much congested and friable, but floated in water; finally, the large arteries were stained deep red. The fracture was slightly comminuted, and there was much blood extravasated around it, but there was no suppuration. On microscopical examination the lungs were found to be crowded with fat embola, the greater number being found in the small arterioles, few having reached the capillaries; in the kidneys the embola were chiefly in the vessels of the glomeruli, which were sometimes completely injected; there was no fat to be seen in

the lumen of the tubules; but some of the epithelial cells were loaded with minute globules of fat. In the heart the embola were rare.

The appearances seen in the lungs are represented in the accompanying drawing (Pl. XIII. fig. 1).

This case resembles in its chief characters the classical descriptions of death from fatty embolism already quoted, and the appearances in the lungs justify the belief that this was in fact the cause of death.

Since making this observation, we have examined the lungs in a large number of cases dying from disease or accident. Out of ten cases of wounds or injuries, embola were found in eight. The two exceptions were—1. tracheotomy; and 2. herniotomy. The eight in which embola were found were—

(1) An elderly woman, leg amputated for old standing ulceration. Death twenty-four hours after operation. Heart very fatty. Death probably from heart failure due to shock.

(2) An elderly man, fracture of the skull, with hæmorrhage from middle meningeal artery. Death occurred in twenty-four hours, from compression by the effused blood.

(3) A girl, compound fracture of forearm and of both legs, with simple fracture of both femora. Death occurred a few hours after admission.

(4) A middle aged man, fracture of ribs, and laceration of lung. Death two days after admission.

(5) A middle aged man, brought in dead, fracture of skull, with hæmorrhage compressing brain.

(6) A child, simple fracture of femur, with rupture of spleen, kidney, and diaphragm. Death occurred two days after the injury.

(7) A middle aged man, simple fracture of pelvis and femur; the injuries to the pelvis were very extensive. Death occurred two days after the injury.

(8) A middle aged man, fracture of femur and tibia, with opening into knee joint, and fractures extending into joint. Death occurred after two days.

In none of these cases were there any symptoms during life which pointed to fat embolism, and in all but two the amount of

embolism found after death was very slight. In two, however, Nos. 3 and 7, the embola were much more numerous, and this excess corresponded to the extensive character of the injuries sustained.

Yet, although in these two the embola were relatively numerous when compared with the remaining six of the series, they were very few in proportion to those visible in the case already described in which death was attributed to this cause.

In the cases of disease no fat embolism was found, with one exception to be referred to immediately. The cases in which no embola were found included a case of diabetes without milky blood, and a case of dysentery.

The exception to which reference has been already made was a case of diabetes with milky blood, notes of which have been published by Dr. Rickards.[1] In that case, the patient, a lad, died rather suddenly, without premonitory symptoms, the terminal coma being preceded by a slight convulsive seizure. At the autopsy the blood was found to be very fatty; when first seen it looked like melted strawberry ice, and on standing the surface became milky white. On collecting some of the blood, and putting it to stand in a conical glass, half its volume became milky white. The heart contained clots of white material like coagulated milk. These were apparently composed of a mixture of fat and fibrine. Under the microscope, the fat was seen to be in a state of extremely fine division, with very few large globules. We extracted some of the fat from the blood by ether, and obtained it again by evaporation. Attempts to stain the fine emulsion with osmic acid failed. The lungs and kidneys were examined for fat embola, and the appearances found are represented in the drawings (Pl. XIII. figs. 1 and 2).

In our opinion these appearances do not constitute embolisms, but are merely due to the presence of fat globules in the coagula which have formed in the vessels after death. It will be noticed that they do not distend the walls of the vessels, as may be seen in the case of death after fracture, but are evidently of less diameter than the lumen of the vessel. Moreover, it is probable that even these globules are to a considerable extent of *post-mortem* formation, due to the running together of the fine

[1] *Birmingham Med. Review*, June 1882.

oil granules when lying side by side and separated from the serum.

The relation of fat embolism to diabetes has assumed a special interest since, in July 1879,[1] the late Prof. Sanders of Edinburgh, and Prof. D. J. Hamilton, now of Aberdeen, published a paper, in which they contended that the peculiar terminal dyspnœa and coma of diabetes are due to lipæmia and fat embolism; and in support of this proposition they relate a case of diabetic coma in which the blood was fatty, and in which fat embola were found in the lungs and kidneys. These so-called embola are not, however, identical in appearance with those seen in injuries to adipose tissue. If we refer to the plates which illustrate their paper, we find that the oil globules do not as a rule fill up the lumina of the vessels, but appear to be embedded in the thrombi. Moreover, the authors state that the oil drops in the blood, which were at first small, not larger than a blood-corpuscle, had by the following day run together to form large globules. It seems, therefore, quite probable that the large globules were formed after death, and that these so-called fat embola are mere *post-mortem* thrombi with fat globules embedded in them.

Dr. Louis Starr[2] has published a case of diabetes with milky blood, in which so-called fat embola were found. But we are in doubt whether these appearances are correctly described. The drawing shows, in the words of the author, "transverse sections of the small arteries in the larger trabeculæ of fibrous tissue having their lumen congested, and among the blood-corpuscles are seen globules of fat, stained black by the acid." The oblong branching masses are probably similar masses of blood clot containing more or less fat.

Moreover, the retina was examined by Dr. A. G. Heyl, and he has described its appearance. There was no retinal lesion, no hæmorrhages, or embola, but the retinal vessels, veins, and arteries were of a light salmon colour.

Finally, the patient was very much debilitated, and croupous pneumonia was present, sufficient in amount, in Dr. Starr's opinion, to have determined the fatal issue. Here, again, there

[1] *Edin. Med. Journal*, July 1879.

[2] The *New York Medical Record*, May 1, 1880.

was undoubtedly fat in the blood, but not in such a state as to cause true embolism.

Dr. R. H. Fitz[1] has reported a case in which a few fatty embola were found in the lungs; he gives no drawings or details of their appearances, but there can be little question that a "few fatty embola" in the lungs could not give rise to the phenomena of diabetic coma.

Dr. Arthur Gamgee[2] has published an account of two cases of diabetic coma. In the first, analysis of the blood showed it to contain 13·55 parts of fat per thousand; but Dr. Dreschfield was unable to discern any embola in the lungs, kidney, or brain. In the second case the blood, *post-mortem*, was found to contain only 1·88 parts of fat per thousand, a quantity which cannot be regarded as pathological.

Dr. Frederick Taylor[3] states that the viscera of three cases of diabetes dying comatose, with milky blood, have been carefully examined at Guy's Hospital, but no embola were found.

So far, then, there has been very little confirmation of the statements of the Edinburgh pathologists. Hertz,[4] indeed, has stated that milky blood is common in drunkards and in acute pneumonia, but that he has never seen any harm result from it.

It is certainly true that some cases of diabetic coma have not milky blood, and it is only exceptionally the case that those with milky blood present anything that could be called fatty embolism. What we know of the history of fat embolism in fractures warrants our contending that much more decided *post-mortem* appearances are needed to justify our regarding it as yet proved that any case of diabetic coma has been rightly attributed to fatty embolism of the pulmonary vessels.

Dr. Howard Bendall[5] has described the occurrence of very extensive fatty embolism of the pulmonary vessels in acute farcy. The patient suffered from intense dyspnœa, but there was considerable coarse disease of the lung (pneumonia). Both Rayer and Böllinger have described this dyspnœa as of frequent

[1] *Boston Medical and Surgical Journal*, Feb. 10, 1881.
[2] *A Handbook of Physiological Chemistry*, vol. i. p. 169.
[3] *Guy's Hospital Reports*, vol. xxv. p. 158.
[4] *Deutsch. Med. Woch.* 1881, No. 27.
[5] Proceedings of the Pathological Society, *Brit. Med. Journal*, Feb. 11, 1881.

occurrence, but the latter attributes it to the coarser lesions so often present. Dr. Bendall was able to show that the source of the embola was the free oil which was formed in the intramuscular abscesses. When the pus fell into water a considerable quantity of oil rose to the surface, 10–15 minims in an ounce at least. There was besides a rapid fatty necrosis of the tissues around the pustules, and the adipose tissue had broken down, the contents of the cells running together.

This subject requires further investigation, as Mr. Stanley Boyd [1] shortly afterwards described the microscopical appearances in another case of farcy, in which the changes described by Dr. Bendall were not present.

EXPLANATION OF PLATE XIII.

Fig. 1. Fatty embola in lung after fracture. Death by coma. Hartnack, oc. 3, obj. 4. Tube drawn out.

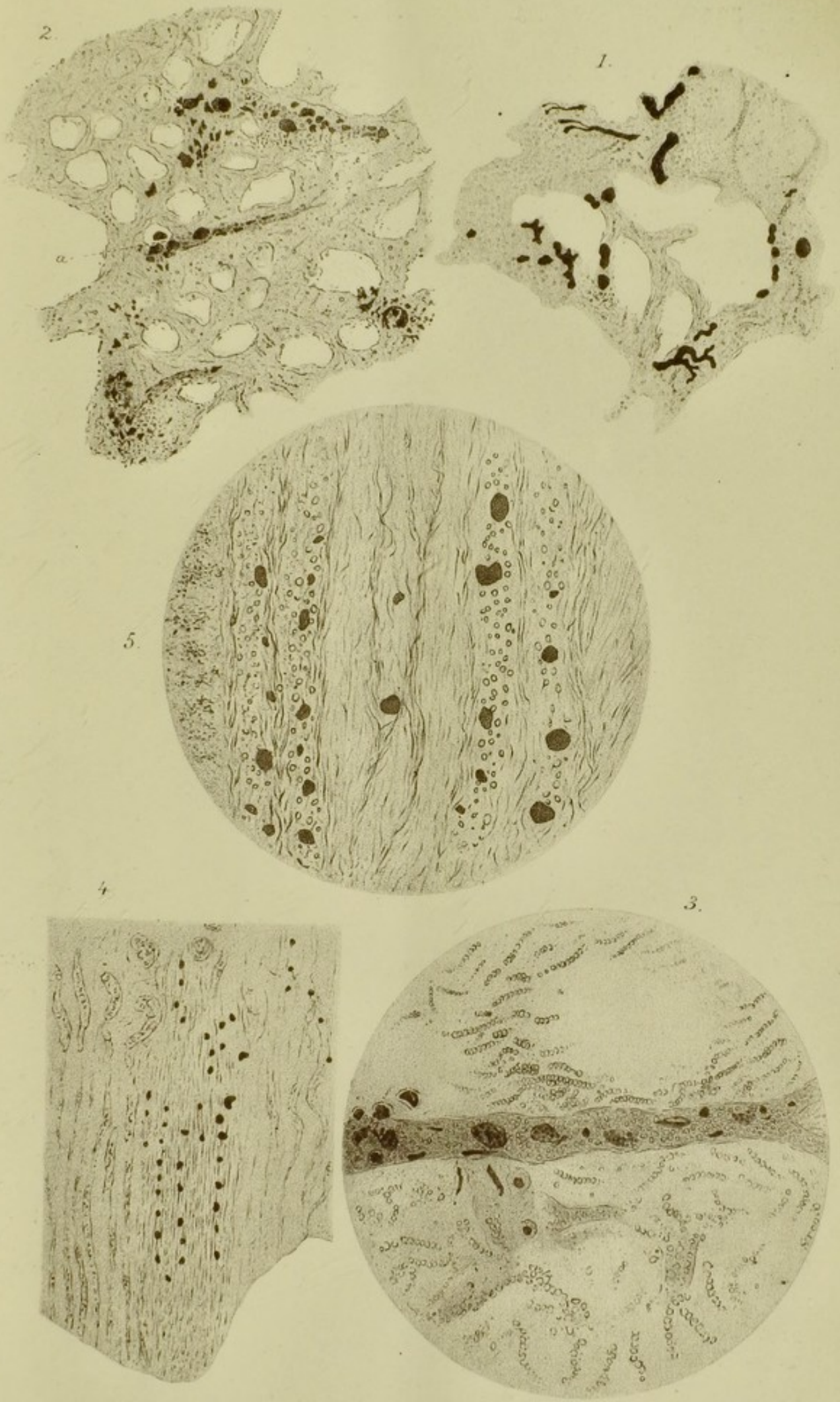
Fig. 2. Condition of pulmonary vessels in a case of diabetic coma with milky blood. Hartnack, oc. 3, obj. 4. Tube drawn out.

Fig. 3. The vessel marked *a* in the previous figure, highly magnified. Hartnack, oc. 3, obj. 8. Tube drawn out.

Fig. 4. Medullary portion of kidney from the same case of diabetes. Hartnack, oc. 3, obj. 4. Tube drawn out.

Fig. 5. Portion of the same, highly magnified. Hartnack, oc. 3, obj. 8. Tube drawn out.

[1] Pathological Society's Proceedings, *Brit. Med. Jour.*, April 15, 1882.


2.
a.
1.
5.
4.
3.