it may have been from progenitors in whom the abnormality was slight and easily overlooked.

Apert has described an apparently related form of dysostosis in which the cranial deformity is associated with webbing of the digits. To this he has given the name of acrocephalosyndactyly. Crouzon excludes this from his group on the ground that it is not hereditary, but Apert claims that it is hereditary, and that the two types are different grades of the same hereditary mutation. Syndactyly is not the only congenital anomaly which occurs in association with the dysostoses. Other suggestions of a teratological origin may be found in the deformity of the hand found in one case described by Garcin, Thuret and Rudaux (1932), in the punctate lens opacities found by Crouzon and Legras (1933), and in the coloboma of the iris in my own case.

Vogt (1933), on the strength of two cases which seem to combine the characters of Crouzon's and Apert's cases, proposes a new nomenclature distinguishing these three types; but there seems to be no justification for thus adding to the confusion which already exists through the multiplicity of names. It would be wiser, in the present state of our knowledge, to refer to these anomalies under the general title of cranio-facial dysostosis, qualified, when necessary, by reference to Crouzon's, Apert's, or some other type.

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Discussion.—Mr. J. GRAY CLEGG said that deterioration of vision in certain cases was a result of the size of the optic foramen. Variations in the dimensions of the optic foramen were found in association with skull deformities. He asked whether skiagrams had been taken in this case to ascertain the diameter of the optic foramina.

Mr. A. J. BALLANTYNE (in reply) said it had been reported that the optic foramen was deformed in some cases of this kind, but he had seen no skiagrams showing this deformity of the optic foramen.

A Clinical Study of Fifty-four Cases of Occlusion of the Central Artery of the Retina and its Branches

By JOSEPH MINTON, F.R.C.S.

HISTORICAL

GRAEFE reported the first case of embolism of the central artery of the retina in 1859.

The first criticism of this name came from the ophthalmologists of the English-speaking nations. Loring, in 1874, writing in the *American Journal of Medical Sciences*, reports five cases, and suggests that "the attack in the eye is due to trouble in the minute branches of the circulatory system" or, "to formulate more precisely, may not the trouble have been caused by some morbid local process producing a stasis of the blood at the spot, rather than by the importation of a plug from some distant source?"

Priestley Smith, in 1884, writing in the *Ophthalmic Review*, reports seven cases. Four of his cases had an associated heart lesion, but Priestley Smith suggests that the loss of vision in those patients was due to heart failure—slowing of retinal

circulation followed by formation of a local thrombus. One of his patients was a woman, aged 70, and he attributes the sudden loss of sight to arterial degeneration.

Loring's and Priestley Smith's views were later fully confirmed when the ophthalmoscopic appearances in arteriosclerosis were made more known as a result of the great work of Raehlmann and Marcus Gunn in 1889 and 1891, and also following the anatomical and pathological studies of Clemens Harms in Germany and George Coats in England.

In 1906, George Coats described in the *Ophthalmic Hospital Reports*, 24 published cases of obstruction of the central artery of the retina in which a pathological examination had been made. He concludes by saying that

> " From the clinical and pathological standpoints, a strong case has been made out for endarteritis as the cause of obstruction in many instances. Thrombosis must, no doubt, be reckoned as a very important factor in these cases, but it is not a primary factor, and, so far as the pathological evidence goes, does not occur as a causal agent except in association with disease of the vessel wall."

He does not deny that, in certain cases, embolism furnishes the explanation. This view was being shared by Haab and his pupils, Reimar, Nettleship, and many others.

In 1912, Harms published in von Graefe's *Archives* an account of 25 cases of bilateral loss of sight from obstruction of the central artery of the retina. In the following year, the views of the English ophthalmologists were crystallized at a " Discussion on vascular and other retinal changes in association with general disease," held at the Ophthalmological Society of the United Kingdom.

Coats gave a full account of the pathological changes which occur in the central vessels of the retina. There is very little to be added, in view of our present knowledge, to the pathological interpretations of endarteritis of retinal vessels, as given by George Coats in 1913. He then made his well-known statement that, in endarteritis of the retinal vessels, it seemed to be almost a matter of chance whether the thrombosis which put the finishing touches to the process occurred in the half-occluded artery or in the vein.

James Taylor, who opened the discussion, stated that, " in view of the pathological research, it has been proved beyond doubt that obliterative endarteritis, with or without thrombosis, can produce a sudden obstruction of the central artery or of any of its branches, and that this form of obstruction is a much more likely event than embolism."

It was surprising, therefore, that Leber, writing in Graefe-Saemisch's " Textbook of ophthalmology," in 1915, stated that, in his opinion, 70% of all cases of obstruction of the central artery of the retina were due to embolism associated with the disease of the heart and large blood-vessels. Leber's views, which were based on the analysis of old cases reported by Fisher from Giessen in 1892, influenced the opinions of ophthalmologists to such an extent that the old name of embolism of the central artery of the retina, which term is incorrect clinically and pathologically, is still used in standard up-to-date English textbooks, and the better name of obstruction or occlusion of the central artery of the retina has been discarded.

Angiospasm

The view that spasm of the retinal arteries is responsible for attacks of temporary total or partial blindness, was first brought forward, in 1874, by Raynaud, who then

described acute retinal arterial spasm occurring in a patient suffering from the disease which now bears Raynaud's name.

This observation has, for many years, caused a great deal of discussion. Since 1874 many observers have reported cases in which, after many attacks of transient blindness, the patients developed total blindness with optic atrophy. In 1898 Elshnig carefully described a case of sudden blindness in both eyes in a man suffering from lead poisoning. Elshnig watched for several days the spastic contraction of the retinal arteries.

A number of observers, watching cases of migraine, reported contraction of the retinal artery and its branches. Ophthalmoscopic observations were also reported in cases of epilepsy ; at the height of the attack, contraction of the retinal arteries, pallor of the retina and pallor of the disc was observed. These changes in the fundus disappeared after several hours or a day.

Priestley Smith, in the discussion in 1913 by the Ophthalmological Society, suggested that prolonged spasm of the retinal arteries is responsible, in some cases, for partial or total permanent blindness. On the other hand, Foster Moore, in 1916, discussing the pathology of obstruction of the central artery of the retina, said that " it seems very unlikely that a highly diseased vessel should be capable of spasm."

Since that date a great deal of experimental and clinical evidence has been brought forward to prove the existence of angiospasm of retinal arteries.

Cohen and Bothman claim to have proved conclusively, in 1927, that the retinal, choroidal and ciliary arteries are supplied with vasoconstrictor fibres which run in the cervical sympathetic. These have been demonstrated by the injection of adrenaline, which causes a marked vasoconstriction of all three sets of arteries, and, what is more conclusive, stimulation of the pure sympathetic nerve in both rabbits and dogs produces a marked vasoconstriction in all three sets of arteries. Redslob repeated many of these experiments with the same results.

Mylius, in 1928, published an account of ophthalmoscopic observations in eclampsia and pre-eclampsia and in polycythæmia, together with photographs in which he describes spasmodic contractions proceeding in peristaltic form along the arteries, and producing marked and irregular narrowing and even complete emptying of the blood in one or more of the arterial branches extending over short or longer distances.

Abadie, Wagener, Gipner, and many others, have reported clinical and ophthalmoscopic observations on angiospasm producing transient and permanent blindness.

In view of all this evidence, one must accept angiospasm as a contributory cause in many cases of occlusion of the central artery of the retina and its branches.

Clinical Investigation

The clinical investigation of the following group of patients was carried out at the Royal Eye Hospital, London. On searching the records of the patients for the last six years, I found that 54 patients were treated who were suffering from occlusion of the central artery of the retina or its branches. I wrote to those patients, and recently I was able to examine 27 of them. Information was received that 14 patients had died ; the remaining 13 patients could not be traced.

The first table (Nos. 1-27) deals with the important clinical data of all the patients examined in October 1936.

The second table (Nos. 28-41) refers to patients who died.

The third table (Nos. 42-54) deals with patients who could not be traced.

TABLE I

Name and year of attendance	Age	Sex	Surgeon	Vision at first attendance	Heart	B. P.	Urine	Present vision
1. H. L. 1932	55	M.	Mr. Letchworth	R. E. C. F. L. E. $\frac{6}{9}$	Normal	140/100	Normal	R. E. $\frac{6}{60}$ L. E. $\frac{6}{12}$
2. R. F. 1932	72	F.	Mr. Sorsby	R. E. No P. L. L. E. $\frac{6}{12}$. A few days later no P. L.	Normal	180/100	Normal	R. E. No P. L. L. E. No P. L.
3. W. W. 1933	54	M.	Mr. Savin	R. E. $\frac{6}{5}$ L. E. H.M.	Normal	115/70	Normal	R. E. $\frac{6}{6}$ L. E. No P. L.
4. W. T. 1933	14	M.	Mr. Letchworth	R. E. $\frac{6}{9}$ L. E. H.M.	Mitral stenosis	—	Normal	R. E. $\frac{6}{9}$ L. E. No P. L.
5. J. W. 1933	30	F.	Mr. Rycroft	R. E. $\frac{6}{5}$ L. E. No P. L.	Mitral disease	120/80	Normal	R. E. $\frac{6}{6}$ L. E. No P. L.
6. H. G. 1934	60	F.	Mr. Sorsby	R. E. H.M. L. E. $\frac{6}{18}$	Normal	190/90	Normal	R. E. $\frac{6}{9}$ L. E. $\frac{6}{6}$
7. H. C. 1934	24	M.	Mr. Sorsby	R. E. $\frac{6}{5}$ L. E. $\frac{6}{9}$	Enlarged. Mitral disease	120/80	Normal	R. E. $\frac{6}{9}$ L. E. $\frac{6}{6}$
8. H. B. 1934	54	F.	Mr. Sorsby	R. E. $\frac{6}{5}$ L. E. $\frac{6}{9}$	Enlarged. Mitral disease	160/80	Normal	R. E. $\frac{6}{18}$ L. E. $\frac{6}{18}$
9. K. N. 1934	23	F.	Mr. Savin	R. E. C. F. L. E. $\frac{6}{9}$	Mitral disease	—	Normal	R. E. C. F. L. E. $\frac{6}{9}$
10. W. M. 1934	65	M.	Mr. Savin	R. E. $\frac{6}{18}$ L. E. P. L.	Normal	130/80	Normal	R. E. $\frac{6}{18}$ L. E. P. L.
11. H. S. 1934	63	M.	Mr. Sorsby	R. E. No P. L. L. E. C. F.	Enlarged	280/150	Normal	R. E. No P. L. L. E. $\frac{6}{18}$

(Nos. 1—27)

Ophthalmoscopic appearances	Fields	Present health	Remarks
R. E. Optic atrophy. Occlusion of superior temporal and nasal arteries. Veins full. L. E. No evidence of arteriosclerosis.	R. E. Nasal sector-shaped defect with loss of central vision. L. E. Full.	Good.	—
R. E. Arteries larger than the left, collapsed with no light reflex. Veins full. Both discs atrophic. L. E. Occlusion of all branches.	None	Good.	Bilateral occlusion.
R. E. Arteries normal. L. E. Disc atrophic. Occlusion of all branches. Veins small.	R. E. Full. L. E. None.	Good.	—
R. E. Fundus normal. L. E. Disc atrophic. Arteries straight with no branching. Occlusion of macular branch. Other arteries seem to contain circulating blood.	R. E. Full. L. E. None.	—	—
L. E. Disc white. Retina has lost its transparency but retained its red colour. All arteries narrowed. Veins smaller than usual.	L. E. None.	Good.	—
Right and left eyes. Evidence of retinal arteriosclerosis with crushing of smaller veins.	Fields full.	Good.	This patient had temporary occlusion of superior branch. Seen four days after first attendance she could count fingers at 2 in., and three days after that her vision improved to $\frac{6}{18}$.
R. E. Fundus normal. L. E. Occlusion of superior temporal arteries. Other arteries normal.	L. E. Nasal sector-shaped defect.	Good.	
R. E. No atheromatous changes. Disc normal. L. E. Temporal half of disc atrophic. Occlusion of superior temporal and superior nasal arteries. (Arteries straight. No branching, no anastomosis.)	L. E. Loss of lower half of field was found in 1934 and in 1936.	Good.	
R. E. Disc white. Superior temporal artery occluded. All other arteries narrow. L. E. Fundus normal.	R. E. None.	Good.	Patient lost sight in R. E. when getting out of bed on the tenth day after her confinement with the second child. (No history of excessive hæmorrhage or puerperal fever.) She was feeling quite well at the time. No history of rheumatic fever.
R. E. Slight crushing of veins. L. E. Disc atrophic. Occlusion of all branches. Veins full.	L. E. Perception of light in temporal field only.	Good.	—
R. E. Complete detachment of retina. L. E. Intense arteriosclerosis. Temporal half of disc atrophic.	R. E. None. L. E. General constriction.	Fair.	Patient lost sight in both eyes in 1934. Left eye recovered sight after a few days. This patient developed a detachment of retina six months after occlusion of artery.

TABLE I

Name and year of attendance	Age	Sex	Surgeon	Vision at first attendance	Heart	B. P.	Urine	Present vision
12. A. C. 1934	47	M.	Mr. Bickerton	R. E. C. F. L. E. $\frac{6}{18}$	Normal	—	Normal	R. E. C. F. L. E. $\frac{6}{9}$
13. J. M. 1934	58	M.	Mr. Savin	R. E. C. F. L. E. $\frac{6}{18}$	Enlarged. (Myocardial disease)	110/70	Normal	R. E. $\frac{6}{60}$ L. E. $\frac{6}{9}$
14. H. F. 1935	73	M.	Mr. Sorsby	R. E. $\frac{6}{18}$ P. L. L. E. P. L.	Normal	220/120	Normal	R. E. $\frac{6}{24}$ L. E. $\frac{J}{60}$
15. J. I. 1935	71	M.	Mr. Griffith	R. E. No P. L. L. E. $\frac{6}{12}$	Enlarged. Accentuated second aortic sound.	170/100	Normal	R. E. P. L. L. E. $\frac{6}{12}$
16. T. J. 1935	73	M.	Mr. Sorsby	R. E. $\frac{6}{9}$ L. E. $\frac{6}{60}$	Normal	120/80	Normal	R. E. $\frac{6}{9}$ L. E. $\frac{6}{9}$
17. M. H. 1935	69	F.	Mr. Griffith	R. E. $\frac{6}{24}$ L. E. $\frac{6}{12}$	Normal	130/80	Normal	R. E. $\frac{6}{18}$ L. E. $\frac{6}{9}$
18. F. M. 1936	61	F.	Mr. Sorsby	R. E. $\frac{6}{36}$ L. E. No P. L.	Normal	215/100	Albumin present	R. E. $\frac{6}{60}$ L. E. No. P. L.
19. M. B. 1936	60	F.	Mr. Rycroft	R. E. $\frac{6}{12}$ L. E. $\frac{6}{36}$	Normal	220/130	Normal	R. E. $\frac{6}{18}$ L. E. H.M.

(*continued*).

Ophthalmoscopic appearances	Fields	Present health	Remarks
R. E. Optic atrophy. Occlusion of superior temporal artery. All other branches narrowed. Active spasm seen in arteries. L. E. Normal.	R. E. Loss of inner half of field and loss of central vision.	Good.	Loss of sight in right eye set in on the day following a severe hæmatemesis.
R. E. Disc pale. All arteries narrowed. Inferior temporal artery occluded. Inferior nasal artery narrow. L. E. Very little evidence of arteriosclerosis.	R. E. Loss of upper field.	Good.	—
R. E. Intense arteriosclerosis. L. E. Occlusion of inferior nasal and inferior temporal branches and of macular branch. Other branches narrowed. Disc pale. Veins full. Active spasm of arteries seen.	R. E. General constriction. L. E. Loss of temporal field. Central vision present.	Fair. Very forgetful.	Constriction of field in right eye is most probably the result of endarteritis without actual occlusion.
R. E. Disc atrophic. Occlusion of temporal and macular branches. Superior nasal branch larger than the rest. L. E. Crushing of veins.	R. E. Small field present in upper nasal segment. L. E. Field normal.	Good.	
R. E. Disc and vessels normal. L. E. All arteries are somewhat narrowed and discs pale.	R. E. Full. L. E. Nasal constriction.	Good Cerebral thrombosis. (Aphasia ten years ago.)	This patient had transient blindness in left eye lasting for several days. Occlusion of artery did not follow, but permanent damage to retina resulted as shown in constriction of field of vision.
R. E. Disc grey. Superior temporal artery narrowed at the disc. Inferior temporal artery, lumen irregular. Active spasm of inferior temporal artery observed. All other arteries straight with no branching. L. E. Normal.	R. E. Loss of superior nasal portion of field. General constriction. L. E. Full.	Good.	Lost sight of right eye during an attack of influenza.
R. E. Disc normal. Veins crushed. Hæmorrhages and exudates near the disc. L. E. Disc white. Extreme narrowing of superior arteries which are straight and show no branching. Lower arteries larger. No hæmorrhages or exudates.	L. E. None.	Poor. Headaches.	—
R. E. Intense arteriolar arteriosclerosis. Veins crushed. Narrowing of small branches. L. E. Occlusion of superior temporal artery and vein. New vessels formed near the disc. Worm-eaten appearance near the macula. Veins crushed. Other arterial branches present.	R. E. Central vision only.	Fair.	—

TABLE I

Name and year of attendance	Age	Sex	Surgeon	Vision at first attendance	Heart	B. P.	Urine	Present vision
20. W. A. 1936	64	M.	Mr. Savin	R. E. $\frac{6}{5}$ L. E. P. L.	Enlarged	210/110	Normal	R. E. $\frac{6}{5}$ L. E. H.M.
21. F. C. 1936	70	M.	Mr. Sorsby	R. E. $\frac{6}{12}$ L. E. No P. L.	Normal	160/100	Normal	R. E. $\frac{6}{12}$ L. E. No P. L.
22. E. C. 1936	48	F.	Mr. Savin	R. E. C. F. L. E. $\frac{6}{6}$	Normal	120/80	Normal	R. E. No central vision. C.F. in temporal field. H.M. in nasal and upper field. L. E. $\frac{6}{6}$.
23. C. M. 1936	62	F.	Mr. Sorsby	R. E. No P. L. L. E. H.M.	Normal	170/90	Normal	R. E. No P. L. L. E. P. L. present.
24. A. D. 1936	36	M.	Mr. Tyrell	R. E. $\frac{6}{6}$ L. E. H.M.	Mitral disease	—	Normal	R. E. $\frac{6}{6}$ L. E. H.M.
25. R. A. 1936	62	M.	Mr. Sorsby	R. E. H.M. L. E. $\frac{6}{6}$	Normal	160/100	Normal	R. E. $\frac{6}{60}$ L. E. $\frac{6}{12}$
26. S. B. 1936	71	M.	Mr. Sorsby	R. E. $\frac{1}{60}$ L. E. H.M.	Enlarged. No valvular disease.	160/80	Normal	—
27. L. W. 1936	73	F.	Mr Savin	R. E. No P. L. L. E. No P. L.	Normal	165/90	Normal	R. E. No P. L. L. E. No P. L.

(*continued*).

Ophthalmoscopic appearances	Fields	Present health	Remarks
R. E. Intense retinal arteriosclerosis. L. E. Disc atrophic. Superior temporal, superior nasal and inferior nasal arteries occluded. Inferior temporal artery wider than the rest with white patches of perivascularitis along its walls. Veins full.	R. E. Full. L. E. H.M. best appreciated in temporal field.	Headaches. Attacks of angina pectoris.	Patient lost sight suddenly on 16.5.36. He was admitted. Paracentesis was done and sub-conjunctival injections of acetyl-choline were given. No improvement followed.
R. E. Atheromatous changes. Arteries narrowed. Veins crushed. L. E. Superior temporal and macular branches converted into white lines. Superior nasal and both inferior arteries larger and circulation present. Disc atrophic.	R. E. Full. L. E. None.	Good.	Patient lost sight in left eye one morning in 1931 but the sight returned on the following day. A few months later he lost sight in left eye again but this time permanently.
R. E. Disc pale. Superior and inferior arteries large and full size, but straight with no branching or anastomosis in macular region. No arterial branches seen. L. E. Normal.	R.E. Loss of central vision.	Good. Sickly headaches.	Patient lost sight suddenly while walking in the street. For a few minutes she could not see with either eye. After a short while she recovered sight in left eye but became permanently blind in right eye (spasm).
R. E. Fundus cannot be seen. Dense capsule present. L. E. Disc atrophic. Arteries very small. Veins slightly larger.	None	Good.	Patient lost sight suddenly in left eye twenty-one years ago. Eighteen years ago the right eye became blind. W. R. negative.
R. E. Normal. L. E. Disc atrophic. All arteries narrowed.	R. E. Full. L. E. None.	Good.	Patient was admitted on 4.8.36. Amyl nitrite inhalations given and paracentesis was done. No improvement.
R. E. Disc atrophic. Partial occlusion of both superior vessels. Superior temporal and nasal narrowed at the disc, then get wider in the periphery. Macular branch seen only near the disc. Inferior branches larger. Active spasm of arteries seen. Veins narrowed at the disc. L. E. Disc normal. Some irregularity in the lumen of the arteries.	R. E. Loss of nasal half of field. Central vision present. Temporal field constricted. L. E. Full.	Good.	—
R. E. Arteries extremely thin. Disc atrophic. Choroidal sclerosis present. L. E. Fundus cannot be seen.	None.	Poor. Diabetes. Cerebral thrombosis (left hemiplegia) three years ago.	Patient lost sight in the right eye suddenly nineteen years ago. In left eye he lost sight suddenly six months ago, in February, 1936. He then developed secondary glaucoma following thrombosis of central retinal vein.
R. E. Arteries very narrow but not occluded. Veins full. L. E. Arteries narrow but not occluded.	None	Good.	In April 1936 patient lost sight in both eyes for fifteen minutes on two successive days. On the third day she lost sight again and has remained blind ever since. (Attacks of transient blindness and ultimate permanent blindness were most probably due to spasm of retinal arteries.)

TABLE II

	Year of attendance	Age	Sex	Surgeon	Vision at first Attendance	Ophthalmoscopic appearances at first attendance
28.	1931	81	M.	Mr. Letchworth	R. E. $\frac{6}{36}$ (lens changes) L. E. No P.L.	L. E. Disc pale. Arteries narrow. Veins full.
29.	1931	84	F.	Mr. Letchworth	R. E. $\frac{6}{24}$. L. E. No P.L.	L. E. Œdema of macula. Disc pale.
30.	1931	72	M.	Mr. Savin	R. E. C.F. L. E. $\frac{6}{18}$	R. E. Cherry red spot. Retina pale round macula. Arteries narrowed. L. E. Macular changes.
31.	1931	59	F.	Mr. Letchworth	R. E. $\frac{6}{9}$ L. E. H.M.	L. E. Cherry red spot at macula. Arteries thin.
32.	1932	60	F.	Mr. Sorsby	R. E. $\frac{6}{60}$ L. E. H.M.	R. E. Small punctiform hæmorrhages. Arteries and veins narrowed. Crushing of veins. L. E. Disc pale. Arteries narrowed. The whole of the central area is œdematous, the macular region standing out dark red.
33.	1932	72	M.	Mr. Bickerton	R. E. $\frac{6}{9}$ L. E. H.M.	—
34.	1932	68	F.	Mr. Griffith	R. E. $\frac{6}{24}$ L. E. H.M.	L. E. Retina œdematous. Cherry red spot at macula. Arteries contracted.
35.	1933	59	M.	Mr. Sorsby	R. E. $\frac{6}{9}$ L. E. H.M.	R. E. Marked retinal arteriosclerosis. Punctate hæmorrhages above macula. L. E. Arteries narrowed. Cherry red spot. Retinal hæmorrhage.
36.	1934	57	F.	Mr. Sorsby	R. E. $\frac{6}{36}$ L. E. $\frac{6}{18}$	Right and left eyes. Marked arteriosclerosis. Diabetic retinitis. R. E. Occlusion of superior temporal artery.
37.	1934	55	M.	Mr. Sorsby	R. E. H.M. L. E. $\frac{6}{9}$	R. E. Disc white. Cherry red spot at macula. Arteries contracted.
38.	1934	55	M.	Mr. Sorsby	R. E. H.M. L. E. $\frac{6}{9}$	R. E. Marked arteriosclerosis. Thrombosis of temporal tributary retinal vein. Occlusion of nasal branches of retinal artery.
39.	1934	65	M.	Mr. Letchworth	R. E. H.M. L. E. $\frac{6}{24}$	Disc pale. Hæmorrhage to the nasal side of disc.
40.	1934	64	M.	Mr. Letchworth	R. E. $\frac{6}{60}$ L. E. $\frac{6}{9}$	—
41.	1934	73	M.	Mr. Griffith	R. E. $\frac{6}{24}$ L. E. H.M.	Œdema of retina. Large cherry red spot.

(Nos. 28—41)

Date of death	Remarks
August 1936, at the age of 86. (Thirteen years after occlusion of central artery of the retina.)	The patient first attended the Royal Eye Hospital in 1923. Mr. Brookshank James then diagnosed obstruction of the central artery of the retina. Patient gave a history of having had several temporary obscurations of vision followed suddenly by loss of sight in left eye.
1933, at the age of 86. (Nine years after occlusion.)	This patient first attended the Royal Eye Hospital in 1924. Mr. Letchworth then diagnosed obstruction of the central artery of the retina. She last attended in 1931.
1934, at the age of 75. (Three years after occlusion.)	
1932, at the age of 60. (One year after occlusion.)	Chronic myocardial disease.
(Date not known.)	Patient also had diabetes.
1933. (One year after occlusion.)	
1935. (Three years later.)	
1936. (Three years after occlusion.)	
1936.	
1934. (Three months after occlusion.)	
(Date not known.)	Patient developed secondary glaucoma in R.E. The R.E. was excised. *Pathological report by Mr. Sorsby :* Secondary glaucoma. Occlusion (thrombosis) of a branch of retinal artery and vein. Considerable degree of arteriosclerosis.
April 1936.	
1935. Cerebral hæmorrhage.	
1934.	

TABLE III. (Nos. 42—54)

	Year of attendance	Age	Sex	Surgeon	Vision at first attendance	Remarks
42.	1931	61	F.	Mr. Savin	R. E. $\frac{6}{9}$ L. E. H.M.	
43.	1931	56	F.	Mr. MacCallan	R. E. H.M. L. E. $\frac{3}{60}$	
44.	1931	56	M.	Mr. Letchworth	R. E. $\frac{6}{12}$ L. E. H.M.	Lost sight in left eye for a few seconds two weeks previous to first attendance. Four days later he lost sight in left eye permanently.
45.	1932	53	M.	Mr. Griffith	R. E. H.M. L. E. $\frac{6}{9}$	Was first seen by Mr. Griffith in 1929, when obstruction of the central artery of the retina was diagnosed. Patient attended again in 1932. R. E. $\frac{6}{60}$. (Disc atrophic.) L. E. $\frac{6}{9}$.
46.	1932	56	F.	Mr. Griffith	R. E. H.M. L. E. $\frac{6}{6}$	Lost sight in right eye five years before attendance at the Royal Eye Hospital. R. E. Disc atrophic. Arteries small.
47.	1933	78	F.	Mr. King	R. E. H.M. L. E. $\frac{6}{12}$	Lost sight in right eye three months before first attendance. Disc pale. Vessels very small. Occlusion of superior temporal artery.
48.	1933	52	M.	Mr. Sorsby	R. E. $\frac{6}{5}$ L. E. C.F.	
49.	1933	72	M.	Mr. Savin	R. E. No P.L. L. E. $\frac{6}{12}$	Lost sight in right eye a day before first attendance.
50.	1933	66	F.	Mr. Bickerton	R. E. C.F. L. E. $\frac{6}{12}$	W.R. positive. Obliterative endarteritis. Lost sight five days before attendance at the Royal Eye Hospital.
51.	1934	58	F.	Mr. King	R. E. $\frac{6}{9}$ L. E. No P.L.	Lost sight in left eye two days before coming to the Royal Eye Hospital.
52.	1934	75	F.	Mr. Letchworth	R. E. $\frac{6}{36}$ L. E. H.M.	W.R. positive. Lost sight suddenly one day before attendance at the Royal Eye Hospital. Blood-pressure 220/118.
53.	1934	71	M.	Mr. Savin	R. E. $\frac{6}{12}$ L. E. H.M.	Lost sight three days before first attendance.
54.	1936	50	F.	Mr. Rycroft	R. E. $\frac{6}{9}$ L. E. C.F.	Intense arteriosclerosis with hæmorrhages and exudates in right eye.

An analysis of these tables brings out the following facts:—

Age.—Occlusion of the central artery of the retina or its branches occurred in the following age-groups:—

Age	No.	Age	No.
Under 30 ...	3	50 to 60 ...	17
30 to 40 ...	2	60 to 70 ...	14
40 to 50 ...	3	70 to 80 ...	15

Sex.—30 males, and 24 females.

Side affected.—In unilateral cases the left eye was affected in 31 patients, the right eye in 20. Three patients developed bilateral blindness as a result of bilateral occlusion of the central artery of the retina.

Heart disease.—In the five young patients valvular disease of the heart was present. One patient, aged 54, also had mitral disease. None of the other patients showed any evidence of valvular disease. Myocardial disease was present in nine patients, and was associated with intense arteriosclerosis and very high blood-pressure.

Retinal arteriosclerosis.—The majority of the patients showed evidence of retinal arteriosclerosis in the non-affected eye. There was, however, a group of patients who showed no arteriosclerotic changes in the non-affected eye.

Occlusion of a branch of the artery.—Among the 27 patients who are now alive

and were very carefully examined, I found that the superior temporal branch only was occluded in three patients. In five cases the superior temporal was occluded, together with the superior nasal branch and macular branch.

In patients who had no perception of light in the affected eye and complete optic atrophy, it was noticed that the occlusion of the retinal arteries was always more marked in the superior branches, and also in the macular branch. It seems that the superior branches are more vulnerable to the process of occlusion. In the study of thrombosis of the retinal vein and its tributaries, Foster Moore, in 1924, pointed out that the superior temporal vein is thrombosed three times as frequently as the lower branch, and suggested that the fact that arteriovenous crossings are more common in the upper temporal region of the retina accounts for the greater frequency of thrombosis of the superior temporal vein.

I cannot offer any anatomical explanation for the more frequent involvement of the superior temporal branch of the retinal artery.

Occlusion of the artery with an associated thrombosis of the vein.—In two patients, occlusion of a branch of the retinal artery occurred together with a thrombosis of a tributary of the retinal vein. In one patient, thrombosis of the retinal vein occurred in the right eye twenty-one years after loss of sight in the left eye as a result of occlusion of the retinal artery.

In only one of these patients, who had an associated thrombosis of the vein with an occlusion of the artery, was there any rise of intra-ocular pressure and development of secondary glaucoma. All other patients suffering from occlusion of retinal arteries did not develop any rise of intra-ocular tension.

It is interesting to note that, in the same period of years (1931-1936), there were 205 patients attended to in the Out-patients Department of the Royal Eye Hospital who were diagnosed to be suffering from thrombosis of the central retinal vein or its tributaries. It was George Coats who first pointed out that it is a matter of chance whether the thrombosis occurred in the half-occluded artery or vein, but this chance occurs four times as frequently in the retinal veins as it does in the retinal arteries.

Fields of vision.—The more frequent occlusion of the temporal branch accounts for the corresponding loss of the nasal field (often sector shaped) in patients with branch occlusion of the artery. It was also noticed that, in patients who only had perception of light in the affected eye, the perception was only appreciated in the temporal field (from the nasal part of the retina). This was also noticed by Fisher and Coats in 1905. Coats suggested a satisfactory explanation, namely that the capillary anastomosis between the ciliary and retinal vessel systems in this region is capable of keeping up the nutrition of a small zone of retina in the immediate vicinity of the disc.

Pathology of occlusion.—Summarizing all these facts, one is driven to the following conclusions:—

(1) Occlusion of the central artery of the retina or its branches occurs very rarely (10% of all cases) in young people, and in such cases is the result of valvular disease of the heart. True embolism of the retinal artery may be the pathological explanation of these cases. In 90% of all the cases, the condition occurred after the age of 50, and was not associated with valvular disease of the heart or gross disease (aneurysm) of the larger arteries. Most of these cases showed ophthalmoscopic evidence of endarteritis of the retinal vessels. It is therefore obvious that the sudden loss of sight had only followed a slow and gradual process of obliterative endarteritis.

Occlusion of the artery may occur at any time of the day, though, in most cases, it occurred in the early hours of the morning. The patient feels, at the time, no discomfort, headache or faintness, and it is most likely that even a small fall in blood-pressure will cause a diminished flow of blood through the retinal artery,

collapse of the half-occluded vessel and ultimately, thrombosis and permanent occlusion. Many of these patients had attacks of transient loss of sight, followed eventually by permanent blindness.

In other cases, loss of sight in one eye was followed a few days later by full or partial recovery. On the basis of these clinical facts and the ophthalmoscopic observation of the frequent occurrence of angio-spasm in the diseased retinal arteries, we must also accept the possibility of active spasm of the diseased retinal vessel acting as the exciting cause of the temporary occlusion which is, in many cases, followed by thrombosis and permanent occlusion. The clinical history of three patients points to the fact that prolonged spasm without permanent occlusion will cause permanent damage to the retina, resulting in total or partial blindness.

In a group of cases reported there was no ophthalmoscopic evidence of endarteritis of the retinal vessels. The absence of pathological changes in these arteries cannot be fully established, because, in many cases, vessels which show no opthalmoscopic evidence of disease have been found histologically to be affected with atheromatous changes. Bridgett reported in 1926 on the results of 200 consecutive necropsies performed in the Philadelphia General Hospital and Hospital of the University of Pennsylvania. Histological examinations were made of the optic nerve, with especial reference to the normal or pathological structure of the central artery of the retina. In 27 of his cases, previous careful opthalmoscopic observations by competent ophthalmologists established the fact that, in 17 cases, the visible retinal arteries were normal, and yet, at necropsy, histological examination revealed that only seven were normal. In the same group of 200 cases, Bridgett found that, in 22 instances, the vessel of one side only showed disease changes.

General health and prognosis as to life.—It is interesting to note that, in the majority of cases, the general health of the patient was in no way affected, and remained good in spite of occlusion of the central artery of the retina, and evidence of retinal arteriosclerosis. This fact bears out the modern clinical observations that arteriosclerosis has an irregular and patchy distribution.

Involvement of the retinal arteries does not in all cases indicate disease of the cerebral or coronary vessels. Bridgett, in his analysis, found that in the same number of cases sclerosis occurred in the aorta, coronary or cerebral arteries, but that only in 30% of cases were the central arteries of the retina also involved. Conversely, in about 18% of the patients with grossly normal aortæ, coronary, or cerebral vessels, sclerosis of the central arteries was found. It was also most instructive to find, in the present investigation, that, in the five young patients suffering from valvular disease of the heart, embolism of the central artery of the retina and subsequent occlusion occurred in the chronic stage of endocarditis. The patient felt quite well at the time, all are now alive and the heart is well compensated.

Many of the 54 patients with occlusion of the retinal arteries lived for very long periods after the onset of occlusion. One man is still alive twenty-one years since, though he had a cerebral thrombosis three years ago. Others lived for thirteen and nine years after sudden occlusion. Many are still alive five, four and three years since the onset.

A prognosis as regards life cannot be given in these cases of obliterative endarteritis of retinal arteries, with occlusion unless there is evidence of gross arterial disease of the coronary or cerebral vessels.

Treatment of sudden occlusion.—In 1861, Graefe performed an iridectomy for a patient suffering from embolism of the central artery of the retina. In 1884, Priestley Smith advocated paracentesis for these patients. In 1882 and 1883, Wood White and von Mauthner pointed out the value of ocular massage in restoring vision to the affected eye.

With the acceptance by many ophthalmologists of the theory of active spasm of the artery causing sudden occlusion, amyl nitrite inhalations, nitroglycerine and, recently subconjunctival injections of acetylcholine have been suggested.

In cases of genuine active spasm, these remedies seem to have helped. One must, however, accept these cures with reservations, because in cases of transient blindness recovery follows spontaneously, and, during the past century, ophthalmologists have reported many cases in which the patient suddenly recovered his vision while the instruments were being got ready for the operation.

I wish to thank Mr. Arthur D. Griffith and Mr. Arnold Sorsby for their advice and criticism, and all the members of the staff of the Royal Eye Hospital for allowing me to publish the clinical notes of their cases and for their permission to show the patients at this meeting.

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Discussion.—Mr. T. HARRISON BUTLER said that two or three years ago he had, as a patient, a master from a large public school, who had been liable, for thirty years, to evanescent attacks of blindness, their duration varying from twenty to fifteen minutes. Latterly he had had temporary obfuscations of intellect, for example, while taking his class, so that for two or three seconds he "lost himself." Abadie, in his book, said he thought thrombosis was very rare, and that nearly all these conditions were due to spasm. There was much to be said in favour of that view, because if an artery was thrombosed he (the speaker) did not see how it could, in a short space of time, become efficient again. In cases of obstruction one watched the fundus, and after a day or two the circulation was seen to have become re-established, and it was possible to see blood columns pulsating to and fro in the arteries. Circulation was re-established even though the sight might not return. He had had another patient who, for twenty minutes, lost the sight of one eye and then sight returned; he did not know whether there had been any further attacks. Of the occurrence of embolism one could not doubt. He had had a patient, brought to the Eye Hospital ten minutes after he had lost his sight, and he (the speaker) saw an embolism at the bifurcation of the artery. He performed paracentesis and gave amyl nitrite. Shown at a meeting the same day, the patient said he could see normally. He (the speaker) could not then see anything, but another observer found the embolus at the periphery of the retina. Spasm probably occurred more often than was generally supposed. It would account for cases such as he had described.

Sir JOHN PARSONS said that an optimistic paper had been written about acetylcholine by Orr and Young. He (Sir John) had tried it in one case, but the result was not very good. He would be glad to know whether it had been tried on many occasions by any members.

The PRESIDENT said he had tried acetylcholine in one case, but without benefit.

Mr. FRANK JULER said he thought that acetylcholine had been tried several times for this condition by house-surgeons at the Royal London Ophthalmic Hospital but, he believed, without any benefit.

Mr. F. T. RIDLEY said that one aspect of the subject was rare, namely, that the occlusion might follow a blow on the head. An apparently normal girl had a typical lesion of this kind following the severe bumping of her head on a telephone bracket. Compensation had been awarded in that case. He had found notes of another case, in which a man had received a glancing blow from a falling sack, but unconsciousness had not followed.

Mr. HUMPHREY NEAME said that paracentesis was worth trying in such a case, in spite of the possibility that the embolus might happen to shift of its own accord. He had a patient, over 60, who, six months before, had lost the sight of one eye. There was the same event in the other eye twenty-four hours before he came to the hospital. He (Mr. Neame) performed paracentesis, and vision returned, in forty-eight hours, to $\frac{6}{12}$. It was possible that the time of operation coincided with the shifting of an embolus of itself.

Mr. RANSOM PICKARD said that in one case he had tried acetylcholine, and it did some good, as the field was partially restored. Obviously that drug would not be of use except in cases with distinct spasm, whether or not there was partial occlusion by clot or anything else. Spasm entered into the history of many cases, because on several occasions in some cases vision had disappeared and then reappeared. That was on all fours with what happened in the brain, when there were several attacks of temporary aphasia or temporary hemiplegia. Seeing they were such transient attacks they could only be accounted for by spasm. Spasm was probably a much more frequent factor than many imagined.

One type of loss of vision which had not been referred to was that intermittent loss in people who had atheroma of the retinal vessels, and whose blood-pressure was reduced because of the failure of the heart. Those people were very liable to have intermittent blindness. If in those cases one could restore the circulation, the sight also was restored. The failure of vision was often obviously *pari passu* with the failure in the circulation.