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BLINDNESS ASSOCIATED WITH HAEMORRHAGE*

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DURING the syncopal phase which may accompany or immediately follow massive haemorrhage, it is not uncommon for the patient to complain of visual disturbances and even blindness. This condition is invariably brief and seldom exceeds one hour in duration. More often it only lasts a few minutes. Jones (1947) describes a typical case of this kind. (Also see Appendix.) Blindness of a longer duration and in most cases associated with permanent damage to the visual apparatus is a rare though well-recognised complication of massive and repeated haemorrhage.

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Although this latter variety of blindness may occur simultaneously with the haemorrhage, it occurs just as frequently after an interval of several days and even weeks. It would seem to be important to separate these two varieties of blindness in view of their different prognosis, and also in view of the fact that the former recovers with no therapy, whereas the latter variety has a uniformly bad prognosis in spite of treatment. These cases are not, however, clearly separated in the literature. It would seem that the former variety is associated with a rapid reduction in the blood-volume and a fall in blood-pressure. The latter or second variety would seem to occur when the blood-volume has returned to normal with the resulting fall in haemoglobin and plasma-proteins. However, there are other apparently essential requirements for the latter variety to occur, and they will be discussed more fully later in this paper. In the literature most of the described cases are of this second or delayed amaurosis, but for the purposes of the table in this paper they will all be grouped together, and where possible the date of onset after the last haemorrhage is given. Treatment has often been coloured by the physicians' ideas as to causation, and also limited by the means available to the earlier medical authorities who investigated the condition in any detail. Groenouw (1904) estimated that 25 per cent. of cases of amblyopia occurred during or immediately after haemorrhage, 20 per cent. occurred within 12 hours, and in more than 50 per cent. it appeared within three weeks of haemorrhage. Pergen (1896) said 21 per cent. of cases occurred simultaneously with the haemorrhage, 9 per cent. within twenty-four hours of bleeding, 51 per cent. within 7 days, 14 per cent. in the second week, and 5 per cent. in the third week. Terson (1922) from his collected cases gave 8.3 per cent. during haemorrhage, 11.6 per cent. immediately after haemorrhage, 14.2 per cent. within 12 hours, 19.2 per cent. within two days, 39.2 per cent. between three and sixteen days, and 7.5 per cent. after sixteen days.

The causal mechanism is obscure. It has been variously stated to be due to haemorrhage into the optic nerve-sheaths (von Graefe, 1860); ischaemic fatty degeneration of the optic nerve (Westhoff and Ziegler, 1899); retrobulbar neuritis (Hoffman, 1899); oedema and multiple haemorrhages (Leber, 1877); changed consistency of the blood (Wilbrand and Saenger, 1904); thrombotic foci causing degeneration (Goerlitz, 1920); toxins (Groenouw 1904, Terrien 1921, Kummel 1932, Rønne 1932, Holden 1899, and Duggan 1943). Duggan even identifies the toxin as adrenalin. Langdon (1933) believes that anaemia is the sole factor; Wolff (1935) gives arterial spasm as the cause; Hartmann and Parfonry (1934) blame vasoconstriction; acidosis, ischaemia and the effects of light (Røe,

1942); Cox (1944) thrombosis of the central retinal vein, and Fisher (1929) exudation of fluid into nerve-sheaths. According to Duke-Elder (1940) the mechanism in some ways resembles that of amblyopia due to quinine poisoning.

The main difficulty appears to lie in the fact that it is very seldom that eyes affected are available for detailed pathological examination, and then not at all at the very necessary phases in the evolution of the condition leading up to the fully developed optic atrophy.

There are numerous difficulties besetting all the above explanations, but the major difficulty appears to be to explain the late onset of the condition in so many cases. If severe anoxaemia is the cause, why does it not occur during or immediately after the haemorrhage in all or most cases; why does it so very rarely, if ever, follow massive haemorrhage in the healthy? Some authorities say that blindness never follows a single haemorrhage in a person in normal health. According to Zentmayer (1912) de Wecker, and according to Grout (1914) de Wecker, Kneis, Wilbrand and Saenger did not see a single case in the Franco-Prussian War, where visual disturbance could be attributed to surgical haemorrhage alone. Harbridge (1924) could find only 18 cases recorded in the literature between 1912 and 1924 and including the war of 1914-18. MacRae (1928) says that no instance of this form of sudden blindness was recorded in the 1914-18 war, although Terrien (1921) quotes one case. Goulden (1935) had seen records of two cases. Wolff (1935) quotes Pincus as having seen two cases following amputation.

It is probable that the amblyopia is not due to a single cause, that all the above factors participate in varying degree depending on the underlying local and systemic pathology. However, in view of our knowledge of the changes that occur in the blood after haemorrhage, it would seem that of all these changes, dilution of the blood by tissue-fluids is the one that is most likely to require some hours or even days to occur, and would most closely parallel the onset of the delayed amaurosis. In chronic illness, particularly if it is associated with recurrent small haemorrhages and restricted diet, not only would anaemia occur, but the plasma-proteins and the body-reserve of proteins would be much depleted.

If one of the important features in causing the ischaemic amblyopia to occur were oedema of the optic nerve, this might and could cause some compression of the entering, and even more of the leaving blood-vessels. We would expect this oedema to be more marked in the presence of low plasma-proteins, damage to capillary and vessel walls by ischaemia, acidosis, capillary vaso-dilatation, arteriolar constriction and compression of venules. A vicious

circle would thus ensue. The oedema fluid by compressing the outgoing veins would, if the blood were dilute and anaemic, cause an appreciable fall in the quantity of blood perfusing the retina at any instant. Holden (1899) found after experimental haemorrhage in dogs "changes indicating the presence of a non-coagulable liquid in the nerve fibre layer." Diffuse retinal oedema was also noticed by Goerlitz (1920), and oedema of the optic nerve was held responsible by Leber (1877) and Fisher (1929). Lehane (1941) quotes a case with chronic anaemia, gastro-intestinal in origin, in which transient total blindness lasting three hours occurred suddenly 15 minutes after transfusion had been completed at the rate of 2.0 c.c. per minute. Although he does not believe it is the cause, it is likely that sudden rapid increase in blood-volume above normal with resulting optic nerve oedema may have been the cause here. A similar case of post-transfusion blindness is reported by Jiron (1939).

There would thus seem to be several factors involved, each of which alone is incapable of causing the amblyopia. Anaemia alone does not appear to cause amblyopia, as common clinical experience indicates. Although optic atrophy has been described in pernicious anaemia (Cohen, 1936; Box, 1936; Panton, 1935; Kampmeier and Jones, 1938) it is extremely rare and may perhaps not be related to the anaemia but rather to the underlying sub-acute combined degeneration of the cord. Varying degrees of optic atrophy following retrobulbar neuritis had been noted in chlorosis or iron-deficiency anaemia by a few early authors (Gowers, 1904). Newton (1944) describes a case following anaemia not due to haemorrhage. Amblyopia in the nephrotic syndrome with its low plasma-protein and only mild anaemia must be extraordinarily rare, and we know that amaurosis following a single massive haemorrhage is almost unknown. A fortuitous association of these three factors would thus seem the most likely underlying mechanism. The haemorrhage presumably causes the initial though temporary generalised arteriolar vaso-constriction. The increased capillary permeability thus produced helps the other factors to set the entire cycle in motion.

If we accept the low plasma-proteins in the presence of anaemia as the essential background of the cycle, then we would expect, should the plasma-proteins be very low, sudden onset of blindness simultaneously with a sudden brisk though small haemorrhage. In other anaemic patients, in whom the plasma-protein level is not low, though tissue protein reserves are depleted, we would expect loss of sight to occur not at the time of the haemorrhage but in the post-haemorrhagic state, when the blood-volume is being made up by tissue-fluids, and hence plasma-proteins are being

depleted. Lastly, in fit, healthy, well-fed subjects such as young soldiers, even after severe haemorrhage, anaemia and blood dilution could not occur to the necessary degree causing the required fall in plasma-proteins and yet preserving life. Hence blindness does not arise.

In view of the variety of interest of this condition, two further personal cases are now described. A third case, of which full details are not available, is described in the appendix.

After the description of these two cases, the results of a survey of the literature from January, 1924, until mid-1947 are produced in table form with a brief analysis.

CASE 1.—Mr. C. B., aged 58 years—a farm bailiff—was admitted to hospital with a history of haematemesis 18 hours before admission. On examination, he was a well-covered man in moderately good physical condition; pulse 90 per minute, B.P. 120/60 and haemoglobin 2.7 gms. Apparently he had recurrent attacks of dyspepsia for some years, and a similar attack of bleeding in 1938.

A barium meal at this hospital three weeks after admission showed no evidence of a peptic ulcer. His vision had been bad for some time past, and the fortnight before admission it was definitely getting worse. Two weeks before admission "he walked into a telegraph pole and thought it was the road."

Five days after admission he had a throbbing headache and complained of gradual onset of blindness on the right side. The following day the field of vision of the right eye was restricted to the central part of the temporal field. The right pupil did not react to light. Cranial nerves were otherwise normal. On examination he was very pale, quite orientated, no tachycardia, B.P. 105/65. Heart normal. Urine contained a trace of albumin. Left pupil reacted to light, right pupil was dilated and much larger than left, irregular in outline with a very slight light response. Both fundi showed slight swelling with marked pallor of the discs, the edges of which were indistinct, and faded imperceptibly into the pale retinae. In both eyes the veins were congested and the arteries narrowed and showed a marked light reflex with nipping of veins; there were no exudates and no macular abnormality. Both eyes were presbyopic with limitation of field of vision of the right eye in all directions. But the right eye showed tiny linear haemorrhagic streaks radiating out like a fan from the papilla.

1.35 litres of blood were given by slow continuous drip. Improvement of vision rapidly began and was maintained. The visual field in the right eye increased daily. On discharge six weeks after admission his right eye had improved considerably, but there was still some limitation of the field of vision in all directions. His haemoglobin was 11.5 gm.

When seen six months later, both discs were rather pale, the right more than the left, but there was no definite evidence of optic atrophy. Vision was normal for him in the left eye, but the right eye had limitation of its field in all directions.

When heard of on August 29, 1947, four years after his first admission to hospital, he was still at full employment as a bailiff. He had no further haemorrhage or dyspepsia. He wrote and informed me that vision in his right eye was slight, but there was no apparent deterioration of the sight of his left eye.

The patient was last seen on December 13, 1947. On that day vision in his right eye was restricted entirely to perception of light in the centre of the field. The right disc was evenly pale and showed a clearly demarcated periphery (optic atrophy). The retina appeared normal except for minimal arterio-sclerotic change in the arteries. There were no exudates or haemorrhages. The left eye showed presbyopic change but a normal fundus oculi.

CASE 2.—Mr. A. G., aged 71 years—a clerk—was first admitted to hospital May 31, 1946, after a road accident. He was discharged August 22, 1946, in a walking-cast, with non-union of his leg fractures. He attended as an out-patient

in the orthopaedic department, and was re-admitted June 26, 1947, with a progressive paraplegia of two weeks' duration. Investigation suggested that this was due to neoplasm. His prostate was large and hard. Formalin-stable serum acid phosphatase was first found to be 2.0 K.A. units. A later figure was 13.0 K.A. units. Treatment was begun with stilboestrol, but in spite of large doses there was no improvement. Laminectomy revealed compression of the cord by secondary growth, probably from the prostate. He developed a urinary infection, and had recurrent

Case II.—Before Transfusion.

Case II.—After Transfusion.

attacks of haemorrhagic cystitis which responded to alkalis, fluids and sulphanilamide. In spite of a suprapubic cystotomy this haemorrhagic cystitis recurred. On July 3, 1948, and July 4, 1948, he bled severely from his bladder—this lasted for 24 hours and then ceased. On July 5, 1948, his haemoglobin was returned as 3.7 gm. In the evening of July 5, 1948, he complained of failing vision. On the morning of July 6, 1948, his sight (both eyes) was very poor—he could not read and could just distinguish outlines. He was transfused with two pints of blood on the 6th and 7th July. On the 8th he reported that his vision had improved considerably.

Retinal change at no time was marked. There was only pallor of disc and retina, and 48 hours after transfusion the pale retina became more normal in appearance. However, the discs still looked pale. Except for slight arterio-sclerotic vascular changes, there were no exudates or haemorrhages. On July 13, 1948, his haemoglobin was 7.2 gm. and he could read the large print in the daily newspaper. His blood pressure was 160/80 mm. Hg and his blood urea 41 mgm. per 100 ml.

On July 22, 1948, both fundi appeared normal and he could read small newspaper print, though for short periods only. On August 7, 1948, further vesical bleeding occurred, his haemoglobin was then 4.4 gm., and there was further deterioration of vision. Bleeding ceased in 24 hours and visual recovery began again. Discs now definitely pale. A few hard exudates, arteries very thin; veins congested; no haemorrhages. On August 11, 1948, plasma-protein estimation gave the following result: total protein 4.5 gm./100 ml.; albumin 2.8 gm., and globulin 1.7 gm. Although these proteins are not very low, we feel that during the few days that elapsed after haemorrhage they have been made up from the tissue-protein reserve, as we have endeavoured to maintain this patient on a high protein and high calorie diet. What part bone secondaries play in the blood changes is impossible to assess in this case.

In view of the definite evidence of persistent renal and vesical infection in this case it may be thought that haemorrhage was not the only factor. The low blood-globulin is rather against any marked systemic disturbance of the blood-proteins by the infection. The close relation too between the bouts of prolonged vesical haemorrhage and loss of sight clearly showed that the key mechanism was the haemorrhage, and that at first the process was reversible. This patient later died. No further macroscopical vesical haemorrhage occurred, and no further visual deterioration. However, samples of urine always contained red cells. As we were at no time really able to control his urinary infection, this could not be the cause of the loss of sight, though it was an important accessory factor. The eye was not examined after death, as permission for autopsy could not be obtained.

In this case the main features would appear to be prolonged disability from fractures, paraplegia, carcinoma of the prostate with secondaries in the cord and elsewhere, chronic vesical infection and recurrent vesical haemorrhage. All these features together did not produce loss of sight until profuse bleeding occurred, necessitating transfusion. We feel, therefore, that this can legitimately be considered a case of loss of sight due primarily to haemorrhage.

Discussion

These two cases and the third case described in the appendix illustrate many features common to this condition. The causes are respectively, gastric haemorrhage, vesical haemorrhage and uterine haemorrhage. Out of 106 cases recorded up to 1876 by Fries (quoted by Barr, 1934), 36 per cent. were due to bleeding from the stomach, 25 per cent. due to uterine haemorrhage, and 25 per cent. due to blood-letting. In a survey of the literature from 1924 until the end of 1947 we were able to find 47 cases, including the three in this paper. (See Table.) Where it was possible to ascertain the cause the percentages were as follows: gastric haemorrhage and melaena 47 per cent. (20 cases), uterine haemorrhage 32.5 per cent. (14 cases), nasal haemorrhage 9 per cent. (4 cases) and miscellaneous haemorrhages 11.5 per cent. (5 cases).

Author	Year	Age	Sex	Lesion	Time of Onset (days)	Degree of Recovery of Vision	Source
JONES, F. A. ...	1947	56	M	Gastric Haem.	8	—	<i>Brit. Med. J.</i> , 2, 477.
		70	M	"	Immediate	—	<i>Ibid.</i> 2, 477.
BLACK, J. ...	1945	53	M	Haematemesis and Melaena	—	—	<i>Ibid.</i> 2, 920.
COX, R. A. ...	1944	22	M	Trauma	?3	—	<i>Arch. Ophthalm.</i> , 32, 368.
LONG, A. E. ...	1943	52	F	Epistaxis	6	—	<i>Amer. J. Ophthalm.</i> , 26, 1179.
RØE, O. ...	1942	66	M	D.U. Melaena	2-3 weeks	Nil	<i>Acta Ophthalm.</i> , 20, 48.
TIDY, H. L. ...	1941	30	F	Post-partum	11	Complete recovery	<i>Brit. Med. J.</i> , 1, 774.
BUSHPAN, A. ...	1941	—	F	Post-partum	—	—	<i>Vestnik oftal.</i> , 18, 325.
ROBERTSON, C. K.	1941	55	M	D.U.	—	—	<i>Edin. Med. J.</i> , 48, 414.
GIQUEAUX, R. E. ...	1941	—	—	—	—	—	<i>Ann. argent. de oftal.</i> , 2, 155.
BAMFORD, C. H. and BARBER, H. ...	1940	46	M	Haematemesis	5	—	<i>Lancet</i> , 2, 715.
MITTELSTRASS, H.	1938	26	F	Post-abor.	—	Partial	<i>Deutschemed. Wöchenschr.</i> , 64, 936
WAUTERS ...	1938	—	F	Post-abor.	—	—	<i>Bull. Soc. belge. d'ophth.</i> , 76, 59.
RUGG-GUNN, A. ...	1938	47	M	Haematemesis	3-4	Nil	<i>Proc. Roy. Soc. Med.</i> , 31, 665.
		23	M	"	—	Nil	<i>Ibid.</i> 31, 665.
		18	F	Uterine H.	<1 week	Slight	<i>Ibid.</i> 31, 665.
TATARZYNSKA, H.	1938	—	—	—	—	—	<i>Polska gaz. lek.</i> , 17, 338.
DUGGAN, W. F. ...	1936	23	M	Blood Donation	2 weeks	Nil	<i>Arch. Ophthalm.</i> , 16, 380.

KORZHENYANTS, S. F.	1935	—	—	—	—	—	Sovet. vestnik oftal., 7, 391.
HARTMANN, E. and PARFONRY, J. ...	1934	—	—	—	—	Partial	Bull. Soc. d'opht. de Paris. Feb. 54.
BARR, A. S. ...	1934	32	F	Post-abor.	Several days	Partial	Amer. J. Ophthalm., 17, 396.
GOLDFEDER, A. E. and *RAPOPORT K. N. (Fuchs case) ...	1934	29	F	Post-part.	Immediate	Recovered	Klin. Monatsbl. f. Augenheilk., 93, 666.
HARTMANN, E. ...	1933	—	—	—	—	—	Bull. Soc. d'opht. de Paris. May, 347.
LANGDON, H. M. ...	1933	39	F	Uterine H.	1 week	Partial	Arch. Ophthalm., 10, 99.
de SCHWEINITZ, G. E.	1933	—	F	Post-abor.	6	Partial	Ibid. 10, 102.
HERMAN, M. ...	1933	—	F	Post-abor.	—	—	Ibid. 10, 102.
LANGERON ...	1933	64	F	Venesection	Immediate	Nil	Lyon méd., 152, 176.
WEIL P. E., BOUSSER J. and HALBRON, P. ...	1933	70	M	Nasal haem.	2	Partial	Bull. et mém. Soc. méd. d. hôp. de Paris, 49, 386.
LAPORTE, A. ...	1932	—	—	Haemat.	—	—	Prat. méd. franç., 13, 727.
GENET, L. ...	1931	40	F	Miscarriage	10	Nil	J. de méd. de Lyon, 12, 585.
SATANOWSKY, P. ...	1930	22 30	F F	Piles Uterine haem.	1 25	Slight Slight	Semana méd., 2, 956. Ibid. 2, 956.
WHITING, M. ...	1929	40 48 47 41 48	M F M M M	Melaena Melaena Melaena Haemat. Haemat.	— — — 3-14 7	Nil Nil Slight recov. Nil Nil	Tr. Ophth. Soc. U.K., 49, 144. Ibid. Ibid. Ibid.
LISTER, A. E. J. ...	1929	—	F	Uterine H.	2-3 weeks	Complete	Ibid.

Author	Year	Age	Sex	Lesion	Time of Onset (days)	Degree of Recovery of Vision	Source
GOULDEN, C. ...	1929	18	M	Melaena	—	Nil	<i>Tr. Ophth. Soc. U K.</i> , 49, 151.
COYON, A., CERISE, CLOG, W. ...	1927	—	—	Gastro-Intestinal	—	—	<i>Bull. et. mém. Soc. méd. d. hôp. de Paris</i> , 51, 53.
GRIMMINGER, W. ...	1925	38 21	M M	D.U. Melaena Epistaxis	— —	— —	<i>Z. f. Aug.</i> , 57, 106.
HARBRIDGE, O. F. ...	1924	40	F	Epistaxis	1½	Mod.	<i>Amer. Jl. Ophthal.</i> , 7, 192.
TERRIEN, F. ...	1930	58	F	Haematemesis	9 days	Slight	<i>La Presse Médicale</i> , 2, 954.
LOCKET, S. ...	1948	58 71 25	M M. F	Haematemesis Vesical Haem. Post-abor.	6 1 Immediate	Partial Partial Complete	Present article.

* These authors quote another case which I have not included in the series in view of considerable doubt. A farmer's wife aged 34 years said to have lost her sight four days after uterine haemorrhage. Transfused with 440 c.c. of blood four months after onset of amaurosis with ophthalmoscopic optic atrophy. Apparently, however, she almost completely recovered and her optic atrophy disappeared. At the time of the transfusion her haemoglobin was 66% and her R.B.C. 4,810,000.

ANALYSIS :

Total No. of cases : 47.

Age Range : 18-71 years.

Site of haemorrhage :

Gastric Haem. and Melaena ...	20
(Male 15, Female 2)	
Uterine Haem. ...	14
Nasal Haem. ...	4
Misc. Haems. ...	5

Time of onset of blindness after last haem :

Immediate 4	15%
Within first week 15	56%
Within second week 5	18%
Within third week 2	7%
After third week 1	4%

Sex Ratio : 20 males to 20 females.

Recovery :

Complete 4	= 14%
Partial 14 (5 only slight recovery)	= 50%
Nil 11	= 36%

Age :

12 under 30 yrs. (7 females)	= 34%
18 under 40 yrs. (11 females)	= 51%
17 over 40 yrs. (13 males)	= 49%
11 over 50 yrs. (7 males)	= 31%

The ages of the three cases were respectively 58 years, 71 years, and 25 years. Terson gives the age range as 2 years to 77 years, but most commonly over 40 years. In the group from 1924 to 1947 the age range was 18 to 71 years. Classically the condition mainly affects males. In our collected survey there were 20 males to 20 females, but in the very small group confined to amblyopia from gastric and duodenal haemorrhage, including melaena, the ratio was 15 males to 2 females. In my own unpublished series of 189 cases of gastric and duodenal haemorrhage the ratio of males to females was 124 to 65. This indicates that in the group of blindness due to gastric haemorrhage males are more likely than females to develop blindness.

The extent of recovery of sight is variable. In the three cases described, one developed almost total blindness in one eye. The second case improved considerably, but he relapsed with further vesical haemorrhage, whereas the third case recovered completely. According to Tidy (1941) in 33 per cent. of all cases there is complete blindness, in 50 per cent. no improvement from maximal loss, 10 per cent. recover good or complete vision, and 7 per cent. show a variable degree of improvement. Singer's statistics were— not improved 45.9 per cent., improved 39.1 per cent., complete recovery 13.5 per cent., recovery with subsequent failure 1.5 per cent. Terson (1922), however, put the figure of permanently blind at 50 per cent. and Pergen (1896) at 54 per cent. Loss of sight is bilateral in 85 per cent. of cases (Duke-Elder, 1940).

The retinae usually show marked pallor and venous congestion. Much less frequently papilloedema, venous stasis and haemorrhages occur (Hurst, 1929), but the retinae may be completely normal without any vision being present. Later the disc becomes increasingly pale, the arteries fine and narrow, and optic atrophy becomes ophthalmoscopically evident. When loss of vision is permanent, it is due to optic atrophy (von Graefe, 1860).

Treatment would seem to be a full diet, including adequate protein intake, in all those chronic diseases in which haemorrhage may occur. Should haemorrhage occur, immediate transfusion of whole blood is advisable. When transfusion has not been available during the early stages, then slow transfusion with packed red cells is indicated.

Summary

- (1) The early literature and aetiology of blindness following haemorrhage are briefly surveyed.
- (2) Three further cases are described.
- (3) An analysis of the cases in the literature from 1924 to 1947 follows.

APPENDIX

Mrs. "X," a young thin woman aged 25 years, was seen while bleeding from an abortion. She was pale and appeared almost exsanguinated. She had evidently been bleeding for some days. Ten minutes earlier she had a severe vaginal haemorrhage, and complained that she was unable to see. Rapid evacuation of her uterus was carried out, and the bleeding ceased. After she had been laid flat on her back with the foot of the bed elevated, she rapidly recovered her sight. In all she was apparently blind for 30 minutes.

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SOME OBSERVATIONS ON THE CAUSATION AND ELIMINATION OF SATTLE'S VEIL*

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IN reading this paper, I lay no claim to any new or original theory on the causation of corneal veiling. On the contrary, it is my opinion that there has been too much theorising on this subject and all too little attention paid to the practical side of the subject of fitting with which, I believe, the causes of veiling are inextricably bound up. My aim in this address, therefore, will be to impart my views on the commoner errors in *fitting* which I have found to be constantly present in those cases in which veiling occurs to any appreciable extent. In doing this I shall appear firstly to be departing from the main subject of this paper, namely, corneal veiling; and secondly, to be making an attack on the methods of fitting of certain other practitioners and other types of lenses than moulded lenses. If I convey the latter impression, I offer my apologies in advance and hasten to justify myself on the grounds that, as in all other techniques which aim at precision, there is always a correct and incorrect method of approach.

As a preface to the statements I have to make, I would like to offer some justification for the decided views I hold. I first studied the methods of fitting moulded lenses under Dr. Joseph

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