biotics is easy once the diagnosis has been established and organisms isolated, but I feel that when the patient is first seen and admitted a high dose broad spectrum antibiotic is justified but agree that it should be accompanied by fluocoxacillin. The diagnosis of necrotising fascitis is first made on clinical grounds, and it can take 24 to 48 hours for the pathognomonic clinical features to appear. I suspect that many cases are missed. I would strongly recommend that, as in this case, the microbiologists are involved from the start and that an opinion should also be sought from one's dermatological colleagues.

Disseminated intravascular coagulation (DIC) can indeed occur and cause death but was not found in the patient I described.

One should always watch out for orbital cellulitis when confronted with lid infections. However, my understanding is that necrotising fascitis is confined to the skin and should not directly affect deeper tissues or penetrate the orbital septum to cause an orbital cellulitis.

I am aware that some cases of necrotising fascitis of the eyelid have been treated conservatively and that the patient has survived, though requiring reconstructive lid surgery. However, I still feel that early surgical debridement (together with intensive antibiotic treatment) is the treatment of choice provided the diagnosis is established early enough.

I have recently heard of a case in which the affected area of skin was surgically reflected and the deep surface cleaned of inflamed and necrotic tissue. The underlying fascia was also cleaned. The skin was then laid back into position (as per a skin graft with a pedicle) and recovered well. This is an interesting approach and may be a way of avoiding the serious cosmetic consequences of excision of a large area of affected facial skin.

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Optic nerve involvement in methanol poisoning

SIR, I was very much interested to read the paper entitled 'Optic nerve involvement in a case of methanol poisoning' by Peter Naeser. This paper contains some serious errors of omission as well as commission. To set the record straight, I would like to point these out. They are as follows:

1. We produced experimental methyl alcohol poisoning in rhesus monkeys and investigated exhaustively the subject of ocular and optic nerve toxicity by conducting detailed clinical, morphological, and biochemical studies on the subject. The author makes no mention of our studies at all. Our studies proved that the principal ocular lesion seen in methanol poisoning is the development of toxic optic neuropathy; this contradicts the statement by the author that 'the optic nerve has only infrequently been investigated.' We investigated the optic nerve in detail by light and electron microscopy and found swelling of the oligodendroglial cytoplasm in contact with the axons and of the astrocytes in the retrolaminar optic nerve and the intraorbital optic nerve. We found no vascular lesions in the optic nerve on light and electron microscopy, nor on horseradish peroxidase studies.

2. The author states that 'The perfusion of the central parts of the nerve from a central optic nerve vessel with different extension may be of importance.' The author cites a 25-year-old paper by François and Neetens in support of the existence of a central artery of the optic nerve; a few months after that paper was published I pointed out the fallacies in this paper by François and Neetens and disproved conclusively the existence of any such central artery of the optic nerve, based on my studies. My detailed studies on the ophthalmic artery and blood supply of the optic nerve (to which the author makes no reference in the paper) showed that no such artery exists in man. This finding was later confirmed in studies by several other authors, including François' own group subsequently. When they stated that in their series of 40 optic nerves they did not find any central artery of the optic nerve, I thought the mythical central artery of the optic nerve expired long, long ago. The concept of a watershed zone in the retrolaminar part of the optic nerve, postulated by Rootman and Butler, totally lacks any scientific proof. Thus the basic assumptions regarding the blood supply of the optic nerve in this paper are invalid.

It is a tragedy that the subject of the blood supply of the optic nerve has been plagued for decades by a very serious problem. To explain ischaemic disorders of the optic nerve according to their preconceived theories many authors from time to time have resorted to misstating and distorting the observed facts about the blood supply of the optic nerve: to suit their own convenience they have ignored well established anatomical and physiological facts. To create anatomy to suit a pet theory instead of vice versa is the exact opposite of scientific inquiry. No matter how often they are disproved, such pet theories once published, persist like skeletons which may tumble out of the closet years later, like the mythical central artery of the optic nerve, so miraculously resurrected in this paper.

The author, based on one case, implies that optic nerve damage in methanol poisoning is due to vascular disturbance in the optic nerve. Our studies, and those by many other authors, based on much more extensive material, lend no support to this view. Thus this misleading paper, in an effort to explain the optic nerve lesions in one case, ignores all the weight of evidence available on the subject of changes in the optic nerve in methanol poisoning and on the blood supply of the optic nerve. This seriously undermines its scientific credibility.

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References


Correspondence
Correspondence


Sir, Thank you for the opportunity to reply to the query posed by Professor Hayreh.

(1) The statement in my report that ‘the optic nerve has only infrequently been investigated’ points to the human situation and not to the counterpart in the experimental animals. The lack of registration of the optic nerve changes in humans most likely reflects the insufficient enucleation technique applied in post-mortem examinations, where the nerve is usually cut close to the eye ball and the optic nerve is left in the orbit. It is true that Professor Hayreh and his group in their very thorough morphological investigation of the optic nerves from three rhesus monkeys showed swell-

Fig. 1 Cross section of the left optic nerve 2 cm behind the eye globe showing a central vessel (arrow). (×34).

Fig. 2 Larger magnification from the central part of the nerve with the small artery surrounded by necrotic axons. (×340).
ing of oligodendroglia and astrocytes in the retrolaminar and the intraorbital optic nerve.1,2 There is, however, no reference made to the optic nerve morphology within the optic canal or intracranially. Whether the changes are distributed centrally, peripherally, or evenly in the whole transverse sections is not commented on, and it is therefore difficult to compare their results with my observation.

(2) Professor Hayreh furthermore comments that I, in the discussion of my article, mention that 'The perfusion of the central parts of the nerve from a central optic nerve vessel with different extension may be of importance.' In Figs 1 and 2 in this letter, where the micrographs are taken from the left optic nerve 2 cm behind the eye (i.e., the same section as Fig. 2E in the article), such a vessel is seen. In the greater magnification the vessel can be identified as a small artery. In my case the central retinal arteries enter the optic nerve bilaterally 1 cm behind the eye, as can be seen on Fig. 2A and 2D in my article. Thus, it is shown that the central optic vessel is posterior to this in the nerve. I think that this finding is difficult to dispute despite the statements given by Professor Hayreh.

Finally I will point out that the finding of bilateral central optic nerve necrosis in methanol poisoning is new. However, its pathogenesis is still unclear.

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References

Notes
Panhellenic congress
The 22nd Panhellenic Ophthalmological Congress will be held in Athens on 25–28 May 1989. The congress will be at the Hotel Caravel, 2 Vas. Alexandrou Street, 116 10 Athens. Full details from the Secretariat, 10 Loukianou Street, 106 75 Athens, Greece. Registration fee after 28 February 1989 $250.

Institute’s 40th anniversary
Professor Adam Sillito delivered the Duke-Elder foundation lecture on 4 November 1988 as part of the celebrations commemorating the 40th anniversary of the Institute of Ophthalmology. Under the title ‘The eye’s way to the brain’, he discussed retinal redundancy, synaptic gates, and attention’s key to the mind – their implications for neuroophthalmology.