and following the bright light and blindness (Acts 22:11), would be quite unusual for the events during and after a complex partial or generalised seizure. Furthermore, he apparently was immediately and desperately aware of his deficit, unlike the typical Anton’s syndrome of cortical blindness. Finally, unlike the expected gradual resolution seen in the post-ictal states, Paul’s blindness remitted in the sudden fashion described by “immediately something like scales fell from his eyes and he regained his sight” (Acts 9:18).

The Acts of the Apostles, which records the events shaping the faith of the early Christian church, is ascribed to Luke, a physician (Colossians 4:14) who was a companion to Paul in many of the subsequent events which are described in the book. He is noted to be a careful observer of the cultural, political, and geographical facts pertinent to his story. How one interprets his descriptions of the conversion of Paul is, of course, highly dependent on one’s presuppositions regarding supernatural workings in the natural world; however, the information available does not suggest epilepsy.

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References

Landsborough replies:

I am grateful for the comments in the letter from Dr Brorson and Ms Brewer.

It is generally agreed that the Apostle Paul suffered from some kind of chronic illness or handicap which he describes as a “thorn in the flesh”. The evidence from Paul’s letters that this handicap was epilepsy is in my view substantial. But Drs Brorson and Brewer concentrate on the single event in Paul’s life on the Damascus road, recorded in the book of Acts, and I would agree with them that the possibility of his having on that occasion an attack of temporal lobe epilepsy ending in a convulsion is more speculative.

They point out that an epileptic attack involving Paul would not have involved his companions in the way described (for example, in one account they all fell to the ground, not only Paul). But this does not completely exclude the possibility of Paul’s having such an attack. Luke wrote Acts at the earliest in AD63, after Paul had reached Rome, which was about 30 years after his conversion. Oral transmission of the details of a momentous event may become modified with the lapse of time. Discrepancies appear; for example, one of the three records states Paul’s companions heard the voice speaking to Paul, another account states that they heard no voice. The differences between the records of the reactions of Paul’s companions are unimportant compared with the central fact of his conversion.

There is no mention of loss of consciousness in the records of the event, nor is there any mention elsewhere of Paul’s having a convulsion—unless the “thorn in the flesh” does indeed refer (as I think likely) to occasional convulsive seizures. Following an epileptic attack the degree of confusion is variable. Usually the patient is mentally normal. That Paul was able to continue his journey, apparently at once, to Damascus does not therefore negate his having had an ictal episode. Nor would it be unusual that Paul should retain a distinct memory of his experience.

Regarding the question of post-ictal blindness, Paul was certainly aware of his deficit—he had to be led by the hand into Damascus. Not all cases of cortical blindness display Anton’s syndrome. I agree with Drs Brorson and Brewer that the rapid return of vision, after three days, in Paul’s case is unlike the more usual gradual resolution as reported in cases of post-ictal blindness by Sadeh et al. But it cannot be said to exclude it. The categorical record of complete blindness after Paul’s aura of a flash of light and his falling to the ground, together with other already named evidence, is marginally in favour of the concept of a post-ictal complication—albeit a rare one.

The patients described by Barry et al appear to be in a different category. The emphasis is on ictal blindness rather than post-ictal, caused by focal epilepsy of the occipital lobes. If prolonged, with EEG monitoring, the term status epilepticus aamauroticus is used. The patients with postictal blindness studied by Sadeh et al were not so monitored; they argue for hypoxia, not status epilepticus, as the cause.

Natural events may influence individual decisions. Martin Luther’s experience in 1505 on a road near Erfurt, Germany, is a parallel. He was overtaken by a thunderstorm, feared for his life, was prostrated by a flash of lightning, and vowed forthwith to become a monk. A cataclysmic natural event such as a first epileptic attack may have influenced Paul at a critical point in his thoughts. The incidence of such an event in his life does not diminish the reality of his spiritual change—from which he never wavered.

Caird writes, “According to Paul himself, as well as the three accounts in Acts, the episode on the road to Damascus was a great act of God which by itself sufficiently explained the change produced in his life.”

D Landsborough

Hydrobaric oxygen and multiple sclerosis

Sir: It is pleasing that, after the positive findings of their earlier report,1 Barnes et al now recommend that further studies of hydrobaric oxygen therapy are undertaken in multiple sclerosis patients. Their failure to substantiate the patient’s reports of improvement in bladder function, which were also noted by Fischer et al, is curious. The improvement has been objectively demonstrated in one uncontrolled4 and two double-blind studies.5 6 In the UK study, recently reported by Wiles et al,5 6 the twenty patients most severely affected by bladder dysfunction were evaluated by cystometry. Of the nine patients who received hydrobaric oxygen, five showed improved bladder capacity and four were unchanged. This contrasted with the control group of eleven patients, where one patient deteriorated, one

References
improved and nine remained unchanged. This result is significant (p < 0.03), the same level of significance found by Fischer et al. and by Barnes et al in their preliminary report.

It is inappropriate, in view of the pathology found in multiple sclerosis, to expect severely affected patients to experience dramatic benefit and Schumacher has pointed out that the object of any therapy is to stabilise the disease. The chronic nature of the blood-brain barrier changes in multiple sclerosis strongly argues for the continuation of oxygen therapy, but it is difficult to test this under double-blind conditions. Nevertheless a long-term study of hyperbaric oxygen with continuation therapy has now demonstrated benefit.

Barnes et al emphasise the level of barotrauma experienced in their study, but this appears to have been provoked by pressurisation over a fixed period, as part of their double-blind protocol. To generate 21 cases of barotrauma, including the perforation of an ear drum, when it is possible to avoid this problem by using a slower pressurisation is regrettable. There are now 56 centres in the United Kingdom using hyperbaric oxygen in the management of multiple sclerosis, attended by several thousand patients. Over 500,000 individual sessions have been undertaken with no significant barotrauma.

The reduction of the amplitude of the visual evoked responses in some patients, found by Barnes et al, is attributed by them to changes in the retina. The emotive term damage is used to describe the effects, which is surely inappropriate when they were found to be reversible. Nichols et al. actually complete loss of vision in one eye of a patient with a history of retrolubar neuritis by a six hour exposure to oxygen at 2 ATA. The patient was taking part in experiments on pulmonary oxygen toxicity. The loss of vision was completely reversed over a period of 14 hours.

As Fischer et al argued, it is necessary to reiterate that the demonstration of small benefits to severely disabled patients with advanced sclerosis, indicates that studies of oxygen therapy should be undertaken when the first symptoms typical of multiple sclerosis develop. At this time the majority of patients have multiple areas affected and there is the prospect of minimising the development of the irreversible sclerosis. The use of hyperbaric oxygen therapy in the presence of focal oedema and blood-brain barrier dysfunction is based on fundamental scientific principles and supported by experimental and clinical data. Despite the benefits in chronic patients being modest, they can make important contributions to the quality of life and can generally be sustained by continuing hyperbaric oxygen therapy. In this area a neurologist (Davidson DLW, personal communication) has adopted the sensible policy of advising patients of the mild benefits from therapy and its availability in the self-help setting provided by the charity Action for Research into Multiple Sclerosis (ARMS).

Barnes et al must be congratulated for being prepared to revise their negative stance on the efficacy of oxygen supplementation in chronic multiple sclerosis, but it remains to be seen if they can reverse the damage caused by the premature conclusions of their preliminary report.

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References

Barnes et al replies:

We must point out to Dr James that we have not fundamentally changed our view about hyperbaric oxygen. The conclusion of our first paper was that "hyperbaric oxygen is unlikely to have a role in the management of a patient with multiple sclerosis". The conclusion after our final results was that we felt it "unlikely that hyperbaric oxygen would play any practical role in the management of a patient with chronic multiple sclerosis". We have not denied the subjective benefit in urinary symptoms found in some patients nor do we deny that there appeared to be a slowing of cerebellar deterioration in the treated patients. We must emphasise that the improvement of bladder symptoms was mild and of a degree that could be obtained by the prescription of an anticholinergic agent. Whether the lack of deterioration in cerebellar function is a real or statistical phenomenon must wait the results of other studies. Our study does not indicate a sub-group of patients that benefited from oxygen therapy and in practical terms we do not feel that an unpredictable and mild benefit could justify the widespread use of hyperbaric oxygen.

We must also point out to Dr James that pressurisation was not conducted over a fixed period but was dependent on patients' ability to equilibrate pressure in the middle ear. In most cases barotrauma was of grade one severity. For those who are not familiar with diving medicine terminology this simply means discomfort in the ear accompanied by injection of the tympanic membrane. It is not usually of clinical significance and our percentage of grade four barotrauma is entirely compatible with experience in diving medical practice.

We feel that retinal damage is an appropriate term to describe the effects that we found on visually evoked potential testing. Our findings were reversible and not accompanied by changes of clinical significance. We simply wish to emphasise the potential hazards of long term oxygen therapy on the vulnerable retinal circulation.