PAPERS AND ORIGINALS

Mountain sickness, retinal haemorrhages, and acclimatisation on Mount Everest in 1975

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Summary

During the 1975 British Everest Expedition, which made the first ascent of the south-west face, observations were made in relation to mountain sickness and the appearance of retinal changes. Two Sherpas with cerebral oedema and one Briton with pulmonary oedema were treated. Retinal haemorrhages occurred in four out of six Britons who were newcomers to altitudes over 6000 m (19 685 ft) but in only two out of 14 Britons who had previously visited these altitudes. Intraocular pressures during ascent to 6000 m were within normal limits. The relevance of the ocular findings to acclimatisation in previous years was examined, the results supporting the hypothesis of a "carry-over" effect from previous visits to high altitude.

Introduction

The mountain-sickness syndrome, which includes its severe forms high-altitude pulmonary oedema and cerebral oedema, has been much studied in the past decade,[1 2] and it has become clear that rapid ascent to above 4500 m (14 765 ft) is associated with a high incidence of these alarming and potentially fatal conditions. Retinal haemorrhages, which were first described in 1968,[3] have also been seen in many people above 5000 m (16 405 ft). Reports differ on whether these changes are associated with clinical mountain sickness, and there has been little work on these findings in relation to acclimatisation on previous expeditions to high altitudes. We summarise the findings on the 1975 British Everest Expedition, which made the first ascent of the south-west face (fig 1) of Mount Everest.

FIG 1—Mount Everest (8848 m; 29 028 ft) from south west. + = Camp 2 at 6500 m (21 325 ft).

Team and methods of investigation

The team consisted of 23 British climbers and 75 Sherpas, and it is relevant that, though many had had transient mountain sickness in the past, their selection was influenced by previous good performance at high altitude. The party marched in two weeks from near Katmandu (1500 m; 4920 ft) to Khumde (3800 m; 12 470 ft), and there spent three days before climbing in five days to the Everest base camp at 5400 m (17 720 ft). Thereafter we usually made a "day-return" journey to the next higher camp before sleeping there, representing at the fastest an ascent of 600 m (1970 ft) every fourth day. No diuretics were used prophylactically. Supplementary oxygen from a Blume-Robertshaw demand system was used above 7600 m (24 935 ft) when climbing and sleeping.

The British members were questioned frequently and their symptoms noted. It was difficult to apply the same detail to our Sherpas,

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but since we ran the hospitals at base camp and camp 2 (6500 m; 21 325 ft) as "open houses" with no fixed surgery hours we felt that we would see most Sherpas who had any but trivial symptoms.

Fundal examinations were carried out on all members of the expedition, usually in darkness but without mydriatics; a Keeler pantoscope was helpful in evaluating difficult cases. Retinal photographs were taken in selected members with an Olympus Pen-F fundus camera adapted to run from a 12-volt battery. Intraocular pressure was measured with a Perkins tonometer, but readings (made between 1800 and 2100 hours with subjects seated) were confined to the early part of the expedition because the investigation was unpopular.

Results

PULMONARY AND CEREBRAL OEDEMA

Three cases of pulmonary and cerebral oedema occurred.

Case 1—A 38-year-old British journalist who had never been above 5000 m became unduly breathless on reaching 5400 m and gradually became progressively worse, so that on the fourth day of his illness he was so dyspnoeic that he had to rest from typing his dispatches after a minute and a half. He complained of slight morning headache and an unproductive cough. He became cyanosed, with a resting respiratory rate of 36/min and a pulse rate of 110/min. Nocturnal Cheyne-Stokes respiration was noted. There was no evidence of heart failure, and even at his most dyspnoeic no crepitations were heard. Several retinal haemorrhages developed. He was treated with oxygen and by evacuation on foot to 4000 m (13 125 ft), but as this led to a speedy recovery no diuretics were used. Chest *x*-ray appearances at 3800 m three days after leaving the expedition were normal.

Case 2—A 28-year-old Sherpa whose previous experience had included visits to 7300 and 7600 m (23 950 and 24 935 ft) in 1974 and 1973 respectively became ill at 7300 m after he had made two journeys to this altitude in the preceding week. He complained of severe headache, and examination at 6500 m showed him to be drowsy and unduly breathless. Bilateral papilloedema, gross retinal venous tortuosity, and many retinal haemorrhages were seen (fig 2), but apart from slight unsteadiness of gait there were no neurological signs. He was treated with oxygen, frusemide, and dexamethasone and evacuated on foot to 5400 m, where in two days he made a complete symptomatic recovery. There was no retinopathy on the 12th day at 4000 m.

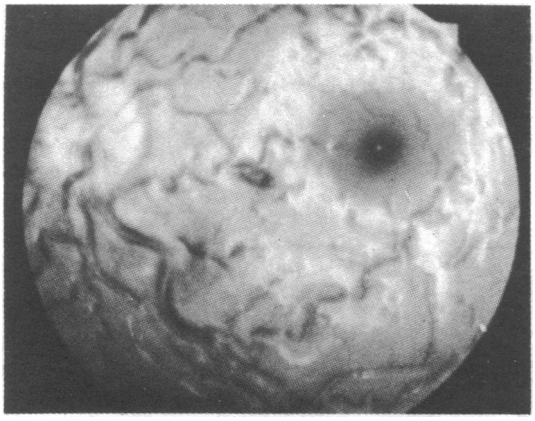

FIG 2—Case 2. Left fundus at 6500 m.

Case 3—A 30-year-old Sherpa whose previous experience had included visits to 6700 and 8200 m (21 980 and 26 905 ft) in 1974 and 1971 respectively developed an illness and retinal changes similar to those in case 2, also at 7300 m. Both he and the patient in case 2 were seen in the evening at 6500 m after descending from the south-west face and were not taken to a lower camp until dawn. Their condition greatly improved during the night before their descent, with rest, oxygen, and a single injection of dexamethasone 4 mg.

MILD MOUNTAIN SICKNESS

The remaining 22 British members of the team were generally well, but six had morning headache and lacked appetite for several days at 5400 m, and this pattern was repeated as they climbed higher. Despite frequent questioning, the other 16 admitted only to slight insomnia and occasional morning headache after the first night at a higher camp. The four who reached the summit, though perhaps stoical, denied any symptoms. There were few complaints from our Sherpas.

SYMPTOMLESS RETINAL HAEMORRHAGES

Sherpas—No symptomless retinal haemorrhages were seen in the Sherpas other than those in cases 2 and 3.

British members—In six British newcomers to altitudes over 6000 m (19 685 ft) four cases of retinal haemorrhages were seen. Two of these are described below.

Case 4—A symptomless 28-year-old man developed a haemorrhage with a pale centre (probably subhyaloid) that was first seen after he had descended from his first two nights at 6000 m. He returned to 6000 m and five days later developed several flame haemorrhages near the left optic disc (fig 3).

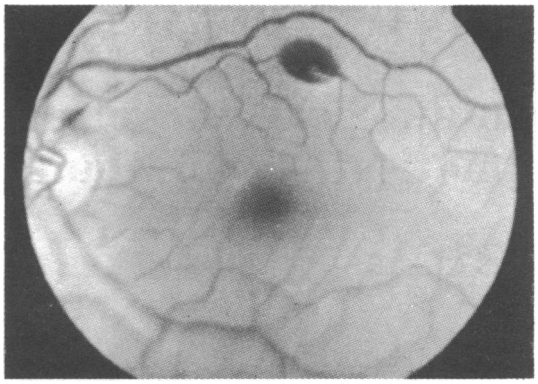

FIG 3—Case 4. Left fundus at 6500 m.

The corrected visual acuity was 6/5 and the visual fields to confrontation with a 4-mm white pin were full. He continued climbing strongly throughout the expedition and eventually spent five nights at 7750 m (24 425 ft).

Case 5—One of us (JD), aged 28, was seen at 6500 m after returning from three nights at 7300 m. He complained of slight morning headache. Several flame haemorrhages near the right optic disc were present and there was gross retinal venous tortuosity (fig 4).

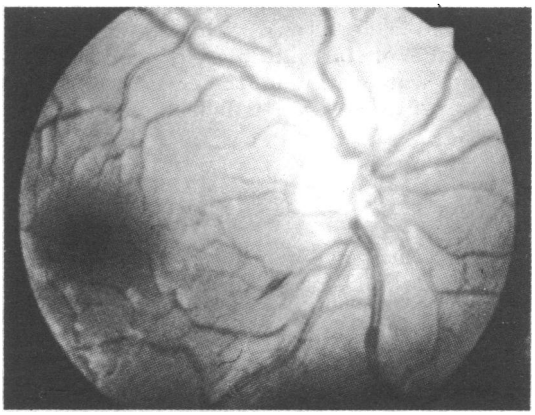

FIG 4—Case 5. Right fundus at 6500 m.

In these cases the retinal changes resolved only on descent; both men had normal fundi at 1500 m (4920 ft) two weeks after leaving the mountain. No haemorrhages were seen in the other two newcomers to heights above 6000 m, though in common with most of the members the retinal vessels were often considered to be dilated.

Among the 14 British climbers who had made previous visits to above 6000 m two cases of retinal haemorrhages were seen. This group included six who had been on Everest in 1971 or 1972, and, with two exceptions, all had climbed to over 6000 m in the previous two years. In both cases several flame haemorrhages developed near the optic

disc, one after six days above 7000 m (22 965 ft) and the other after 10 days at 6500 m; there were no symptoms in either case.

INTRAOCULAR PRESSURE

Measurement of intraocular pressure was unpopular, and only four studies were done. The results (see table) showed no noticeable fall in pressure with increasing altitude.

Intraocular pressures (mm Hg) during ascent to 6000 m (19 685 ft)

Subject		Day 1: 1500 m (4920 ft)	Day 10: 3000 m (9845 ft)	Day 20: 5400 m (17 720 ft)	Day 24: 5400 m (17 720 ft)	Comments
1	R	12	11	13	11	Retinal haemorrhages on Day 24 (case 4)
	L	11	13	12	13	
2	R	14	13	14	13	Retinal haemorrhages on Day 22. Climbed to 6000 m on Day 21
	L	12	13	13	14	
3	R	11	12	11	11	No haemorrhages
	L	12	12	11	12	
4	R	13	12	11	14	No haemorrhages. Climbed to 6000 m on Day 23
	L	12	11		12	

OTHER OCULAR OBSERVATIONS

Two members of the expedition used "permanent" soft contact lenses (Permalenses) up to 7300 m (23 950 ft). This is the subject of a separate report.[4] Two members of the party (one emmetrope and one myope who did not use contact lenses) noted quite independently difficulty with accommodation during the journey back to 4000 m at the end of the expedition. This recovered after several days. Neither had had retinopathy.

OTHER ILLNESSES AT HIGH ALTITUDE

Few members escaped the well-recognised and persistent high-altitude cough, which is associated with mouth breathing and cold, dry air. Those who used oxygen (there was no humidifier in the set) were particularly badly affected. Numerous remedies were tried but the most rewarding was a "Dr Nelson's Inhaler" (often communal) filled with hot water, 5 ml of rum, and Mac lozenges.

There were three cases of mild frostbite to the fingertips. "Frost-numb"[5] of the feet occurred in four members, one of whom had had severe frostbite in 1974, but sensation returned to normal six weeks after returning to Britain. The remarkable survival of two climbers who bivouacked without oxygen on the south summit of Everest has been mentioned elsewhere.[6]

One Sherpa had a severe attack of migraine at 6500 m and there was one case of renal colic at this altitude.

Discussion

PULMONARY AND CEREBRAL OEDEMA

The single case of pulmonary oedema in the British journalist was similar to the many clinical descriptions[7] of this syndrome, but we are unaware of a previous description of cerebral oedema with retinopathy in Sherpas, who are natives of altitudes of about 4000 m (13 125 ft). Although in South America severe mountain sickness has been noted in natives returning home to the highlands after several months,[8] this has not been mentioned in Nepal. Both of our Sherpa patients had spent several months in Katmandu (1500 m; 4920 ft) before the expedition.

In the treatment of these syndromes it cannot be emphasised often enough that rapid descent is probably the most important measure and that in prevention gradual ascent is the most sure way to avoid them. On our return from Everest it was alarming to see about 100 trekkers planning to do the last stages of our approach march in under half the time we had taken two months before.

ACCLIMATISATION IN BRITISH CLIMBERS WITH HIGH-ALTITUDE EXPERIENCE

Many climbers from Britain have noted that on their annual or biennial visits to the Himalayas they seem to perform better on later expeditions. One of us (CC), who has visited the same valleys at 5000 m (16 405 ft) three times in the past 10 years, noted that he acclimatised more rapidly on the most recent trip. It was striking that on Everest the "British veterans of altitude," though not older than the newcomers, complained scarcely at all of symptoms of mild mountain sickness and could usually do more sustained work. The physiology of this apparent "carry-over" effect of acclimatisation is unexplained.

RETINAL HAEMORRHAGES

Several recent reports[9–11] have discussed the pathogenesis of retinal haemorrhages, suggesting causative roles for increased retinal blood flow, dilatation of vessels in response to hypoxia, and increased venous pressure, either caused locally by cerebral oedema or transmitted by coughing or straining. Changes in intraocular pressure have also been questioned.[12] Some authors emphasise the high incidence of associated headache,[12] and in the management of the condition advice varies between regarding haemorrhages as a medical emergency[10] and as a common clinical sign without ominous implications.[9]

From our few observations it seems that the appearance of isolated retinal haemorrhages is not necessarily a warning sign of impending cerebral oedema. Probably in these symptomless cases the changes are related to increased blood flow and dilatation of vessels, whereas in symptomatic cerebral oedema retinal venous obstruction becomes more important. We found no noticeable fall in intraocular pressure, and it is hard to believe that ocular hypotony contributes to the clinical picture.

It was of great interest that the haemorrhages were seen so rarely in those accustomed to altitudes above 6000 m (19 685 ft), and it perhaps supports the hypothesis that the effects of previous acclimatisation are longlasting and that the response of the retinal circulation to hypoxia may differ in those who have been to high altitudes in previous years.

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