Chapter 21

THE EYE AT ALTITUDE

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Abstract
High altitude retinopathy (HAR) was first described in 1969 as engorgement of retinal veins with occasional papilloedema and vitreous hemorrhage. Since then various studies have attempted to define the incidence, etiology and significance of this phenomenon, usually with small numbers of subjects. Recently studies on relatively large groups of subjects in Nepal, Bolivia and Tibet have confirmed that the retinal vasculature becomes engorged and tortuous in all lowlanders ascending above 2500m. Sometimes this leads to hemorrhages, cotton wool spots and papilloedema, which is the pathological state better known as high altitude retinopathy. These studies have also shown a significant change in both corneal thickness and intraocular pressure at altitude. The retinal blood vessels are the only directly observable vascular system in the human body and also supply some of the most oxygen-demanding tissue, the photoreceptors of the retina. New techniques are being applied in both hypobaric chamber and field expeditions to observe changes in retinal function during conditions of hypobaric hypoxia. This work allows better advice to be given to lowlanders traveling to altitude either if they have pre-existing ocular conditions or if they suffer from visual problems whilst at altitude. This especially applies to the effect of altitude on refractive eye surgery and results of recent studies will be discussed so that physicians can advise their patients using the latest evidence. Retinal hypoxia at sea level accounts for the developed world’s largest cause of blindness, diabetic retinopathy. The investigation of retinal response to hypobaric hypoxia in healthy subjects may open new avenues for treatment of this debilitating disease.

Key Words: altitude, high altitude retinopathy, corneal pachymetry, intraocular pressure, refractive surgery
INTRODUCTION

The eye, like every other organ, is affected by the hypobaric hypoxia of high altitude. This can cause decreased vision which can be life-threatening in a mountain environment, and is of interest to the clinician treating hypoxic eye disease at sea level.

There have been no previous measurements of corneal thickness at altitude in man; studies of intraocular pressure (IOP) have been hampered by small numbers of subjects and hostile conditions. High altitude retinopathy (HAR) was first recognized in 1969(71) but since then many studies have tried to assess its incidence and clinical significance with little success.

The preliminary results of ocular studies carried out on three large medical expeditions are discussed in this paper, the Medex expedition to Chamlang base camp in Nepal, the Apex 2 expedition to Chacaltaya in Bolivia and the Irvine Lovett Everest Expedition to the north side of Mount Everest in Tibet. These research expeditions had large numbers of subjects and different ascent profiles; as such they provided an excellent model for studying the effect of altitude on the eye.

An excellent review is available of the risks associated with ascent to altitude with pre-existing ocular conditions, so this will not be covered.(46) However the popularity of refractive surgery amongst high altitude mountaineers warrants discussion, as this has opened up new potential visual problems.

INTRAOCULAR PRESSURE

Intraocular pressure (IOP) at high altitude has been the subject of controversy for many years. In 1918 Wilmer and Berens measured IOP in 14 aviators in a hypobaric chamber but came to no significant conclusion.(83) More recently some groups have found decreased IOP,(10) whereas others have found increased IOP,(15) and normal IOP.(5, 17) Butler et al reported a significant relationship between IOP and flame hemorrhages associated with high altitude retinopathy(14) and one previous study showed a reduction in IOP that occurred within hours of ascent and recovered during acclimatization.(18)

IOP is determined by the rate of aqueous secretion, the resistance encountered in outflow channels and the level of episcleral venous pressure. The distribution of IOP within the normal population has a range of 11mmHg to 21mmHg (mean 16±2.5mmHg).(37) However IOP varies with the time of day, heart beat, blood pressure and respiration. The mean range of diurnal IOP fluctuations in normal eyes is 5mmHg with a tendency to be higher in the morning and lower in the afternoon and evening. It is therefore important to standardize the time of day when IOP readings are taken.

Approximately 80% of aqueous is produced by the non-pigmented ciliary epithelium as a result of an active metabolic process.(37) Therefore aqueous secretion should be diminished by factors that inhibit active metabolism such as hypoxia and hypothermia. Aqueous drainage is also partially an active process. There is no data on IOP changes in other chronic hypoxic conditions at sea level, such as chronic obstructive pulmonary disease and congenital cyanotic heart disease, and subsequently it is not known at what arterial oxygen saturation the autoregulatory control of IOP fails.

During the Apex 2 expedition, the opportunity arose to measure the IOP of 104 healthy