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VP News



Venus Transit – Training Programmes

Vigyan Prasar, in association with NCSTC, has launched a country-wide programme on popularization of science and technology with activities built around the on-coming transit of Venus on June 08, 2004. The first of a series of training programmes for Master Resource Persons for the Southern Zone was organized at Hyderabad on March 18-19, 2004. Some 45 participants from Andhra Pradesh, Tamil Nadu, Kerala, Karnataka, Andaman & Nicobar Islands and Pondicherry participated in the programme. A.P. Council for Science & Technology were the local host. Professor Naidu, Member Secretary, APCOST, also participated in the programme.

Similar programme for the Western Zone was organized at Mumbai during April 1-2, 2004. Some 42 participants from Gujarat, Maharashtra, Rajasthan, Madhya Pradesh, Silvassa, and Dadar & Nagar Haveli participated in the programme. The programme was organized at Nehru Science Centre, Mumbai, jointly with S&T Cell, Government of Maharashtra. The programme was inaugurated by Shri M.V. Kamath, President, Vigyan Prasar. The inaugural talk was delivered by Prof. S.M. Chitre, well-known Astrophysicist. Shri Khullar, Secretary (S&T), Government of Maharashtra, Shri Routella, Director, Nehru Science Centre, and Dr. A.V. Sapre of S&T Council, Government of Maharashtra, were present during the inaugural session. Shri Anuj Sinha, Head, NCSTC, was present in the valedictory session.

In both the programme the participants were given five resource articles, Venus transit activity kit and a CD containing six slide shows on different aspects of Venus Transit as resource material.

Vigyan Rail on the Move

During the month of March 2004, Vigyan Rail travelled to Patna, Durgapur, Ranchi (instead to Hatia), Howrah, Bhubaneswar, Cuttack (additional stop), Vishakhapatnam, Durg, Nagpur, Secunderabad, and Tirupati. As usual, Vigyan Rail has continued to secure a huge response. In particular, Patna, Durgapur, Bhubaneswar, Vishakhapatnam, and Durg witnessed huge crowds to visit the Vigyan Rail – Science Exhibition on Wheels. Vigyan Rail has by now completed its run in the North, the North-East, the East and has entered the South.



Vigyan Rail at Vishakhapatnam

... think scientifically, act scientifically... think scientifically, act scientifically... think scientifically, act...



The Sixth Wave

Over the last 600 million years of the geological history, the mass extinction has been witnessed five times though separated by millions of years. The Palaeozoic era (590 to 248 million years ago) witnessed the mass extinction thrice - during the period 505 to 438 million years ago (Ordovician period), during the period 408 to 360 million years ago (Devonian period), and during the period 286 to 248 million years ago (the Permian period). It was during these periods that several invertebrate and vertebrate groups of animals, fish and amphibians evolved and died. The first forests also appeared in this period. In particular, during the Permian period, the climate was dry and hot causing extinction of many marine animals and proliferation of reptiles. The next two waves of extinction were in the Mesozoic era (248-65 million years ago) – during the period 248 to 213 million years ago (Triassic period) and during the period 144 to 65 million years ago (Cretaceous period – between Jurassic and Tertiary periods). It was during this era that dinosaurs became numerous. The climate was warm and sea level rose. First flowering plants emerged. Domination of dinosaurs continued, but they died out towards the end of the Cretaceous period. This is said to be due to the catastrophic impact of one or more meteorites. A drastic climate change is also attributed to the extinction of dinosaurs and many other organisms at this time. Indeed, the possible causes of the five waves of mass extinction were natural - global warmings and coolings, devastating meteorite showers, volcanic activities and so on.

However, disappearance of some species or what is also called extinction, and appearance of others is a natural process that forms the basis of organic evolution. According to the fossil records, no species has yet proved immortal. What is interesting is the fact that as few as only 2 to 4 per cent of the species that have ever lived are believed to have survived till date. The remainder are extinct - the vast majority having disappeared long before the arrival of human beings!

In a recent issue of the journal *Science*, an extensive study on the extinction of birds, butterflies and vascular plants in Britain has been presented. The study is based on large sets of data collected over the past 20 to 40 years in England, Wales and Scotland and analysed at the Natural Environment Research Council Centre for Ecology and Hydrology in Dorchester, UK. If the results are to be believed, then, a *sixth massive extinction event in the*

history of life is upon us yet again. The study shows a 28 per cent decline of native plants, a 54 per cent decrease in abundance of native birds and a 71 per cent decline of butterflies! There appears to be a concrete evidence that insects which account for more than half the described species on Earth are disappearing faster than the birds! This supports the view that the world is indeed on the verge of another great species wipeout.

The rapid loss of species of flora and fauna on the Earth that we are witnessing today is estimated to be between 1,000 and 10,000 times higher than the *background* or expected natural extinction rate - estimated at one species every four years. Why is it so? What could be the reason for the unusual rate of extinction of birds, butterflies and plants? Something as subtle but widespread as habitat loss and degradation because of the human activity could be the plausible reason, compounded by depletion of the ozone layer and greenhouse effect, deforestation and toxic pollution of the soil and water. Over-exploitation of resources like water and forests, agricultural activities, extraction (mining, fishing, logging, harvesting etc) and development (human settlements, industry and associated infrastructure) – all have an adverse impact. Habitat loss and fragmentation leads to the formation of isolated, small, scattered populations. These small populations are increasingly vulnerable to inbreeding depression, high infant mortality and consequently, in the end, possible extinction. Unlike the mass-extinction events of geological history that snuffed out innumerable species from the Earth five times earlier, the current extinction phenomenon is one for which a single species – human beings - appears to be almost wholly responsible for the sixth extinction crisis.

India's animal species account for 7.31 per cent of the faunal species in the world and the flora account for 10.78 per cent of the global total. About 33 per cent of the country's recorded flora are concentrated mainly in the North-East, Western Ghats, North-West Himalayas and the Andaman and Nicobar islands. However, this rich biodiversity of India is under severe threat owing to habitat destruction, degradation, fragmentation and over-exploitation of resources. According to the Red List of Threatened Animals published in 2000 by International Union for Conservation of Nature, India's 44 plant species are

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Eye, Vision and Venus Transit

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The transit of Venus will take place on 08 June 2004 and will be seen throughout the country. Transits of Mercury are relatively frequent occurring 13-14 times a century. However, transits of Venus are extremely rare. The oncoming transit of Venus will occur after a gap of nearly 121 years. Surely, it would be a great occasion to witness it. True, several websites would webcast the entire transit live, and sitting in front of the monitor of your computer would be the "safest" way to observe the transit! However, watching the Venus transit directly requires sufficient safety measures so as not to damage our eyes - temporarily or permanently. Smoked glass or sunglasses are not at all safe. It is not at all safe to look at the Sun without safe filters. It is only during a total solar eclipse that too only during the totality phase that it is safe to look at the Sun directly.

In this article, we shall review the important factors leading to injury to the eyes by naked viewing of the Sun and the means for their prevention. We shall need to have a look at the structure of the eyes and the light radiations affecting it. We shall then consider the injuries caused to the eye especially by viewing at the Sun either with naked eye or through unsafe devices like smoked glass, sun glasses etc., methods of viewing the Sun safely; and the measures to prevent injury to the eye.

Structure of the Eye

The eye does not actually see objects. Instead, it sees the light they reflect or give off. The eye can see in bright light and in dim light, but it cannot see in no light at all. Light rays enter the eye through transparent tissues. The eye changes the rays into electrical signals. The signals are then sent to the brain, which interprets them as visual images.

The visible parts of the eyeball are the white sclera and the coloured iris, shown in Figure 1. A membrane called

the conjunctiva covers the sclera. The sclera is the white part of the eye. The clear cornea lies in front of the iris. The lens is connected to the ciliary body. Inside the eyeball is a clear substance called vitreous humour. The retina, which underlies the choroid, changes light rays into electrical signals. The optic nerve carries the signals to the brain. The fovea centralis, a pit in the macula lutea (explained later), is the area of sharpest vision.

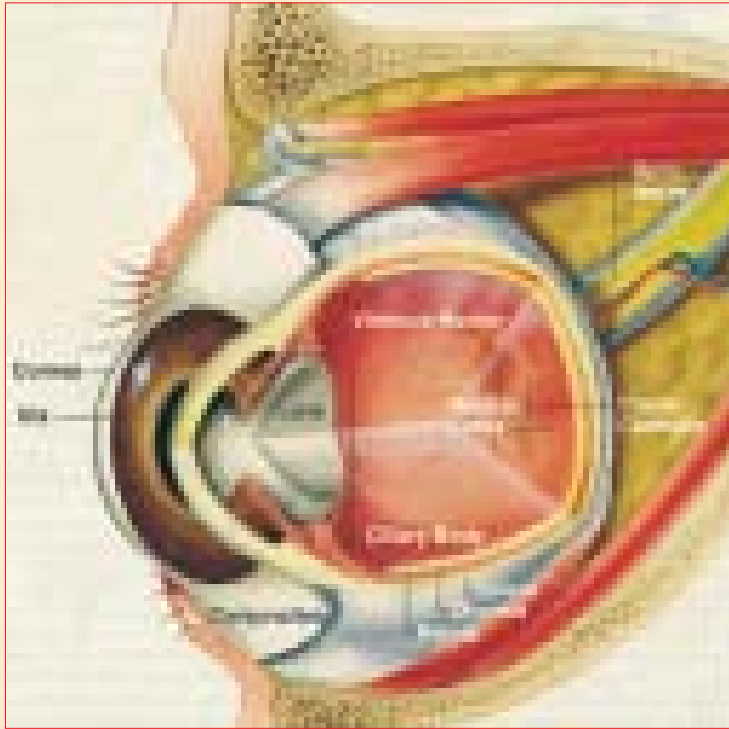


Figure 1: Structure of the eye

The iris is the coloured disk that lies behind the cornea. At the center of the iris is a round opening called the pupil, which looks like a black circle, shown in Figure 2. The pupil regulates the amount of light that enters the eye. Two muscles in the iris automatically adjust the size of the pupil to the level of light. In dim light, the dilator muscle enlarges the pupil. As much light as possible can then enter the eye. In bright light, the sphincter muscle makes the pupil smaller, which prevents too much light from entering the eye. The pupil also becomes smaller when the eye looks at a nearby object, thus bringing the image into sharp focus.

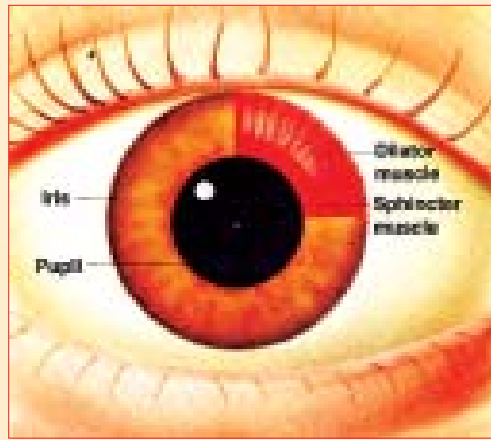
The ciliary body encircles the iris. It is connected by strong fibres to the crystalline lens, which lies directly behind the iris. The lens is a flexible structure about the size and the shape of an aspirin tablet. Like the cornea, the lens is transparent because it has no blood vessels and is relatively dehydrated. The muscles of the ciliary body make constant adjustments in the shape of the lens. These adjustments produce a sharp visual image at all times as the eye shifts foci between nearby and distant objects. The ciliary body also produces a clear watery fluid called aqueous humour. This fluid nourishes and lubricates the cornea and the lens, and it fills the area between them. The ciliary body produces aqueous humour continuously. The old fluid is drained out as the new fluid takes its place.

The choroid forms the back of the uveal tract (the iris, the ciliary body and the choroid considered as one structure). It looks and feels like a blotting paper soaked with black ink. The choroid has many blood vessels. Blood from the choroid nourishes the outer part of the retina.

The retina makes up the innermost layer of the wall of the eyeball. It is about as fragile as a piece of wet tissue paper. The retina is made up of two types of light-sensitive cells - rods and cones. The cells are named for their shape. The retina has about 120 million rods and about 6 million cones, which absorb light rays and change them into electrical signals (Figure 3).

Near the center of the retina is a round area called the macula lutea or macula. The macula consists chiefly of cones. **It produces a sharp image of scenes at which the eyes are directly aimed, especially in bright light.** The rest of the retina provides peripheral vision - that is, it enables the eyes to see objects to the side while looking straight ahead. Most of the rods lie in this part of the retina. Because rods are more sensitive in the dark than cones, faint objects often can be seen more clearly if the eyes are not aimed directly at them. For example, looking to the side of a dim star makes its image fall on the part of the retina that has the most rods and provides the best vision in dim light.

Nerve fibres attached to the rods and cones join at the center of the retina and form the optic nerve. This nerve consists of about a million fibres. It serves as a flexible cable that connects the eyeball to the brain. In fact, the optic nerve and the retina are actually extensions of the



The iris has a round opening called the pupil, which regulates the amount of light that enters the eye. In dim light, the dilator muscle pulls the pupil open wider. In bright light, the sphincter muscle tightens around the pupil and makes it smaller.

Figure 2: The Iris

brain. The optic nerve carries the electrical signals produced in the retina to the brain, which interprets them as visual images.

How we see

Light rays that enter the eye must come to a point on the retina for a clear visual image to form. However, the light-rays that objects reflect or give off do not naturally move toward one another. Instead, they either spread out or travel almost parallel. The focusing parts of the eye - the cornea and the lens - bend the rays toward one another. The cornea provides most of the refracting (bending) power of the eye. After light rays pass through the cornea, they travel through the

aqueous humor and the pupil to the lens. The lens bends the rays even closer together before they go through the vitreous humour and strike the retina. **Light rays from objects at which the eyes are aimed come together at the fovea centralis, a tiny pit in the center of the macula** (Figure 3). This is the area of sharpest vision. Light rays from objects to the sides strike other areas of the retina.

Adaptation to light and dark is partly controlled by the pupil. In strong light, the pupil may become as small as a pinhead and so prevent the eye from being damaged or dazzled by too much light. In the dark, it can get almost as large as the entire iris, thus letting in as much light as possible. However, the most important part of adaptation to light and dark occurs in the retina.



The retina has cells called rods and cones, which absorb light rays and change them into electrical signals. There are more cones than rods in the central area of the retina. The cones are concentrated in the fovea centralis. Nerve fibres attached to the rods and cones join to form the optic nerve.

Figure 3: Retina

Optically important regions in the electromagnetic spectrum

Different sources of radiation emit electromagnetic energy in different parts of the electromagnetic spectrum, say, radio frequency, infra-red, visible, ultra-violet, X-rays or gamma-rays. The wavelength of light is measured in the units of nanometres (nm), where 1nm = 10⁻⁹ metre. The electromagnetic spectrum is shown schematically in Figure 4.

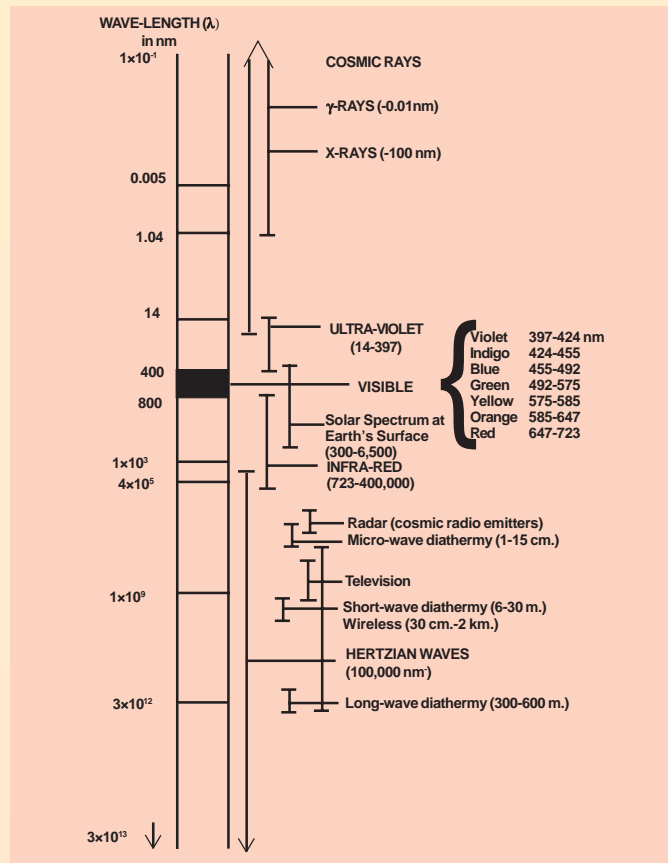


Figure 4 : The Electromagnetic Spectrum

The solar spectrum at Earth chiefly comprises of infra-red rays (say from 6500 nm - 723 nm), visible light (from 723 nm - 399 nm), and ultra-violet rays (from 397 nm - 100 nm). **It would be interesting to note that the sunlight contains about 58% infra-red radiation, 40% visible and 2% ultraviolet.**

The optically important regions are middle (i.e., from 3000 nm - 393 nm) which includes infrared, visible and long ultra-violet rays and the shortest wavelength in the electromagnetic spectrum (i.e., less than 1 nm). These are schematically shown in the Figure 5.

Light transmitted through different parts of the eye

The ocular tissues, like the other tissues of the body, are transparent to the longest waves in the electromagnetic

spectrum: like other tissues they are opaque to the longer infra-red radiations which are readily absorbed by water and are thus absorbed by the outer layers of the cornea. Below

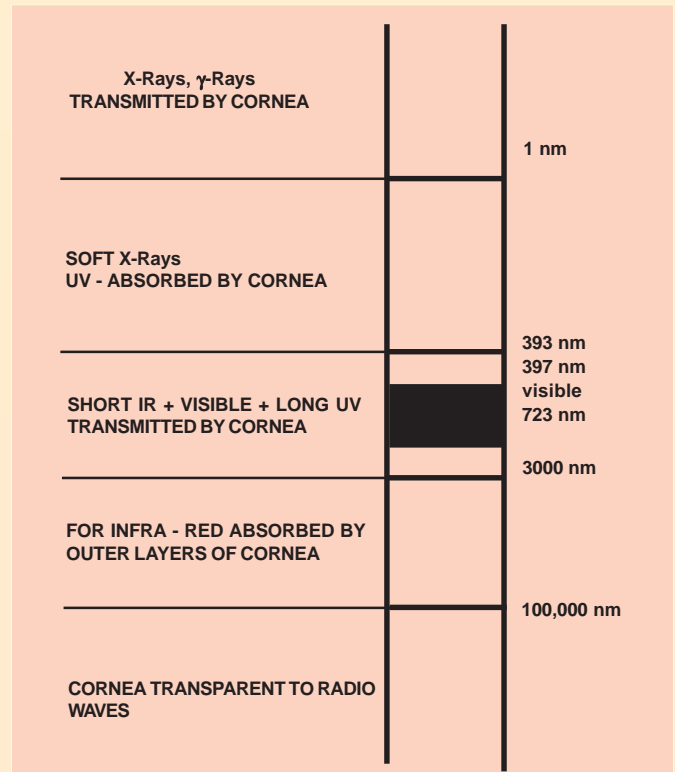


Figure 5 : Optically Important Regions of the Electromagnetic Spectrum

3,000 nm, however, in the shorter infra-red, transparency again begins and throughout the middle regions of the spectrum the limits of absorption and transmissibility vary considerably until, in the long ultra-violet, all radiations below 393 nm are again cut off by the cornea. Another band of opacity exists throughout the short and extreme ultra-violet, a region wherein much of the radiation is absorbed by water and some by air; but at the level of 1.0 nm, through the bands of soft and hard x-rays and y-rays. The two regions of the spectrum, i.e., the middle and the shortest, are of biological significance.

Different parts of the eye, i.e., aqueous (i.e. through aqueous humour between cornea and iris), lens, vitreous and retina transmit light of different wavelength in different proportions. This is shown in Figure 6.

Below 3,000 nm an increasing proportion of infra-red radiation is transmitted through the cornea; there are bands of relatively high absorption in the neighbourhood of 2,000 and 1,400 nm to which this tissue is relatively opaque, but 25% of the incident radiation is transmitted through it at 2,300 nm, 65% at 1,650 nm, 80% at 1,200 nm, and almost 100% at 1,000 nm. To this wavelength in the short infra-red, the cornea is more transparent than to visible red rays (750

nm). Most of the visible radiation, however, is transmitted until absorption becomes apparent and increases steeply

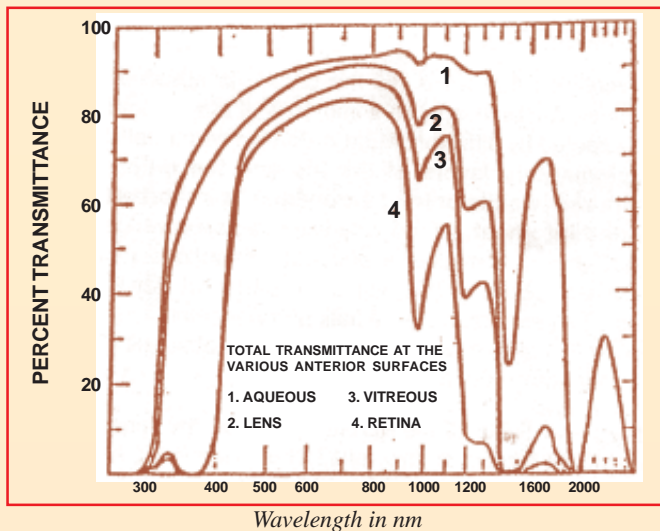


Figure 6: How direct and scattered light is transmitted through the entire eye.

in the long ultra-violet: at 370 nm 90%, at 330 nm 80%, at 305 nm 50%, at 300 nm 25%, and at 290 nm only 2% of the radiation incident on the cornea is transmitted into the inner eye to be largely absorbed by the aqueous humour. Of the remaining 98% of this spectral band, half is absorbed by the corneal epithelium and half by the stroma; and at 230 nm practically all (97.3%) of the incident energy is cut off by the epithelium.

The radiation that traverses the cornea is absorbed in part by the ocular tissues while some 10% of the incident energy is dissipated by diffusion. **Most radiation which falls upon the pigmentary layers of the iris and the retina, whether infra-red, visible or long ultra-violet, is absorbed and converted into heat.** Of the long-wave radiation which traverses the transparent media, the aqueous humour absorbs all the infra-red above 2700 nm and partly at lower wavelengths. The lens absorbs all radiations longer than 2300 nm, but, below it, it shows two bands of selective absorption near 1900 nm and 1500 nm.

At the lower limits of the visible spectrum, the most actively absorbent tissue is the lens. The lower limit of transmissibility varies considerable proportion of ultra-violet rays down to 305.5 nm may pass through this tissue, in the adult the effective zone of partial absorption is from 400-350 nm, although a feeble transmissibility down to 320 nm may exist, and in the aged all rays below 450 nm in the violet may be absorbed.

The concentration of radiant energy in the eye

The concentration of radiant energy within the eye is obviously of considerable importance in the study of its

effects upon the ocular tissues. The longest (radiowaves) and shortest (X- and Y-rays) radiations traverse the ocular

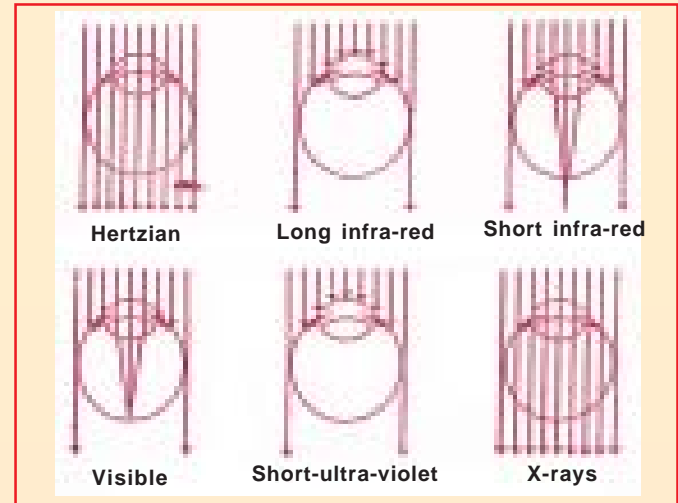


Figure 7: The permeability of the eye to the electromagnetic spectrum

tissues without deflection, but radiations belonging to the infra-red, visible and ultra-violet regions of the spectrum are retarded in their passage through the media and thus suffer refraction to a degree depending on the optical density of the tissue and the wavelength in question; and shorter violet waves are the most refrangible, while refraction becomes progressively less towards the red end of the visible spectrum and the infra-red. This is schematically shown in Figure 7.

With a small source emitting radiations of middle spectral distribution, the density of energy is approximately uniform through the anterior half of the globe, since absorption and dispersion in the media counterbalance fairly exactly the concentrating effect of refraction; but in the posterior part of the globe where the beam is brought to a focus, the latter effect becomes the more prominent. It follows that with a small source (a point source or with small angular diameter) of energy damage may be caused at the retina while the anterior structures are left unaffected. This occurs, for example, in sun-blindness owing to the refraction of the infra-red and visible rays as shown in Figure (8). The effect of concentration, however, is limited by the imperfections of the optical system of the eye.

Hazards from UV Radiation

UV radiation refers to the part of the electromagnetic spectrum subdivided into ultraviolet (UV) rays with wavelengths of $\lambda = 100-380$ nm. UV radiation is further subdivided into UV-C ($\lambda = 100-280$ nm), UV-B ($\lambda = 280-315$ nm), and UV-A ($\lambda = 315-380$ nm).

In general, the shorter the wavelength the more energetic the radiation making it damaging the plants and animals. UV-C can do great damage but fortunately poses

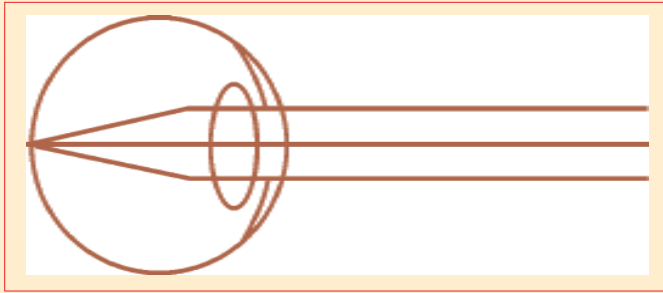


Figure 8: The concentration of radiant energy in the eye

no risk to life on Earth. It is completely filtered out by oxygen and ozone in the stratosphere. Ozone also plays a vital role in filtering UV-B, the ultra violet radiation i.e. the greatest threat to life on Earth. But even a fully functioning ozone layer does not absorb all the UV-B rays. UV-A surpasses the stratosphere virtually unfiltered. But compared to the shorter wavelengths, UV-A causes little harm and even plays an important role synthesizing Vitamin D in humans. So, in discussing the harmful effect of ultra violet radiation we are mainly talking about UV-B. **Too much exposure to this type of radiation can lead to skin cancer, cataract and suppressed immune system.** UV radiation accounts for 20% of cataract cases, and 90% of all skin cancer. Cumulative exposure over a person's life exacerbates wrinkling and discolours the skin. The penetration of UV radiation in the eye is shown in Figure 9.

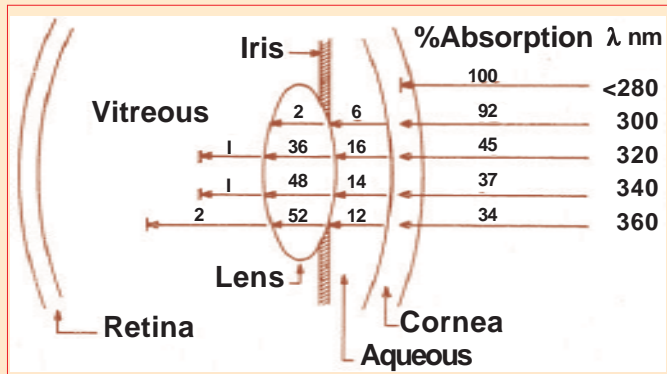


Figure 9 : Depth of penetration of UV into the eye

Radiation can have the greatest effect on the parts of the eye that absorb them. How deep the rays can penetrate into the eye depends on their wavelength. Yet the dividing lines are not quite so distinct. A more accurate distinction is made by considering how easily the different wavelengths pass through the components of the eye. The eye's translucence also depends on a person's age. In early life, the frontal part of the eye is more translucent than it is in old age.

Damage can also occur in places where the eyes are subjected to high intensity UV radiation, say during electric arc welding or in snowy zones under clear skies. One long term effect of UV radiation is a certain clouding of lens or formation of cataract. Certain proteins, so-called crystalline,

BOX 1 WHY LOOKING AT THE SUN DIRECTLY IS DANGEROUS?

The size of the image of the Sun at the retina is of the order of 0.2 mm. Hence the energy available at retina is concentrated approximately in an area of circle within radius 0.1 mm.

$$\begin{aligned}
 \text{Solar Energy incidence on Earth} &= 1.36 \text{ Kw/m}^2 \\
 &= 1.36 \times 10^{-4} \text{ Kw/cm}^2 \\
 \text{Area of pupil (radius 1mm)} &= 0.03 \text{ cm}^2 \\
 \text{Thus power incident on the pupil} &= 1.36 \times 10^{-4} \times 0.03 \text{ kw} \\
 &= 0.040 \times 10^{-4} \text{ kw} \\
 &= 4 \times 10^{-6} \text{ kw} \\
 \text{70\% of this energy is available at retina, i.e., power incident at retina} &= 4 \times 0.70 \times 10^{-6} \text{ kw/cm}^2 \\
 &= 3 \times 10^{-6} \text{ kw}
 \end{aligned}$$

The size of the image being of diameter 0.2 mm, the energy absorbed in the retina is concentrated in area = $0.03 \text{ mm}^2 = 0.03 \times 10^{-2} \text{ cm}^2 = 3 \times 10^{-4} \text{ cm}^2$.

Hence the concentration of solar power in area in which image is formed = $3 \times 10^{-6} / 3 \times 10^{-4} = 10^{-2} \text{ kw/cm}^2 = 100 \text{ kw/m}^2$

Which is about 100 times more than the solar energy incident on Earth and quite powerful to cause retinal burns even if viewed for only a few seconds!

are altered in the lens cells by photochemical reaction in connection with other factors, such as diabetes. This can cause a pigmentation of the cells and clouding of the lens. This process progresses over time until vision is sharply impaired or even blindness occurs. Since the lens tissue in contrast to other tissues in the body - does not grow the new cells, the damage is irreversible. Yet, modern surgical technology does allow the clouding the lenses to be replaced by artificial lenses. The radiation responsible for this condition is UV-A and UV-B which cause the damage. The intensity of the radiation that causes this disease is well below the intensity that can cause an acute inflammation of the cornea or conjunctiva. **What is most important is the cumulative length of exposure, mostly extending over several decades.** Indeed, the disease can also be caused by artificial UV radiation sources. Plain sunlight can also cause the cataract but this often effect people who work a lot outdoors, such as farmers or seamen. Disease can effect practically anyone. The common form occur in people nearing the end of their seventies. The rate of cataract disease in the population increases with the age.

Retinal Burns

Chorioretinal (i.e., involving choroid and retina) burns are most usually produced after looking at the Sun;

occasionally they occur after accidental exposure to lightening, or the short-circuit of a high-tension current or, more rarely, after gazing into a strong artificial source of light such as a carbon arc. Sometimes, following subliminal exposures, only temporary subjective symptoms appear. Following severe exposures, a destructive burn causes permanent damage - a serious matter when, as is usually the case, the macula is involved. A solar chorioretinal burn (sun blindness or photoretinitis) is an injury of this type.

Let us consider the Sun as black body at temperature 60000 K. The energy available in its radiation will be about 1.36 kilowatt/m² at the Earth's surface. If the pupil is strongly contracted to about 2 mm, as is the case when the Sun is observed directly, about 3% of this energy will enter the eye. Slightly over 30% of the energy that enters is lost in its passage through different inner parts of the eye. We make use of this information to estimate the concentration of energy on the retina while observing objects like the Sun (Box1).

Symptoms - In case of an accident

The subjective symptoms (after looking at the Sun without adequate protection) are characteristic; and their severity bears little relation to the retinal appearances. **In most cases nothing abnormal is noticed immediately except the dazzling sensation; but shortly thereafter a diffuse cloud floats with irregular undulations before the eyes, associated usually with irritating after-images, photophobia (fear of light), and occasionally photopsia (flashes of light) and chromatopsia (disturbance in colour vision). After 24 hours, this diffuse cloud contracts into a dense scotoma (a blind spot or area of depressed vision) which may last for weeks or months or even permanently.** The scotoma is typically central and reduces the visual acuity to an average of 6/12 (what a person with normal eyesight may see from 12 feet would be seen by the affected persons from 6 feet) but not infrequently to 6/60 (what a person with normal eyesight may see from 60 feet would be seen by the affected person from 6 feet) or less; it is discovered by the blurring or disappearance of small objects or test letters and in the early weeks, it often undergoes flickering or rotatory movements. Metamorphosia (larger or smaller images of objects rather than their normal sizes) may appear in the central field due initially to displacement of the retinal elements with oedema and eventually to degenerative changes. **Do not take chances! Rush to an ophthalmologist in case of any symptoms mentioned above! To avoid this situation, follow the guidelines for viewing the Sun safely given later in this article.**

The objective signs are typical, but even when the subjective symptoms are marked, the fundus may occasionally appear normal. Initially in the slighter cases, the macula seems somewhat darker than usual, a change doubtless due to choroidal congestion; in the more severe cases the central area may be raised and oedematous,

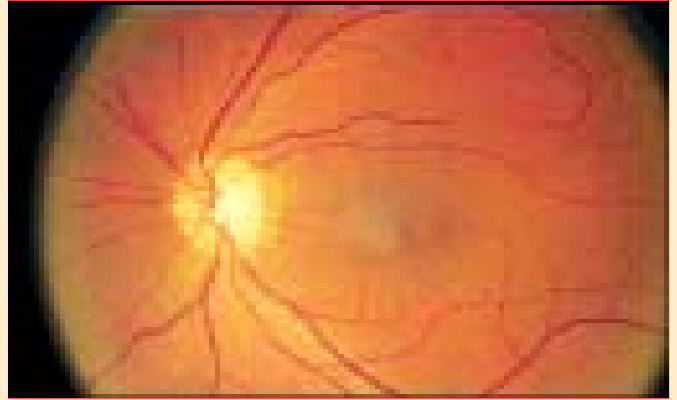


Figure 10 : This photograph is the back of the eye of a man who viewed the partial solar eclipse of 1966 without eye protection. The arc-shaped scars are typical of an eclipse burn, and the vision in this eye has been reduced to 20/30 (6/9). **Source :** BBC News <http://news.bbc.co.uk/1/hi/sci/tech/1376184.stm>

showing perhaps a grey appearance and minute haemorrhages or a dark central spot surrounded by an oedematous retinal detachment. The typical appearance which rapidly develops at the fovea is that of one or more yellowish-white spots, oval in shape or sometimes crescentic, surrounded by an irregular zone of mottled pigmentation fading gradually into the background of the fundus (Figure 10 and 11). This appearance corresponds to the lesions seen experimentally: careful ophthalmoscopic focusing suggests that the central spot is a burnt-out hole in the pigmentary epithelium, while the surrounding stippled ring represents an aggregation of pigment. **In the worst cases a typical macular hole may develop.**

Vision after retinal burns

In the majority of cases the vision improves within the first month or two and the scotoma, if it persists, tends to

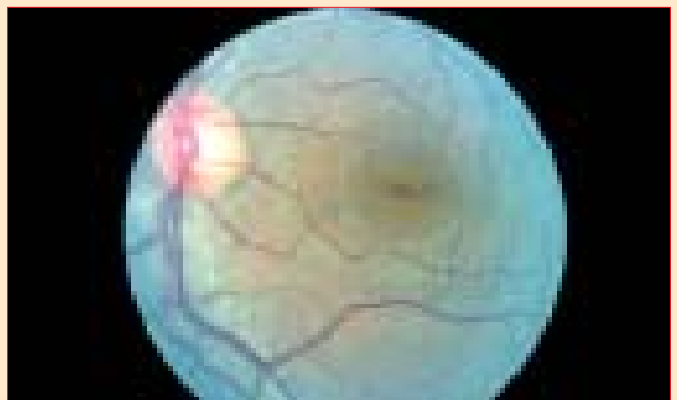


Figure11: This picture shows a more extreme form of solar retinopathy in the left eye of a young man who stared unprotected at a partial eclipse of the Sun. Several crescent-shaped burns can be seen in the central retina, and these have resulted in blindness in this eye, with his vision reduced to below 20/400 (6/120). **Source :** BBC News <http://news.bbc.co.uk/1/hi/sci/tech/1376184.stm>

BOX 2 SOLAR BLINDNESS : A CASE STUDY

Looking at the Sun directly without safe filters can be quite dangerous. Even during a solar eclipse, it is equally dangerous to observe the Sun directly. It is safe to look at the Sun directly only during the total phase of a total solar eclipse. We give below a case study of eclipse blindness following a partial eclipse that was visible in the island of Hawaii in 1962.

On February 4, 1962, a partial eclipse of the Sun was visible from the islands of Hawaii. It was a cloud free day - a Sunday afternoon- and the news media had directed attention at the impending eclipse. These factors combined to produce an incidence of solar retinitis of epidemic proportions. This day was almost cloudless with unlimited visibility, though the eclipse was incomplete, sufficient surface of Sun was obscured to effect a decrease in the amount of measurable incident radiant energy. The study revealed that, time of the year, height of Sun, degree of eclipse and amount of cloud cover, combined with leisure time available to watch the eclipse were the factors primarily associated with the cases reported of solar retinitis.

A study involving Hawaii military personnel and their dependents stated that the day following the eclipse, the first patient with solar retinitis presented at United States Army Tripler General Hospital. Shortly thereafter, the outlying dispensaries began to report a progressively increasing number of patients who had complaints of sudden onset of unilateral and bilateral visual disturbance. Patients varied in the description of their complaints, but most patients noted an initial blur of vision which resolved by the following morning into a central scotoma. In unilateral cases, the presence of the central scotoma was not immediately appreciated. New patients reported months after the eclipse. Three of the four personnel who registered no initial complaint had their lesions detected when they could not fire accurately on the rifle range.

Characteristic of solar retinitis epidemics is the simultaneous onset of ocular injury in all patients. Stage 1 of retinal burns could be described as a central yellow spot that occupied the foveal area of the involved eye. Stage 2 shows a redness of the macula. Its centre contains the yellow point of stage 1 with greatest intensity of redness concentric to this point. Gradual fading of the redness occurs and after about 3-4 weeks, a central pigmentation is observed as beginning of the third stage. Stage 3 may involve a macular hole or a pigmented area located in the deepest retinal layers, which may impair the vision permanently.

Patients examined immediately following the eclipse had diminished visual acuity of varied degree. These patients had not used any mode of protection; or the type of protection used included use of fingers as a pin-hole, photographic films of uncertain density or sun glass, smoked glass or cameral viewfinders. In a total of 52 eyes examined, 27 regained the normal visual acuity while the remainder had visual losses with varying degree and the chance of recovery was approximately 50 per cent. In case of persons with prior existing muscle imbalance in one eye or with ambliopia (i.e., with very poor vision in one eye), solar retinitic lesion was generated in the dominant eye.

become relative sufficiently small; correspondingly the red zone with its yellow spots at the macula may become greyer and also disappear. Few records are available of patients who have been injured for more than a year and fewer and fewer after several years. Improvement may occur over several months but in the majority of cases some disability persists.

The return of the visual acuity to 6/6 does not necessarily mean recovery of normal vision because in some cases small residual central or paracentral scotomata may persist and particularly if these are bilateral, they may lead to permanent impairment of reading or the ability to perform fine work. In most cases with a permanent macular lesion, particularly a hole, a small area of central vision may be permanently lost. After a few years, however, the visual capacity may increase considerably and the resultant scotoma becomes small. Indeed the vision may appear to be unaffected and the minimal disability is unnoticed by the patient - a happy result which unfortunately is by no means invariable. The moral is - Never look at the Sun directly, even during partial solar eclipse. To illustrate the point, a case study of eclipse blindness following a partial eclipse that was visible in the island of Hawaii in 1962 is given in Box 2.

Viewing the Sun

Never view the Sun directly, eclipse or no eclipse, without safe, tested filters, otherwise temporary or permanent damage could be caused to your eyes. The Sun can be viewed safely with the naked eye only during the few brief seconds or minutes of a total solar eclipse. It is emphasised that even when 99% of the sun's surface is obscured during the partial phases, the remaining photospheric crescent is intensely bright and cannot be viewed safely without sufficient eye protection. Hence, **Do not attempt to observe the Sun directly, even during the partial (or annular) phases of any eclipse with naked eye. Unless appropriate filters are used, it may result in permanent eye damage or even blindness!** It is, therefore, necessary to follow certain guidelines for safe viewing of the (partial or annular) solar eclipse. **The fact that the Sun appears dark in a filter such as smoked glass, sun glasses, coloured film, photographic neutral density filters etc. does not guarantee that your eyes would be safe. Damage to the eyes comes predominantly from invisible infra-red wavelengths. Avoid all unnecessary risks.**

Observing a transit is like observing the Sun on any day. It is necessary to reduce the intensity of Sunlight at least by a factor of 100,000 or more for safe viewing. Any filter which reduces the intensity of a standard 60 Watt incandescent frosted electric bulb such that the printed code is no longer readable would be safe enough. To prepare an effective filter, put together two or more thicknesses of over-

BOX 3 DOs AND DON'Ts FOR OBSERVING THE VENUS TRANSIT

DOs

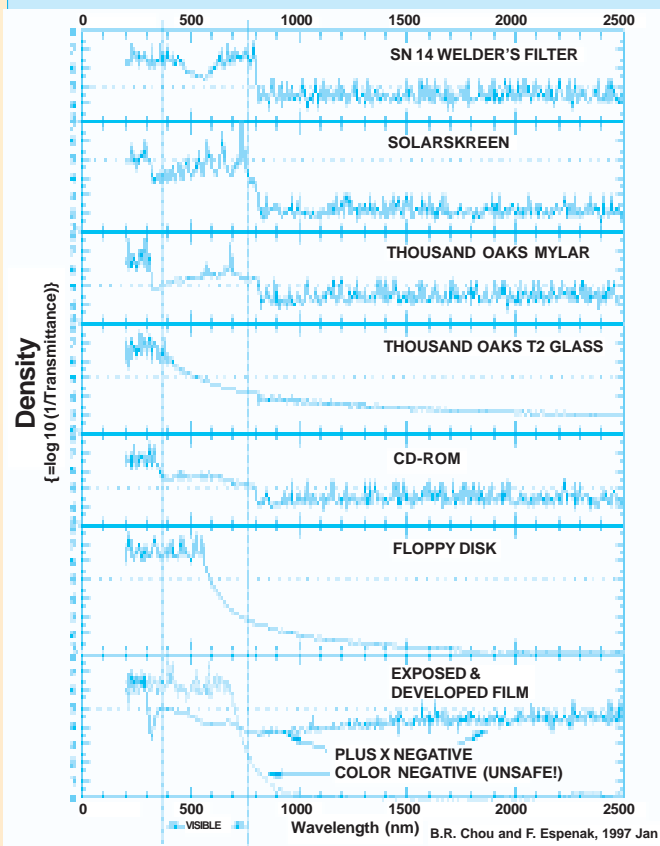
- ☞ Project the image of the Sun on a shaded wall through a pin hole.
- ☞ A small telescope or binoculars can be used to project the image of the Sun on a white card / screen / wall. If binoculars or telescope has any plastic parts, take necessary precautions to protect them from heating and melting by sunlight.
- ☞ Direct viewing of the partially eclipsed Sun should be done only using a scientifically tested filter certified to be safe. A dark welder's glass (No.14) is ideal. The filter provided in the Vigyan Prasar kit can also be used. Always, use only one of your eyes to view the eclipse. In all cases, please examine the filter before use. A filter with pin holes / scratches must not be used. Don't touch, fold or wipe the film with your fingers, under any circumstances. Any scratch or fold on the film would render it unsafe for viewing the eclipsed Sun.
- ☞ Look at the Sun only intermittently.

DON'Ts

- ☞ Don't attempt to observe the Sun with naked eyes.
- ☞ Never look at the Sun through a telescope or binocular without a proper filter.
- ☞ Don't use any filter that simply reduces the visible intensity of the Sun. Fifty-two per cent of the Sun's rays are in the infra-red region of the spectrum. Damage to the eye is predominantly caused by this invisible infrared energy.
- ☞ Don't use smoked glass, colour film, sunglasses, non silvered black & white film, photographic neutral density filters and polarizing filters. They are not safe.
- ☞ Don't use solar filters designed to thread into eye pieces and often sold with inexpensive telescopes.
- ☞ Don't look at a reflection of the Sun from coloured water.

exposed black and white photographic or X-ray film (slower films are best) until a density is obtained which just abolishes the readability of print on a 60-watt bulb (Ref. *Archives of Ophthalmology*, 70 (1964) 138). The light of the bulb in a dark room will then appear as a glow similar to that of full moon on a moderately bright night. The metallic silver contained in the film emulsion is the protective filter. However, black and white film even if fully exposed to light is likely to be unsafe, since they often use dyes instead of silver. A popular inexpensive alternative is aluminized mylar sheet (without any microholes) manufactured specifically for solar observation. Ordinary mylar sheet may not be safe. Dark Welder's glasses (shade No. 14) could also be safely used. **As a further precaution, do not view the Sun even through these filters continuously beyond a few seconds.** Figure 12 gives spectral response for a selection of safe solar filters as given in the NASA Reference

SPECTRAL RESPONSE OF SOME COMMONLY AVAILABLE SOLAR FILTERS



(Reprinted from *Total Solar Eclipse of 1999 August 11*, Espenak and Anderson, 1997)

Note: In addition to the term transmittance t (which describes the light transmitted through a filter in per cent), the energy transmission of a filter can also be described by the term density (unitless) where density " d " = $\log_{10} (1/t)$. A density of "0" corresponds to a transmittance of 100%; a density of 1 corresponds to a transmittance of 10%, a density of 2 corresponds to a transmittance of 1%, etc.

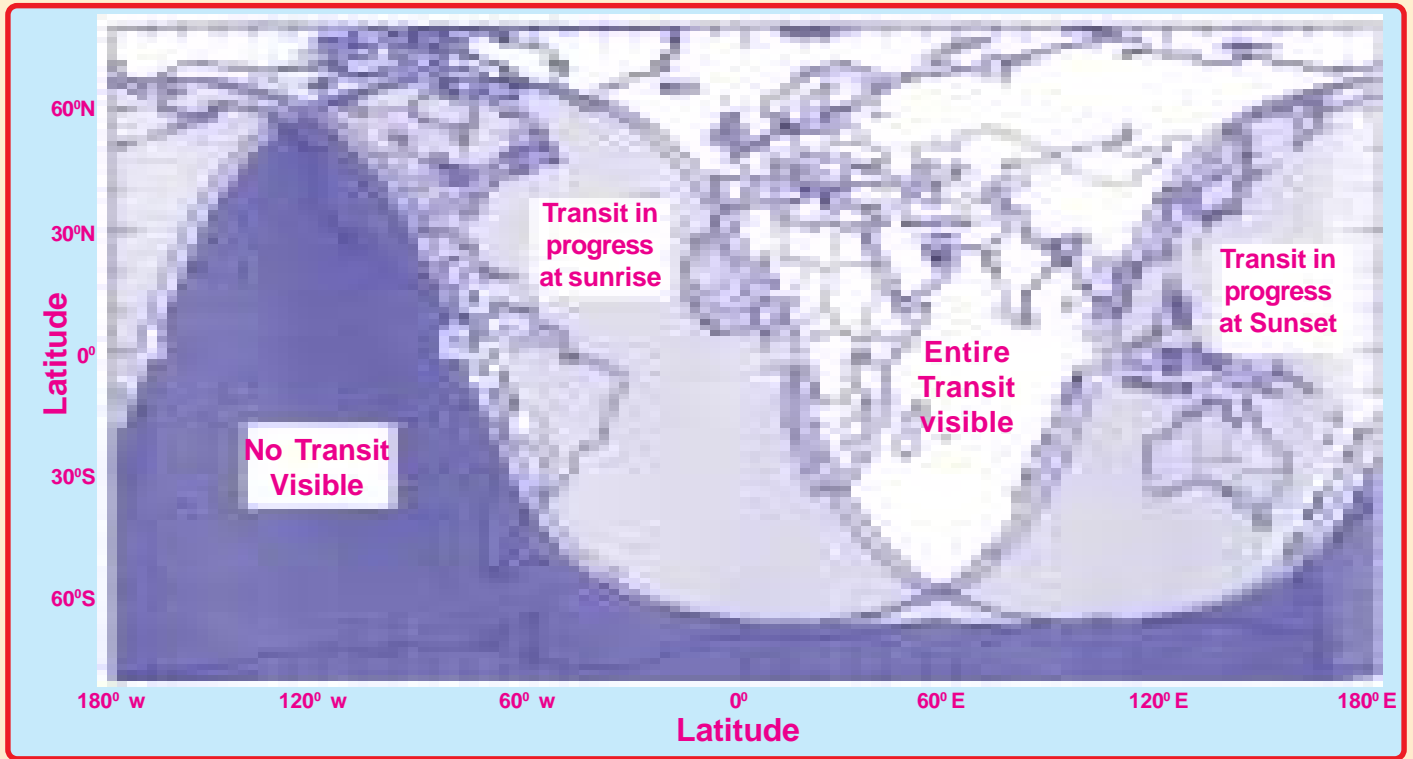
Figure 12

Publication 1398 on Total Solar Eclipse of August 11, 1999. **It is however, emphasised that no legal liability for these recommendations could be accepted since even with best of precautionary warnings, there is every likelihood that accidents will occur with direct viewing of the Sun.** Smoked glass, colour film or sunglasses are not safe. Safest ways are viewing Sun's projected image through a pin-hole on a card-board held at a distance of about 1 metre in a shaded room. DOs and DON'Ts to observe the Sun safely are given in Box 3.

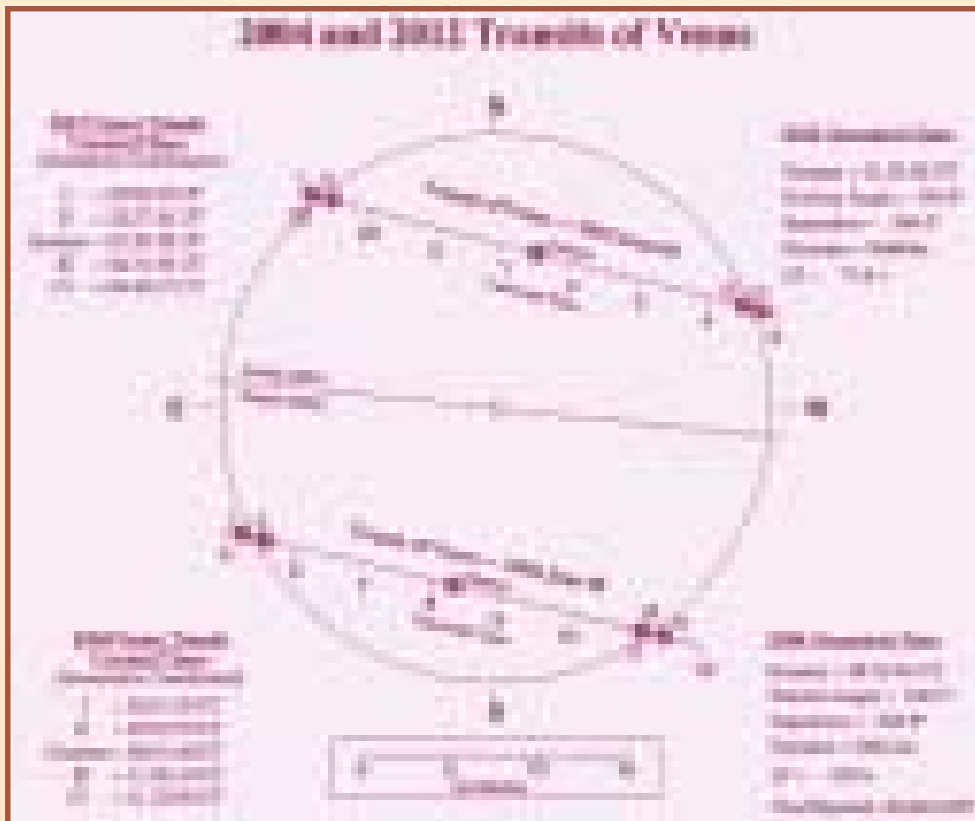
ACKNOWLEDGEMENTS

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Contd. on page...26

08 June 2004 Transit of Venus



World Visibility Map of the Transit of Venus — Fred Espenak, NASA/GSFC



Path of the Venus across the Sun's disk on 08 June 2004 and 06 June 2012 — Fred Espenak, NASA/GSFC



Transit of Venus - 08 June 2004

Circumstances for some important Indian Cities

Venus will pass across the disk of the Sun during a rare transit on 2004 June 08. This uncommon event will be visible from the entire Indian subcontinent. The following table presents detailed predictions for a number of Indian cities. The start and end times are given in Indian Standard Time. We are grateful to Dr. Fred Espenak, NASA/GSFC for providing us with the data on circumstances for important Indian Cities.

Transit Contacts					
Location Name	Contact I	Contact II	Greatest Transit	Contact III	Contact IV
	External Ingress H m s	Internal Ingress H m s		Internal Egress H m s	External Egress H m s
India					
Agartala	10:44:34	11:03:32	13:46:43	16:31:06	16:50:20
Agra	10:46:01	11:05:04	13:48:01	16:31:37	16:50:48
Ahmedabad	10:46:24	11:05:25	13:48:37	16:32:15	16:51:22
Allahabad	10:45:35	11:04:35	13:47:39	16:31:30	16:50:42
Asansol	10:45:00	11:03:58	13:47:08	16:31:20	16:50:32
Bangalore	10:45:31	11:04:24	13:48:14	16:32:44	16:51:46
Bhopal	10:45:57	11:04:56	13:48:07	16:31:56	16:51:04
Bhubaneswar	10:44:58	11:03:54	13:47:16	16:31:38	16:50:48
Chandigarh	10:46:14	11:05:20	13:48:06	16:31:27	16:50:40
Chennai	10:45:15	11:04:07	13:47:56	16:32:33	16:51:36
Coimbatore	10:45:30	11:04:22	13:48:19	16:32:57	16:51:58
Delhi	10:46:09	11:05:13	13:48:05	16:31:34	16:50:46
Dhanbad	10:45:04	11:04:02	13:47:12	16:31:21	16:50:34
Gangtok	10:44:59	11:04:56	13:48:36	16:32:46	16:51:49
Guwahati	10:44:38	11:03:37	13:46:41	16:30:55	16:50:11
Hyderabad	10:45:37	11:04:32	13:48:04	16:32:19	16:51:24
Imphal	10:44:21	11:03:19	13:46:28	16:30:54	16:50:10
Indore	10:46:05	11:05:05	13:48:17	16:32:04	16:51:12
Itanagar	10:44:29	11:03:29	13:46:30	16:30:46	16:50:03
Jabalpur	10:45:41	11:04:40	13:47:51	16:31:47	16:50:56
Jaipur	10:46:13	11:05:16	13:48:15	16:31:46	16:50:57
Kanpur	10:45:46	11:04:48	13:47:48	16:31:31	16:50:43
Kavaratti	10:45:56	11:04:49	13:48:49	16:33:17	16:52:16
Kolkata	10:44:49	11:03:46	13:47:00	16:31:20	16:50:32
Lucknow	10:45:44	11:04:45	13:47:44	16:31:28	16:50:40
Madurai	10:45:20	11:04:11	13:48:13	16:32:58	16:51:58
Mumbai	10:46:15	11:05:13	13:48:39	16:32:33	16:51:38
Nagpur	10:45:42	11:04:40	13:47:58	16:31:59	16:51:07
New Delhi	10:46:09	11:05:12	13:48:06	16:31:34	16:50:46
Patna	10:45:16	11:04:16	13:47:19	16:31:18	16:50:31
Port Blair	10:43:51	11:02:40	13:46:38	16:31:56	16:51:04
Pune	10:46:08	11:05:05	13:48:33	16:32:31	16:51:36
Shillong	10:44:36	11:03:34	13:46:40	16:30:57	16:50:13
Srinagar	10:46:32	11:05:40	13:48:17	16:31:22	16:50:37
Surat	10:46:19	11:05:19	13:48:38	16:32:23	16:51:29
Thiruvananthapuram	10:46:24	11:04:14	13:48:22	16:33:10	16:52:10
Varanasi	10:45:28	11:04:28	13:47:32	16:31:27	16:50:39
Vishakhapatnam	10:45:08	11:04:02	13:47:33	16:31:59	16:51:06

The Global Effort To Eradicate Polio

The Crippler Disease

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The year 2004 might be the year of the last case of polio. If so, by 2007, polio could be certified as the second disease ever to be eradicated. If this landmark achievement is to be realized then Afghanistan, Pakistan, Egypt, Ethiopia, Sudan, Somalia, Angola, Niger, Nigeria and specially India (countries where Polio is still endemic) need to continue their relentless crusade against polio. India bears a dubious distinction of harbouring 83 percent of worlds polio burden. Notwithstanding the adverse increase and outbreak of polio cases in 2002, reduction from 1,600 reported cases of polio in 2002 to 230 in 2003 is a proper reassuring step in the direction of the polio free world. We must remember, however, that the achievement of broad mass public health objectives depends to a great extent on human cooperation—no matter how smart or advanced the technology is, therefore the thrust should be in this direction.

This article will focus on the history and development of the polio vaccine and chronicle the drama and struggles behind the largest public health initiative in history — the global eradication of polio. It will also give an insight into the fascinating story of the pursuit to defeat polio against great odds.

What is Polio?

Poliomyelitis meaning inflammation of the grey matter of the spinal cord — is a unique, fearsome potent disease that has demanded, and continues to demand, a distinctive response and a significant financial investment to control its spread manage its physical effects and ensure its final eradication from the globe. The word “poliomyelitis” was formed by putting together two Greek words for the site of the disease - polios, meaning gray, myelos, meaning marrow, and adding the English suffix, itis, meaning inflammation. Polio has been referred by many names including infantile paralysis, Heine-Medin’s Disease, debility of the lower extremities, and spinal paralytic paralysis. In common usage, the term poliomyelitis is abbreviated to polio. Polio is a highly infectious disease caused by a virus, of which humans are the only natural host. Highly contagious, the poliovirus spreads by contact with

contaminated feces or oral secretions. Children are most vulnerable to the virus. The virus enters the body by nose or mouth and travels to the intestines, where it incubates. A few days later, most patients are either asymptomatic or they experience flu-like symptoms, such as headache, nausea, vomiting, and fever. Whether they are symptomatic or not, people at this stage can pass on the disease to others.

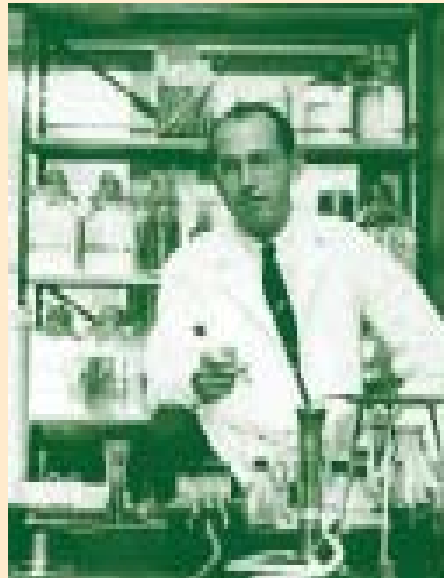
Polio enters the host body through contact with infected feces or through infected droplets traveling through the air, in food, or in water. The virus next enters the bloodstream, and the patient’s immune system develops antibodies against the virus. In most cases, the immune system stops the progression of the virus and a lifelong immunity against the disease is acquired. However 10% of infected people develop symptoms and 1% develops the paralytic form of polio. In serious cases the poliovirus destroys the nerves in the brain and spinal cord, causing paralysis of the muscles in the chest, leg or arms. Once infested the poliovirus invades the nervous system,

specifically damaging the anterior horn of the spinal cord resulting in muscle paralysis, usually affecting voluntary muscles in the arms and/or legs. Five to ten percent of the patients die because of paralysis of breathing and/or swallowing.

Fear of Polio

Paralytic poliomyelitis was perhaps the most feared disease known in the first half of the twentieth century. Polio struck fast, there was no cure, and it crippled its victims for life. Hobbling on crutches, rolling in wheelchairs, or lying immobile in giant iron lungs, the legions of sufferers accumulated from year to year. Even the exact mechanism of polio’s transmission was a hotly debated subject for many years; so many areas were placed under strict quarantine when cases of the disease began to manifest themselves. Only the fear surrounding AIDS can rival the feelings people had about polio in the first half of this century.

Only once in human history have we witnessed the total eradication of a dreaded disease, and that was smallpox more than two decades ago. Now humanity stands



Dr. Jonas Salk, is seen working on polio vaccine in the laboratory at the University of Pittsburgh

on the brink of a second triumph: the global eradication of polio — a scourge that at one time killed or crippled half a million people a year, many of them children. We cannot afford to falter, not when we are so close. As we inch towards that ultimate goal it is time to look back on the developments that have led us to this remarkable human endeavor.

President Roosevelt the crusader of Polio eradication

Franklin D Roosevelt, President of the United States from 1932 to 1945, declared a War on Polio during his presidency tenure. He constructively used the tremendous resources of postwar America to combat the polio menace and aided the scientific community to develop a vaccine that could help prevent polio.

Roosevelt was a polio victim. He wore heavy steel braces on his legs and walking was difficult for him. Most of his time was spent in a wheelchair. Roosevelt was affected with poliomyelitis, or infantile paralysis in 1921, while vacationing at his Canadian summerhouse on Campobello Island. Roosevelt's legs were left permanently paralyzed. In cases such as his, the virus reaches the brain and spinal cord where it multiplies and destroys the nerve tissue. At this point the disease becomes spinal or bulbar (involving the last four or five cranial nerves), depending on which nerves are affected. Both forms are characterized by muscle pain, stiff neck and back, and possible paralysis. The spinal form affects the limbs. The bulbar form affects the lungs and the patients cannot breathe. After a severe attack of polio in its paralytic form, there is no treatment for the disease itself, although symptoms such as muscular paralysis can be helped with physical therapy. How much a person will recover varies from individual to individual.

A few years after he was paralyzed by polio, Roosevelt heard about a young man, also a polio victim, who had showed great improvement after swimming for several summers in a warm-water pool. The pool belonged to an old summer resort, the Meriwether Inn, in the small town of Warm Springs, Georgia. Intrigued, Roosevelt visited the inn. Although the pool held no magical properties, swimming in its warm water helped his weakened legs. Other victims of polio were attracted to the pool and Roosevelt decided to turn the old inn into a centre for the treatment of polio. He founded the Georgia Warm Springs Foundation, with himself as president and his law partner, Basil O'Connor as treasurer. Most of those who came to the centre were



Portrait of Dr. Albert Bruce Sabin

unable to pay for their treatment. So, Roosevelt and a small circle of friends provided the money to keep the centre in operation.

Roosevelt was determined not to let polio illness get the best of him. He not only continued his illustrious political career, resulting in his well-documented and long term Presidency of the United States, but he went on to spearhead the fight against polio, increasing public awareness of the deadly disease and promoting research. Although polio never devastated large numbers of the population like the plague or influenza, it was a frightening, highly contagious disease that attacked both the poor and rich and arose in terrifying outbreaks which seemed impossible to stop in spite of advances in medicine.

March of Dimes

In 1932, Roosevelt was elected President. The fact that the disease had affected a man in the White House seemed to awake the public's interest. The trustees of the Georgia Warm Springs Foundation decided money could be raised for the foundation by holding dances in cities across the nation on the President's birthday, January 30. More money was raised than was needed for Warm Springs, so it was used for scientific research. In January 1938, alarmed by decades of worsening polio epidemics and the terrible toll the virus was taking on America's young, President Roosevelt established the National Foundation for Infantile Paralysis. The Foundation emphasized the nationwide significance and non-partisan character of the polio crusade. Roosevelt believed that people could solve any problem if they worked together. Comedian Eddie Cantor

coined the phrase "March of Dimes" (playing on the popular newsreel feature "The March of Time"), appealing to radio listeners all over the country to send their dimes directly to the White House. The campaign to start with received lukewarm response but within weeks of launching the campaign it became immensely popular and White House was flooded with loads of Dimes thus proving to be hugely successful. The National Foundation officially changed its name to the March of Dimes in 1979. The money collected from this campaign was put to proper use by financing medical research in the leading universities and medical schools to develop a polio vaccine. This research has led, step by step, to the ultimate victory over polio. We are now on the verge of wiping out polio from our planet. The March of Dimes occupies a unique place in American history. Its



Franklin D Roosevelt the President of USA was also the victim of Polio. He began the crusade for Polio eradication

efforts to provide care for the victims of polio while aggressively working to develop vaccines against it, represents the first large-scale, nationwide biomedical initiative, led by a charitable organization. It also helped make the volunteer movement an integral part of the fabric of American life. March of Dimes investment has also been made in other research fields in science, which include support to 11 Nobel laureate scientists whose original work was supported by grants from March of Dimes.



Georgia Warm springs was known for its therapeutic treatment of polio in its warm waters

The early years

The earliest modern clinical descriptions of poliovirus were made in England in 1795, Italy in 1813, and India in 1823, the first documented polio outbreaks occurred in northern Europe, specifically in Norway in 1868 and Sweden in the 1880s. Early Studies suggested that the disease might be contagious, with an initial, infectious pre-paralytic phase with general 'flu like symptoms.

During the 1905 polio epidemic in Sweden, Ivar Wickman was the first to clearly show the infectious nature of polio. This was soon followed by the isolation of the poliovirus in laboratory monkeys in 1908 by Karl Landsteiner in Vienna. The nature of the disease spread by the poliovirus was termed as infantile paralysis. Landsteiner and his assistant E. Popper experimented by injecting suspensions from the spinal cord of a diseased 9-year-old boy into rabbits, guinea pigs, mice and monkeys. Only the monkeys showed signs of disease. They also observed that no bacteria were found in the monkeys and their nervous system changes resembled those of rabies. Based on their findings, Landsteiner suggested that the disease has a viral etiology. He then sent fragments of a spinal cord from a 13-year-old child afflicted with poliomyelitis to the Pasteur Institute in Paris. Poliovirus was shown to be a filterable virus that could spread along nerves and be transferred between monkeys.

The discovery of the virus-causing poliomyelitis was immediately accepted. By 1909-10, the main focus of polio research had shifted to the Rockefeller Institute for Medical Research in New York City. The polio research at the institute was lead by Dr. Simon Flexner and his team. 1910 was a landmark year for polio; the Congress of American Physicians and Surgeons devoted more attention to polio that year than to any other subject. In Flexner's lab the poliovirus seemed to only infect the nervous system, but was also present in a small number of non-neural sites,

particularly the upper nasal area after direct inoculation. Polio thus seemed to be a respiratory infection with the virus spread by infected droplets followed by direct nervous system invasion via the nerves in the nose. This nasal-nervous system model dominated how polio was approached until the late 1930s,

During the next course of the research one of the first questions to be answered was whether just one particular virus caused polio or if there was more than one kind of virus. Research on this

question took several years. But it was finally proved that there are just three strains or types of virus that cause the ailment. This gave hope that a vaccine could be produced to prevent polio.

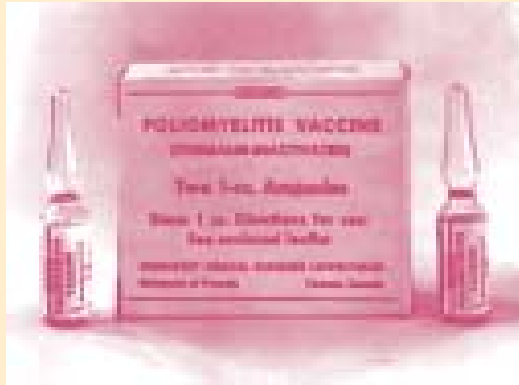
Early efforts in the development Polio Vaccines

The first great hope of developing polio vaccine emerged in 1934-35. Dr. Marice Brodie developed an inactivated polio vaccine and the rival group headed by Dr. John Kolmer who developed an attenuated version of the polio vaccine soon followed it. The success though was short-lived. Their hasty use of vaccines in parts of US proved ineffective and in several cases fatal. This experience left polio researchers hesitant to attempt another polio vaccine for the next 20 years.

An important new era in the history of polio vaccines began when a short paper was published in the journal Science. J.F. Enders, T.H. Weller and F.C. Robbins, of Boston Children's Hospital and Harvard Medical School, published this Nobel Prize winning report. They were awarded the Nobel Prize for Physiology or Medicine in 1954 for their discovery of the ability of poliomyelitis viruses to grow in cultures of various types of tissues. This paper described the means of solving the long-standing problem of culturing the poliovirus in test tubes using non-nervous tissues. This discovery finally provided a method to demonstrate the presence of the poliovirus free from the expensive process of inoculating monkeys, or even mice. This landmark discovery finally opened the door for the development of a practical polio vaccine.

In 1951 a method of providing passive immunity to polio was first tried in North America. During the course of this experimentation it was discovered that the small amounts

of virus that entered the bloodstream could be overcome by a small amount of poliovirus antibodies. Poliovirus antibodies contained in gamma globulin could thus be used to neutralize poliovirus infection over a limited period of time. Further studies showed antibodies against polio are formed in the blood of the victim. That's why a person who has suffered an attack by one strain of virus is immune to that strain thereafter. After more work it became apparent that a practical vaccine for the prevention of polio could be produced.



Inactivated Salk polio vaccine produced by Connaught Medical Research Laboratory, Canada

developed the "Toronto technique" to produce bulk quantities of poliovirus fluids in large bottles. This development paved the way for mass production of Salk vaccines.

Encouraged by Salk's results, in July 1953, the National Foundation for Infantile Paralysis asked Connaught Medical Research Laboratories (now Aventis Pasteur Limited), to provide all the poliovirus fluids required for an unprecedented polio vaccine field trial in the US. Some 3,000 litres of bulk poliovirus fluids produced by Connaught were

shipped to two major pharmaceutical companies, Parke Davis and Eli Lilly in the US to be inactivated and processed into a finished vaccine. Before being released for the field trial, each batch of vaccine had to pass a battery of tests, first by Connaught, then each company, Salk's lab and the US government. Amidst intense publicity, the first children were given the new vaccine on April 26, 1954. The field trial was one of the largest medical experiments in history and involved an elaborate tracking of some 1,800,000 children in the age group of 5-8 years. They were either given the vaccine, or were simply observed to see if they contracted polio or not. The results were dramatic. Cases of polio fell spectacularly in the vaccinated test groups. In 1955, the government quickly granted permission for the vaccine to be distributed to the children of US. On April 12, 1955, the highly anticipated clinical trial results turned into a major media event, perhaps the biggest in medical history. "SALK'S VACCINE WORKS!" screamed the headlines. Dr. Thomas Francis, director of the trial, reported that the vaccine was 60 to 80 per cent effective against paralytic polio. He and Salk stressed that the vaccine was good, but it was not perfect.

The success though was not too long lived. Suddenly, on April 25, 1955, the Salk vaccine euphoria was shattered when the first of a total of 205 cases of polio associated with vaccine made by Cutter Laboratories in California were reported; The problem was traced to incomplete inactivation of some virus particles, which was soon corrected. Since then the vaccine has been highly effective, with a 70 - 90% protection rate. The Salk vaccine is given in two intramuscular injections spaced one month apart and is to be followed by boosters every 5 years.

Developing a Live Oral Polio Vaccine

In 1957, in an effort to improve upon the killed Salk vaccine, Albert Bruce Sabin began testing a live, oral form of vaccine in which the infectious part of the virus was inactivated (attenuated) and not killed. Sabin (1906-93) an American physician and a microbiologist, was born in

The Salk vaccine Story

Dr. Jonas Salk while working at the University of Pittsburgh undertook a major effort to sort out 196 known strains of poliovirus into three immunologically distinct types. After a series of experiments he categorized the poliovirus into three strains namely, Type I (Brunhilde), (161 strains), Type II (Lansing), (20 strains) and Type III (Leon), (15 strains). Jonas Edward Salk, (1914-95) was an American physician and a microbiologist. He did research on the influenza virus at the Univ. of Michigan. In 1946 he became assistant professor of epidemiology at Michigan. By 1951, based on his earlier work of developing an inactivated influenza vaccine, and his experience with the poliovirus typing project, coupled with the work of others studying poliovirus immunity in monkeys, Salk suggested that an inactivated polio vaccine might stimulate active immunity in humans. He developed the polio vaccine by cultivating three strains of the poliovirus separately in monkey tissue. The virus was separated from the tissue, stored for a week, and killed with formaldehyde. He then conducted tests to make sure that the virus was dead. He proved that a series of three or four injections with the killed virus vaccine were required to confer polio immunity. The works of Dr. Andrew J. Rhodes, a leading virologist from England with a special interest in polio, were of special significance to Salk in the development of his vaccine. By 1951, Rhodes' research team was able to grow all three types of poliovirus in a variety of tissues. Salk used the method of growing poliovirus in different tissues in the development of a polio vaccine. This vaccine came to be known as the Salk vaccine. Salk tried his vaccine by first injecting himself and his family including his son to infuse a sense of confidence among the public. He then proceeded to administer the vaccine to residents of an institution for disabled children near Pittsburgh. The encouraging results of the trial were published in March 1953. It was around this time that Dr. Leone Farrell



Russia. He immigrated to the United States in 1921 and was naturalized in 1930. Sabin completed his graduation (B.S) in 1928 and his M.S in 1931 from New York University. He conducted medical research for several organizations before joining the faculty at the University of Cincinnati College of medicine in 1939. He then went on to become a professor of research pediatrics in 1946. He conducted research on viral and other infectious diseases and developed a live oral polio vaccine (OPV) for immunization against poliomyelitis in 1959. Sabin used a trivalent OPV, which contains live attenuated strains of all three serotypes of poliovirus in a 10:1:3 ratios. The viruses were attenuated by serial passage in monkey kidney, Vero, or human diploid fibroblast cell cultures, allowing it to accumulate mutations. Ultimately, this resulted in an attenuated virus that could be given to a patient orally. The weaker virus replicates normally in the intestine, but cannot grow well enough to invade the central nervous system. The vaccine is supplied as a single 0.5 ml dose in a plastic dispenser. The vaccine contains trace amounts of streptomycin and neomycin. The vaccine potency is stabilized with molar magnesium chloride or sucrose. OPV replicates in the gastrointestinal tract and also in lymph nodes that drain the intestine. OPV induces two separate immune responses. First it activates the humoral immune response, prompting the production of serum-neutralizing antibodies in the blood to all three polio serotypes. This systemic response is long lasting and helps to prevent the spread of poliovirus to the nervous system. OPV also produces a mucosal immune response consisting of interferon and virus-specific Immunoglobulin A (IgA) antibody in the epithelial lining of the gastrointestinal tract. Only primates are susceptible to all three serotypes of poliovirus, so the safety of oral poliovirus vaccine (OPV) and its consistency have been tested in the monkey neurovirulence test (MNVT).

Sabin's vaccine could be taken orally and it provided longer immunity than the killed-virus vaccine. The killed-virus vaccine could protect only against paralysis, whereas Sabin's live vaccine could guard against both paralysis and infection. This vaccine became available for use in 1963. The Sabin oral vaccine is given in 3 doses in the first two years of life, and a booster is given when the child starts school. Further boosters are not given unless the patient is exposed to polio or will be traveling to an endemic region. OPV is given orally rather than by injection. Its administration does not require a trained health worker and sterile injection equipment. This vaccine is relatively inexpensive, facilitating mass purchasing of the vaccine

during National Immunization Days. The other advantages of a live, oral vaccine are its long-lasting immunity, the prevention of reinfection of the digestive tract. A single dose of OPV produces immunity to all three-vaccine viruses in about 50% of recipients. Three doses produce immunity to all 3 poliovirus types in more than 95% of recipients. Immunity from oral poliovirus vaccine is probably life long. OPV's ability to stimulate mucosal immunity is responsible for the success of OPV mass campaigns in interrupting wild poliovirus transmission. Therefore, OPV remains the vaccine of choice for the eradication of polio. OPV intestinal immunity reduces the chance that a vaccinated person will become infected with wild virus if he or she is exposed while visiting a polio endemic country. Sabin's oral polio vaccines are now used in India during the National Immunisation Day campaigns.

World Health Organisation and Eradication of Polio

The discovery and use of polio vaccines have eliminated polio from America. In 1960, there were 2,525 cases of paralytic polio in the United States. By 1965, there were 61. Between 1980 and 1990, cases averaged 8 per year, and most of those were induced by vaccination. There has not been a single case of polio caused by the wild virus in US since 1979, with a rare case reported each year from persons coming into the country carrying the virus. In 1994, polio was declared eradicated from all of America. Once the Sabin and Salk vaccines were proven effective, the disease was rapidly eradicated not only in US but also throughout most of the industrialized world. The economic effect has been enormous; it has been calculated that the polio vaccine pays for the costs of its development approximately every three weeks. The benefit to the United States alone for



One of the Popular billboards in support of March of Dimes.

this single breakthrough runs into trillions of dollars. The social impact has been incalculable. The crutches, wheelchairs, and iron lungs of polio victims have at last been banished from children and parents' nightmares, at least in the developed world.

The results shown by the Polio vaccines on the developed nations prompted the World Health Organisation in 1988 to set a goal of eradication of poliomyelitis from the entire world by the year 2000. The results speak for themselves. The number of polio cases worldwide has been reduced dramatically in just over a decade. In 1988, according to WHO, there were an estimated 350,000 cases, of which only 10 per cent were actually reported. By the end of 2001, the number of cases had dropped to 537. Although the number of reported cases increased during

2002, due to polio outbreaks in India and Nigeria, the majority of these cases were concentrated in isolated areas thus giving an optimistic view for a world free of polio.

Polio eradication efforts in India

India officially committed itself to eradicate polio, supporting the WHO resolution to that effect in the very same year 1988. India had more polio cases than any other country in the world. It was estimated that the Indian health care personnel officially reported to the government over 24,000 cases of polio. In reality though, there were probably as many as 10 times that number of cases that went unreported. The sight of children and adults with withered arms and legs throughout the cities, towns and villages of India was routine, and some of the major risk factors for polio virus transmission- crowding, poverty and poor sanitation- were present in India to a degree not seen in most countries.

Progress towards eradication of polio has been steady. The government of India, along with key partners including WHO, UNICEF and Rotary International, has spearheaded a massive effort. The initial attack on polio came with provision of universal immunization in 1985. Under the Universal Immunization Programme (UIP), more Indian children were provided oral polio vaccine (OPV) than ever before in history. The numbers of reported polio cases have dropped from over 24,000 in 1988-89 to less than 5,000 in 1993-94. Although this was encouraging, the government of India soon responded to the need to intensify the polio eradication effort and developed the Pulse Polio Immunization (PPI) strategy. The key innovation was utilisation of mass immunization campaigns to supplement the routine immunization activities. The state of Delhi was the first area to adopt a PPI component in 1994. The first round of National Immunization Day (NID) programme was held in late 1995, which was followed with a second round in early 1996. The NIDs featured booths (fixed sites) to which children under 5 were invited to take two oral polio drops. Over 500,000 booths were set up nationally during the first NID programme, and on a single day a total of 87 million children received oral polio vaccine. The scope and intensity of mobilization utilized for this activity was unprecedented in the annals of the health initiatives in India, and possibly, the world. To understand this remarkable achievement, it is important to comprehend the scale of the efforts made

to meet this gigantic challenge. At the same time as the NIDs were being initiated, it became clear to the government that better information on polio cases was necessary to complete the job of polio eradication. The government of India and the WHO developed a collaborative unit, the National Polio Surveillance Project (NPSP), to provide accurate and rapid surveillance information on polio cases in India. There is now a systematic methodical tracking of cases, finding the source of the infection and flooding infected area with massive doses of polio vaccines. Beginning in 1997, NPSP is now supporting over 200 surveillance medical officers throughout India to coordinate polio surveillance activities. In addition to NPSP network,

a regional laboratory network of 9 highly qualified Indian research centres provides rapid and accurate analysis of samples from patients.

Conclusion

The poliovirus lives in the human intestine, is ejected into the environment through excreta, and spreads by contact with fecal matter. The disease strikes mainly children, cripples the limbs, and is sometimes fatal. Children living in unhygienic conditions without access to clean drinking water are particularly vulnerable to it. This is why a bombardment of the virus through a synchronised

mass immunisation of children in the zero to five age group such as the one carried out nationally on January 4, 2004 is considered the best way to ensure zero incidence. India has done this since 1996 but a cutback in planned immunisation programmes in 2002 was the main factor behind that year's polio resurgence in Uttar Pradesh. It pushed back the goal of a polio-free India and a polio-free world from 2005 to 2007. This year the Government is reported to be considering holding five nationwide immunisation days as against the usual two annually. This decision, despite the costs involved in conducting such an exercise, will be timely and appropriate in reaching the goal. The benefits of total polio eradication, which will eventually include savings on the massive national expenditure in fighting the disease, far outweigh the expenditure. In order to be effective, national immunisation days must cover as many as possible of India's 165 million children under the age of five years. Superstition and fallacies about the perceived side effects of the oral polio vaccine still deter many parents from getting their children vaccinated. Large-scale public awareness campaigns



Salk Polio vaccine was declared effective on April 12, 1955

through mass media, using brand ambassadors, like the ones that involve matinee idols Amitabh Bachhan and Aishwarya Rai, should be continued for the efficacy of the polio eradication efforts. The involvement of voluntary organisations such as Rotary in a door-to-door campaign to build awareness has helped immensely, however educating people about the disease and the vaccine still remains a key challenge for the Government.

Simultaneously, the Government must also ensure that the focus on planned immunisation does not distract attention from routine immunisation through which newborn children get four oral polio vaccine doses from zero to three months. With 15.5 million children born every year in India, routine vaccination is the only way to prevent gaps in immunity from developing. As Tamil Nadu has shown, much also depends on surveillance. The early detection of the two cases reported in Tamil Nadu enabled health officials



Mother Teresa an Apostle of peace is seen administering oral polio vaccine to an Indian child.

immediately to immunise all children in those two areas. The Union Health Minister, Sushma Swaraj, wants zero incidences of polio in India in 2004, so that the country can be declared polio-free after the waiting period of three years. The goal is within reach. It must not be allowed to slip away this time not after so much has already been done.

The Government and other agencies involved in the programme should therefore emphasise on human cooperation and not let their guard down in waging the final assault on one of the world's most debilitating diseases. They should intensify the National Immunisation Programme all through this year, specially targeting states like Uttar Pradesh and Bihar, which have accounted

for most reported cases of polio. They should also address the issues of the emergence of polio in Karnataka, West Bengal, and Andhra Pradesh. As long as the wild poliovirus exists anywhere in India, the risk of it spreading to areas considered free of the disease will remain. Since polio is completely eradicable, even one case of polio is a case too many and therefore we should not miss out on this last opportunity to kill the virus before it can strike back.



The Sixth Wave contd from page... 43

critically endangered, 113 endangered and 87 vulnerable. Amongst animals, 18 are critically endangered, 54 endangered and 143 are vulnerable. India ranks second in terms of the number of threatened mammals and sixth in terms of countries with the most threatened birds. In India, poaching is another insidious threat as one of the primary reasons for the decline in numbers of species, such as the tiger.

True, the underlying causes of biodiversity loss are poverty, economic policies, international trade factors, policy failures, poor environmental laws and / or their weak enforcement, unsustainable development projects and lack of local control over resources. Needless to say that it takes enormous efforts at all levels, from individual to global, to halt species extinction. Data on species, their habitat and threats would need to be periodically collected and analysed to evolve appropriate strategies to prevent their loss. We must act in right earnest now. The time is running out.

No doubt, in earlier extinctions life had always bounced back firmly in all its multiplicity. But, it required millions of years to do so. That may be a blink in geological terms but is considerably longer when viewed against human life span or even human civilization. Most ecologists accept that we are approaching the rates of extinction seen in the past five mass extinctions. The culprit for the current wipe-out is us. We need to quickly develop the capacity to foresee and forestall the sixth extinction wave, or else we shall end by destroying the Earth.

□ **V. B. Kamble**

Eye, Vision contd from page... 35

leading ophthalmologist of New Delhi for her interest, help and for numerous discussions on the topic. She also went through the manuscript and made suggestions for its improvement. Discussions with several doctors and ophthalmologists are also gratefully acknowledged.

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Vacation Health and Safety

Nine Practical Tips for the Summer Holidays



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With the children out of school and colleges enjoying a vacation, it is time to pack your bags and escape from the hustle and bustle of the killing city life into the lap of Mother Nature. A relaxing, fun-filled holiday is a perfect recipe for good health. It casts a magic effect on your physiological and psychological health. And that's one good way to recharge your batteries, cleanse your mind and body of stresses and strains and be at your best once again. Here are some basic mantras:



Just rest and relax : The word "vacation" has been coined from "vacate", which means "to leave", to go away. You must remember this to enjoy your holiday. Go away— not just physically, but mentally as well. A sedentary desk-bound person may wish to go out and stretch his muscles a bit, but a physically active worker may wish to take a quiet vacation. Let the vacation be a complete change from the routine.

Avoid strenuous schedule : The vacation should aim at resting and recreation. Tight, strenuous schedules spoil the vacation, cause fatigue and are of no use. Always plan the itinerary in a way that each person feels comfortable, can relax and does not feel strained. Excessive hours of daily travel lead to fatigue and frayed tempers. An easy schedule ensures a more relaxed and enjoyable holiday.

Give a slip to heat : If you have a choice, travel during the early hours of the day before the sun reaches its zenith. Early evenings also make a good choice. That way you can escape the high temperatures of the day.

Wear easy fitting clothes : Clothing for a trip should be casual and comfortable and should provide for the changes in weather. Wear a T-shirt, shorts and sneakers. Cotton clothing is the best. It is easy on the skin.



Eat at selected places : The places where you plan to stay or go for your meals must be chosen with care. Unhygienic places and substandard food must strictly be avoided. The risks of food poisoning and viral infections are very high during summer months, because high

temperatures promote growth of disease-causing germs. **Avoid foods that are known to be suspect :** It is best to avoid foods which are more likely to be infected.

These include dairy products, cream, potatoes, seafood, egg preparations, chicken and ham spreads, cold sliced meats, and custard.

Carry a first-aid kit : It should consist of a sturdy box with a good catch; a first-aid manual you're familiar with; a pair of blunt-pointed scissors; a roll of one-inch finger bandage; a roll of two-inch roller bandage; a pack of sterile cotton; a pack of sterilised gauze squares that come in sealed envelopes; a roll of adhesive plaster, a small bottle of Savlon, or Dettol; and a clinical thermometer.

Pack in a small medicine kit : It is always a good idea to carry some utility medicines, for instance, paracetamol (Crocic, Calpol, Metacin) or nimisulide tablets for fever, promethazine (Avomine) for nausea, Norfloxacin for diarrhoea and cetirizine for allergy.

Return a day early : You should neither start a trip on the first day of your vacation nor return on the last. This causes unnecessary stress, and adds to fatigue. You can have more fun in fewer days, than being under pressure all the time.

TRAVEL SICKNESS

Timely Prevention Works Best

Some people become miserable during travel. Soon as the wheels roll in motion, their problems begin. There is first a feeling of restlessness, which quickly progresses to a cold sweat, dizziness and then vomiting and that very sick feeling in the tummy. You wish you had never started on the trip. If that's how you feel, try these simple preventive steps:

Eat light : Go easy on your stomach. Eat less before setting out and avoid large meals on the way. That's because a full stomach makes matter worse. The gravitational inertia the stomach must suffer when the body is in movement can set it rolling.

Swallow anti emetic pill : Take an over-the-counter antihistamine pill that can check symptoms for the next four hours or so if taken 30 to 60 minutes before setting out for the trip. The best names in this line are Avomine, Phenergan, Benadryl and Migril. Remember however that these medicines could make you drowsy, so leave the

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1874 Transit of Venus Observations of Pathani Samanta Chandrasekhar

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Pathani Samanta Chandrasekhar, of Orissa, is a poignant figure of a classical Siddhantic Astronomer of India, who survived into the 20th century (he died in 1904).

The year 2004 is a very appropriate year to remember his work and, in particular, to put together his observations of the 1874 Transit of Venus. Not just observations – predictions too, as he was a Siddhantic Astronomer, completely un-influenced by the western schools of Astronomy, and to some extent – unaware of it, during the early phases of his Astronomical efforts.

Samanta Chandrasekhar was born on the 13th of December 1835, at Khandapara, in Orissa. His full name was Mahamahopadhyaya Chandrasekhar Singh Harichandan Mohapatra Samant, but he was better known as Pathani Samanta. His lifetime Astronomy efforts were summarized by him in 'Siddhanta Darpana', which was published in 1899, by Calcutta University. The original manuscript of 2500 Sanskrit shlokas was written in Oriya script, on palm leaves, by Samanta Chandrasekhar.

Samanta Chandrasekhar did not have a formal University education and his interest and efforts in Astronomy were completely self taught, from manuscripts of Siddhantic Astronomical treatises, that he had access to. It is very evident that he had no exposure to the revolutionary advances in Astronomy between the 17th and 19th centuries, until rather late in his Astronomical career, and very little, even towards the end of that. He remained a complete Siddhantic Astronomer in the classical mould, uninfluenced by more recent developments.

Chandrasekhar was a keen observer and made meticulous observations of celestial objects with instruments that he had made himself. He was deeply perturbed on finding that the ephemeral elements calculated from classical siddhantic principles did not agree with his observations. The same perplexity had also been faced by Sawai Jaisingh, early in the 18th century, and had given rise to the construction of his gigantic masonry observatories for the correction of ephemeral elements. One underlying factor that had been responsible for these perplexities was the freezing of classical Indian

astronomical calculations away from observational verifications. The precession of equinoxes (Ayanamasa) had been noticed as far back as the Vedic times, by Indian Astronomers and had been entering the calculation of ephemeral elements as bija corrections – ad hoc corrections that needed to be applied with the passage of time, to incorporate the changes in ephemeral elements arising from precession. For about a thousand years before the time of Sawai Jai Singh or Pathani Samanta – the emphasis had shifted away from observational verifications and ephemeral elements had remained uncorrected.

These perplexities led Samanta to make a life time of observations with simple handmade instruments, correct

the ephemeral elements from these, and create predicted ephemeral elements in the classical Siddhantic format for future observations. The resulting ephemeral elements were amazingly accurate. Samanta's work was in the classical mould – with the assumption of a geocentric Universe, although his own model included the planets other than Earth, as revolving around the Sun.

Equivalent mathematical formulations exist for calculation of ephemeral elements in the two different world systems – Geocentric or Heliocentric – and many observed phenomena require only the appropriate framework of calculations in order to accurately predict possible celestial events. Thus, Samanta's

inability to envisage or accept the Copernican revolution, did not prevent him from making many accurate calculations of contemporary celestial events in his lifetime and observing them. The most interesting of the celestial phenomena in his life time was the 09 December 1874 Transit of Venus.

This rare and inspiring event was visible from India and many other parts of the world. The Transit of Venus 8 years following that, in 1882, was not visible from India. Such an event will again be visible on the 8th of June 2004, from India and other parts of the world, and is generating a lot of excitement amongst the amateur astronomers and educators. The underlying excitement of this event, being the possibility of recreating historical measurements of the Earth-Sun distance by students world wide, through observations of the timings of this transit.



Pathani Samanta Chandrasekhar

Going back to the year 1874 – there must have been considerable excitement at that time too, with efforts from Astronomers worldwide, making expeditions to India, as one of the locations from where, the event was visible. There were also efforts by Observatories under the then British Government in India, to study this event. And then, there were observatories built by private individuals and princely states where activities were intense, for the observations of this event. Some popularizations efforts also seem to have been in evidence. Chintamani Raghunathachary, of Madras observatory, for instance, had made a popular booklet on this event, that had been translated into many languages, including Urdu. In all probability, none of this excitement reached the remote Khandapara regions of Orissa, where Samanta could have heard of this event.

Pathani Samanta observed the 1874 Transit of Venus – and reported it in his Siddhanta Darpana as



(P.C. Naik and L. Satpathy – Bulletin of Astronomical society of India)

Arun Kumar Upadhyaya, in his translation of the Siddhanta Darpana – interprets this Shloka as –

“Solar eclipse due to Sukra (Venus) – To find the eclipse of the Sun due to Sukra, their bimba (angular diameter) and size of other tara graha is stated. In Kali year 4975 (1874 AD) there was a Solar Eclipse due to Sukra in Vrischika Rasi (Scorpio). Then Sukra bimba was seen as 1/32 of solar bimba which is equal to 650 yojana. Thus it is well proved that bimba of Sukra and planets is much smaller than the Sun.”

Did Samanta hear that there was going to be a transit and set out to observe it – or did he find that there was to be such an occurrence from his lifetime work of creating accurate

ephemeral elements? Most probably, the latter, as there seems no evidence that there was any European Astronomical activity in the regions of Orissa, at that time. The Italian expedition from the Palermo Observatory was to Muddapur in Bengal a neighbouring state to Orissa and could there have been some information that reached to Kandapara? It is not certain and there seems no evidence of it. Even if the information did reach, Samanta would not have accepted it without his own calculations agreeing with that.

All in all, it seems possible that not only did Samanta observe this Transit, but, he predicted it from his own calculations, unaware, of the excitement in the rest of the world arising from the Transits of Venus – in the 17th, 18th and 19th centuries.

The mention of the ratio of the bimba or apparent angular diameters of Venus and Sun as 1/32 is very interesting.

On the date of these observations – the 9th of December 1874, the apparent angular diameters of Sun and Venus, respectively, were – 32 minutes, 29 seconds of arc and 1 minute, 3 seconds of arc. The ratio then would have been discernible as 1: 30.93. This ratio would have small variations from one transit to another due to the ellipticity of orbits involved. In the year 2004, for instance, the apparent diameters are – 31 minutes, 31 seconds for Sun and 58 seconds of arc, for Venus so that the ratio discernable, would be 1:32.6 for the coming Transit of Venus.

Pathani Samanta's observations were completely non telescopic, and made with handmade instruments – and the accuracy achieved seems extra ordinary. In theoretical calculations and observations of the Transit of Venus, Samanta's achievement would be considered comparable to that of Jeremiah Horrocks, though poignantly anachronistic.

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wheel to somebody else.

Take the front seat : Sit in the comfort zone. In an automobile, the front passenger seat is the best place to be in because the motion is at minimum. Ask the driver to be careful and desist from sudden acceleration or decelerations.

Look straight as an arrow : It is best to look straight ahead. If you are in the hills, a moving horizon can be most disturbing. Just concentrate on the road in front and stop in between, if you wish to soak in the natural beauty of the valley and the hills.

Don't read : Avoid activities that need close eye attention. Reading, knitting or playing cards may aggravate or even cause motion sickness. Keep your head still, rested against a seat back.

Never be cramped for clean air : Always allow plenty



of fresh air to get in. A close and smelly environment can make matters worse. Don't smoke or sit near smokers.

Avoid bad air : Do not stop where the air hangs heavy with odour or is unpleasant to the nostrils. If a travel companion experiences motion sickness, stay away and let him clean himself thoroughly.

Stay relaxed : Keep yourself cool. A bad mood can make matters worse, while a light mood can ease the situation.

Injectables work, when other things fail : Once vomiting begins, there is little sense in taking the pill now. It's like closing the stable door after the horse has bolted! At this stage, it is best to break journey and take rest. Rest usually helps quieten matters. If you feel particularly bad, the best course is to find a doctor. A shot of antihistamine injectables can check symptoms quickly.

