and reach their maximum in the evening.⁴ In contrast, adrenaline and noradrenaline concentrations and alertness normally reach a maximum at about midday.⁵

Circadian rhythms also differ in the degree to which they are exogenously and endogenously controlled. Noradrenaline concentrations and heart rate are largely exogenously determined, while body temperature, adrenaline, alertness, and various measures of performance have a larger endogenous component.⁶⁻¹⁰ When people arrive in a new time zone or work at night the timing of the exogenous components of their rhythms can adjust almost immediately if they schedule their activities well. The endogenous component, however, can take up to three weeks to adjust¹¹ and is unlikely to do so over the normal maximum of six or seven successive night shifts.¹² Consequently the normal temporal relation between different rhythms is disrupted, which may be responsible for feelings of disorientation, malaise, and "jet lag."¹³ Further, the lack of adjustment of the endogenous timing system results in disturbed sleep.¹¹⁻¹³

Without time cues the endogenous circadian timing system adopts a 25 hour cycle in most people,⁷ which explains why people generally suffer more after an eastward than a westward flight.¹¹ It also explains why shift systems that rotate in the direction "morning, evening, night" are generally better.¹⁴ Extreme "morning types" (those who feel wide awake early in the morning but find it difficult to stay up late) may, however, have fewer problems than "evening types" after an eastward flight since their endogenous timing system may be shorter.¹⁵ Indeed, "evening types" may be able to reduce the problems they have after an eastward flight by "stretching" their rhythms through, say, 16 hours rather than contracting them through eight hours. This means staying awake for 40 hours rather than trying to sleep after only eight hours of wakefulness.

Other attempts to alleviate the problems of shiftwork and rapid time zone transitions are based on "resetting" the endogenous timing system. An "anti-jet lag diet" has been proposed from animal studies,¹⁶ although its efficacy for humans has yet to be established. More promising perhaps are recent suggestions that jet lag may be reduced by the appropriate timing of exposure to bright light,^{17, 18} which suppresses the secretion of melatonin by the pineal gland.¹⁹ Alternatively, melatonin may be given before and after flights.²⁰

Giving melatonin reduces subjective ratings of jet lag severity but may not necessarily improve performance efficiency. Circadian rhythms in certain types of complex cognitive performance can behave very differently from those in most physiological processes,^{8, 21} and subjective impressions of performance at different times of day can differ considerably from actual ability.^{22, 23} Until we understand better the endogenous circadian system controlling performance rhythms we must be cautious about ignoring our diurnal nature.

SIMON FOLKARD

Senior Scientist,
MRC Perceptual and Cognitive Performance Unit,
Laboratory of Experimental Psychology,
University of Sussex,
Brighton BN1 9QG

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Beware congesting nasal decongestants

The bad effects of topical "decongestants" (vasoconstrictors) used long term on the nasal mucosa have long been known, but they remain widely prescribed, are prominently displayed in chemists' shops, and are advertised on posters and television. The *British National Formulary* roundly deprecates their use: "Vasoconstriction of the mucosal blood vessels . . . reduces the thickness of the nasal mucosa. [Decongestants] are of limited value as they can give rise to a rebound phenomenon as their effects wear off, due to a secondary vasodilatation with a subsequent temporary increase in nasal congestion. This in turn tempts the further use of the decongestant, leading to a vicious circle of events. The formulary says that if decongestants are to be used then ephedrine is the drug of choice and should be used for a short time only. In our experience up to seven days' use is relatively safe.

The vasoconstrictors are usually sympathomimetic amines or the chemically related imidazoles. The α adrenergic agonists constrict subepithelial precapillary sphincters, arterioles, and venous sinuses, and the β agonists mediate vasodilatation. Prolonged pharmacological vasoconstriction is probably followed by vasodilatation, and ultimately the muscle responds less well to any repeated nasal application of the vasoconstrictors. The most commonly used sympathomimetic decongestants are ephedrine and phenylephrine, while the common imidazole vasoconstrictors are xylometazoline, oxymetazoline, and naphazoline. Of 19 drugs with local actions on the nose listed in the *Monthly Index of Medical Specialties* 11 contain one or other of these substances.

Entry to the vicious circle of decongestant use commonly occurs during acute episodes of nasal airway narrowing. After coryza, for example, the decongestant is obtained over-

the counter or is prescribed by the doctor. Relief from nasal obstruction is often immediate but is commonly followed by reactive nasal congestion. Once the nasal block develops again, the troubled patient returns to the decongestant. In time the vasoconstrictor drops or spray damages the nasal mucous membrane.^{1 2 4 6} Some patients intuitively give up further use—to their advantage.

Some doctors caution that use should be for a limited time, but others are ignorant of the potential for damage—and prescription and use of these injurious drops continues. Some patients continue use of the drugs by themselves, buying them from chemists after the prescription finishes. Many patients buy the nasal drops directly from the chemist and continue their use unsupervised, and widespread advertising encourages this. The warnings that do exist on some cartons are often not spotted, misunderstood, or disregarded.

In the rhinitis clinic of the Royal National Throat, Nose, and Ear Hospital 460 new patients were seen in 1985. In 60 (13%) there had been prolonged (more than two months) use of vasoconstrictor nose drops for persistent nasal obstruction. One patient had been using them for more than 10 years. These patients' symptoms were made worse by decongestant drops and sprays, and the prolonged use of decongestants was part of, or the whole cause, of the continued symptoms. We substituted low absorption corticosteroid sprays—for example, beclomethasone or budesonide—which themselves have modest vasoconstrictor action⁸—and advised stopping other topical preparations. Usually the dose of the low absorption spray can be reduced gradually. Any persistent purulent nasal catarrh should first be treated by a systemic antibiotic.

It is now commonly suggested that pharmacists could be the first to advise on managing disorders that are popularly considered to be minor. For example, in a BBC Radio 4 broadcast in June 1985 a pharmacist suggested this and gave hayfever as the first example of a disorder where the pharmacist might be of help. Pharmacists will presumably recommend what they have on display and can sell without prescription. These may, however, be drugs which the *British National Formulary* and many doctors warn against—for example, the decongestants.

We suggest, therefore, that nasal decongestant sprays and drops should not be advertised to the public and that a warning, "Use of these drops for more than five days becomes increasingly harmful," should be printed prominently on the containers. Without these safeguards then perhaps decongestants should be available only on prescription.

L H CAPEL

Consultant Physician,
Rhinitis Clinic,
Royal National Throat, Nose, and Ear Hospital,
London WC1X 8DA

A R SWANSTON

Senior Registrar,
Royal National Throat, Nose, and Ear Hospital,
London WC1X 8DA

AIDS and the condom

Since the human immunodeficiency virus (HIV) is transmitted primarily through blood, seminal fluid, and female genital secretions, any barrier blocking exposure to these fluids should reduce disease transmission. This thinking has led to suggestions in some "safe sex" guidelines that using condoms may confer some protection against acquired immune deficiency syndrome (AIDS), but until recently there have not been enough data to support a direct message.¹

Now, evidence is beginning to accumulate. In an in vitro study neither AIDS associated retrovirus nor a mouse retrovirus permeated any of five commercial brands of condoms.² Stronger supportive data were presented at the Paris conference on AIDS in June this year: of 32 sexual partners of adults with AIDS who did not have antibodies to HIV at the start of the study, 13 developed antibodies (M A Fischl *et al*, unpublished findings). Seroconversion was much more likely in those who used barrier methods inconsistently or not at all. Spermicides may give additional protection: HIV is inactivated in vitro in 60 seconds by 0.05% nonoxynol-9, an ingredient present at concentrations of 5-12.5% in several spermicides widely used in Britain.³

The studies are thus few and small. Bigger studies are urgently needed, as is more brand specific testing—preferably in vivo. But the existing findings point resolutely to the HIV retrovirus not being able to permeate the condom membrane and being inactivated by the active ingredient in commercially available spermicides.

There is, however, no evidence that the standard condom membrane will stand up to anal sex. Manufacturers have concentrated on producing ever finer products offering higher sensitivity and only now are beginning to produce condoms especially designed for prophylactic use. A French firm with a British agent, for example, has produced what it claims is a tougher condom, and a firm in the north of England will market early next year a self sealing condom to safeguard against spillage. Some of the larger manufacturers seem reluctant to enter this market. They worry that by promoting their products for prophylaxis they may run an unacceptably high risk of litigation. But behind talk of product liability may lie fears about product image.

Many will argue that only radical changes in sexual behaviour will substantially reduce the spread of the virus. This may well be so but it will take time. Messages urging monogamy or celibacy are not easy to put across, and there is no guarantee of compliance. Clearly the dangers of relying solely on barrier methods to prevent AIDS must be emphasised, as must the need for more efficient contraception to prevent vertical transmission, but in the meantime a "belt and braces" approach can do no harm.

The time is now ripe for a major public information campaign on evidence for the protective effects of condoms and spermicides. People should be encouraged to use them and provided with clear instructions for their use. Some attempt must also be made to improve their public image. The main message of the campaign will need to be made on television and radio. Until now contraception has been one of the taboos of television. The Independent Broadcasting Authority argues that such advertising offends against "public taste and decency." Yet its own consumer research shows that 56% of the public think contraceptive advertising should be allowed on television and 74% say it would not make them feel uncomfortable. There is a case for setting aside the sensitivities of a minority when lives are at stake.

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