CHAPTER 9

Disequilibrium: Jet Lag, Motion Sickness, Cold Exposure, and Heat Illness

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When travelers cross several time zones, go to hot or cold climates, or are subject to novel motion stimuli, they may face problems adapting to new environmental situations. This chapter presents some common states of disequilibrium likely to be encountered by the traveler and suggests practical approaches to the problems. Socioeconomic position, class, or economic status can affect individuals with these conditions profoundly, but like most afflictions, studies ignore these factors. Increasingly, we can expect more displaced people that present with travel-related conditions to be poor. Evidence from which to base prevention, advice, and treatment in these circumstances is limited at best, so experience and clinical judgment, tailored to the specific situation the traveler faces, are paramount.

JET LAG

When many time zones are crossed quickly, the traveler's normal sleep–wake cycle is disrupted and is put into conflict with the body's underlying physiologic circadian rhythms. The traveler experiences disturbed sleep, loss of mental efficiency, and fatigue during the day—symptoms commonly known as "jet lag," which when combined with the fast-paced, security-conscious stress of travel today can make the traveler feel even more uncomfortable. "Surface travel lag" is almost unheard of, but there are stresses inherent in any travel away from home that, combined with the increasing sleep deprivation that has become a part of so-called normal everyday life, can result in similar symptoms.

Jet lag symptoms increase with the number of time zones crossed and generally begin when there is a 2-hour difference. The incidence of jet lag in travelers is almost universal, and symptoms can persist for 1 week or more. Circadian rhythms may take up to 2 weeks to adjust. Specific complaints include insomnia, daytime sleepiness and fatigue, poor concentration, slowed reflexes, indigestion, hunger at odd hours, irritability, depression, exacerbation of major psychiatric disorders, increased vulnerability to infections, headache, myalgias, and dysphoria. Sleep disturbances persist longer than the other symptoms. The lower oxygen content aboard jet planes may exacerbate the symptoms.

Older people tend to have more difficulties, although experienced travelers and those with more convenient travel arrangements report fewer symptoms. "Morning types"—individuals who tend to go to bed earlier and awaken earlier in the day—may be more susceptible than "evening types." There is little data on the impact of co-existing morbidity on jet lag, although those with pre-existing sleep disorders are expected to have worse symptoms. Travelers may have affective disorders from jet lag desynchronization. Chronic jet lag has been associated with neuro-anatomic changes, persistent cognitive deficits, and other health problems.

Performance errors in pilots, reduced functioning among athletes, and decreased mental performance among diplomats are ascribed to jet lag. Those crossing many time zones quickly should be advised to avoid potentially hazardous activities on arrival until symptoms

improve. Operating dangerous machinery including motor vehicles, undertaking risky recreational activities, and making critical decisions are to be avoided. Competing athletes are advised to schedule sufficient arrival days before events. Melatonin and nonbenzodiazepine benzodiazepine-receptor agonists (NBRAs; see below) are not banned by the World Anti-Doping Agency, so athletes could consider their use.

While the role of socioeconomic status, class, or other contextual markers has not been studied in the severity of jet lag symptoms, individuals under both acute and chronic stress can be expected to fare worse than those who have more control over their lives. Wealthy travelers are likely to have increased access to preferred travel schedules, choice airline seats, comfortable layovers, purchased food and refreshments, pharmaceuticals, luggage-handling services, massages, traveling nannies, etc., in contrast to the harried, resource-poor family on a once-in-a-lifetime journey.

Prevention and Treatment

Minimizing the effects of jet lag is best accomplished by a multifactorial approach. Few controlled studies have examined the various means of preventing jet lag, and none has compared different available modalities. Current approaches are reviewed in order of ease of use for travelers.

Melatonin

A nonvisual, photoreceptive, monosynaptic retinohypothalamic tract directly mediates the synchronization of the sleep–wake cycle with the light–dark cycle. Melatonin from the pineal gland modulates this link, as does the light–dark cycle. Melatonin is produced only during nighttime darkness in sighted individuals and is affected only by exposure to bright light. Melatonin resets the phase shift curve marking the circadian phase position. Ingestion of an appropriately timed physiologic dose (0.5 mg) of exogenous melatonin can shift the phase response curve the required number of hours of time zone change. Several studies demonstrate benefits on subjective rating of jet lag. Melatonin delays circadian rhythm when taken in the morning and advances it when taken later in the day. **Table 9.1** shows one possible schedule for timing melatonin ingestion for eastward and westward travel beginning the day before departure and continuing for 3 days after arrival. This is time zone traverse-specific.

Times for exposure to bright light are also given there, since light exposure is synergistic with melatonin. The suggested physiologic melatonin dose can produce drowsiness before departure, so the dose can be adjusted downward to that which does not cause unacceptable drowsiness and repeated every 2 hours to a total of 0.5 mg. Over-the-counter preparations available in the United States in health food stores are not standardized nor can potencies be guaranteed. Liquid preparations allow titration of an appropriate dose for an individual. Melatonin is not available in some countries and in others only by prescription. There are no data on long-term safety of exogenous melatonin ingestion nor any reports of safety during pregnancy. A Cochrane review by Herxheimer found reports of short-term adverse effects of melatonin to include confusion, ataxia, headache, convulsant effects, and blood-clotting issues (prothrombin increased or decreased, suspected interaction with warfarin), as well as cardiovascular symptoms including chest pain and dyspnea. People with epilepsy and those taking warfarin or other anticoagulants should not use melatonin without an informed medical discussion.

An alternative dosing schedule is to take melatonin at bedtime on arrival at the destination. Most trials have used large doses, 5-8 mg, continued for 3-7 days after arrival. A commonly reported side effect on this regimen is drowsiness after ingestion.

Nonbenzodiazepine Benzodiazepine-Receptor Agonists (NBRAs)

Short-term pharmacologic manipulation of the sleep–wake cycle with hypnotic drugs to induce sleep is a convenient way to manage jet lag in healthy travelers. There may not be much improvement in performance, and hypnotics do not appear to adjust circadian rhythms in humans. The short-acting drugs known as nonbenzodiazepine benzodiazepine-receptor

TABLE 9.1 Jet-Lag Treatment with Melatonin

Time Zone Change	1-6 h	7-9 h	≥10 h
	Time to take melatonin the day before and the day of departure		
Travel from east to west	When you awake	When you awake	When you awake
Travel from west to east	About 15:00 h (3 pm)	About 15:00 h (3 pm)	When you awake
Time to take melatonin on arrival			
Travel from east to west	Day 1: when you awake	Day 1: when you awake	Day 1: when it is the same time at departure as when you took it yesterday
	Days 2 and 3: 1-2 h later than the day before	Days 2 and 3: 1-2 h later than the day before	Days 2 and 3: 1-2 h later than the day before
Travel from west to east	Day 1: when it is the same time at departure as when you took it yesterday	Day 1: when it is the same time at departure as when you took it yesterday	Day 1: when it is the same time at departure as when you took it yesterday
	Days 2 and 3: 1-2 h earlier than the day before	Days 2 and 3: 1-2 h earlier than the day before	Days 2 and 3: 1-2 h earlier than the day before
	Time periods to be in or to avoid bright light		
Travel from east to west	Get bright light later in the day	Get bright light in the middle of the day, avoid bright light later in the day	Get bright light in the morning and avoid it the rest of the day
Travel from west to east	Get bright light in the morning	Get bright light in the middle of the day, avoid bright light earlier in the day	Get bright light in the middle of the day, avoid bright light later in the day

Adapted from Bezruchka, S.A., 1999. The Pocket Doctor. The Mountaineers, Seattle, pp. 54-56.

agonists (NBRAs) have become very popular sleep agents, as typified by zolpidem. Others include zopiclone or the enantiomer eszopiclone, and the even shorter-acting zaleplon. They may be used to treat early awakening, while using melatonin, although studies are lacking and more adverse effects may be expected. Zolpidem, like some short-acting benzodiazepines such as triazolam, has been shown to have idiosyncratic side effects including amnesia, dysphoria, excitability, and somnambulism. Consider a home-based trial before departure to gauge the side effects. Some travelers use these agents during flights, taking them at the destination's sleep time.

The usual adult dose of hypnotics should be halved for first-time users and for geriatric patients, and travelers on NBRAs should be warned not to drink alcoholic beverages or to use other medications that cause drowsiness (e.g., antihistamines) concurrently. Triazolam has been banned or allowed only limited use in several European countries due to reported adverse side effects (psychosis). Other benzodiazepines are longer acting and less suitable for use. Use of these drugs has been associated with increased mortality.

Ramelteon

Ramelteon is a melatonin receptor agonist approved for insomnia and is said to not cause rebound insomnia like the NBRAs and benzodiazepines. It appears to have phase-shifting

TABLE 9.2 Pharmaceuticals to Consider for Jet Lag

Drug	Adult Dose[a]	Elimination Half-Life
Hypnotics		
Zolpidem (Ambien®)	5-10 mg p.o. hsb	3-5 h
Zaleplon (Sonata®, Starnoc®Andante®)	5-10 mg p.o. hsb	1-2 h
Eszopiclone (Lunesta®)	1 mg p.o. hsb	6 h
Zopiclone (Imovane®)	7.5 mg p.o. hsb	6 h
Oxazepam (Serax®)	10-15 mg p.o. hsb	8-10 h
Circadian rhythm agents		
Ramelteon (Rozerem®)	1-8 mg p.o. hsb	1-2 h
Melatonin	See text and Table 9.1	
Wakefulness promoters		
Armodafinil	150 mg p.o. am. on arrival	

[a]Use half the dose for elderly patients or first-time users. b, bedtime.

effects for treating jet lag. One study found that doses of 1 mg at bedtime for four nights in the new destination decreased sleep latency, but light exposure may be more beneficial. Drug interactions and hepatotoxicity may limit extensive use in those with co-morbidities. Ramelteon could also be used like the NBRAs above.

Table 9.2 lists pharmaceuticals to consider for jet lag.

Armodafinil

One study has found that armodafinil, a wakefulness-promoting drug, increased wakefulness after eastward travel in a dose of 150 mg in the morning on arrival. Side effects such as headache, nausea, and diarrhea may limit its use. Modafinil may also be useful but neither drug is FDA-approved for this use.

Agomelatine

Older people who adapt less well to jet lag could be treated with agomelatine, a melatonergic antidepressant, although studies of its efficacy are lacking, and it is not available in the United States.

Sleep Schedule Adjustment

Traditional advice has been to adjust the sleep schedule, beginning 3 days before departure, gradually moving bedtime closer to the customary time at the destination. For instance, if traveling eastward, a traveler would try to go to bed 1 hour earlier each succeeding night in the 3-day period before departure. Smartphone applications are available to help calculate sleep times. If traveling westward, the traveler would try to stay up 1 hour later each night in the pre-travel period. Because it is easier to stay up later than to retire earlier, westward flights across a few meridians result in faster adaptation than eastward ones. Naps should be avoided on eastbound trips. It is useful to exercise before, during, and after the flight and to maintain good hydration.

If traveling across multiple time zones and returning after a day or two, it is better not to try to adjust to the proximate destination but to maintain the home sleep schedule. If traversing more than three time zones, scheduling a stop-over of a day or more in the travel itinerary may help with readjustment of the sleep–wake cycle. Resetting the watch early on each flight to the new local time at destination is advisable for orientation. On arrival at the destination, activities should be scheduled that are appropriate for the new local time. For the first few days after arrival at a destination, major decisions should be avoided if possible, and important meetings should be scheduled at the individual's most alert time of

TABLE 9.3 Resetting the Circadian Clock with Bright Light

Direction of Travel	External Clock	Behavioral Change	Circadian Clock	Light Exposure
West to east	Turn watch forward	Earlier bedtime, earlier awakening	Turn back circadian clock	Bright light in early morning at destination
East to west	Turn watch backward	Later bedtime, later awakening	Advance circadian clock	Bright light in afternoon at destination

the day at home. Vigorous physical exercise on arrival—mid-morning for travel east and late afternoon for going west—helps.

Bright Light Exposure

The circadian clock can be shifted by exposure to bright light. Such techniques are of no value for the blind. To use principles of phototherapy to reset the circadian clock, the traveler should expose him- or herself to intense bright light (7000-12,000 lux, comparable to that of natural sunlight at sunrise). An exposure time of approximately 5-9 hours is needed. Light episodes before 0400 (at the originating time zone) retard the circadian clock, whereas those after 0400 (at the originating time zone) advance the clock. Thus travelers going eastward should expose themselves to bright light for a few hours early every morning after arrival at the destination. Those traveling westward should expose themselves to bright light in the late afternoon. Three to four days of such light exposure will entrain the original clock and allow it to be reset for sleeping at the destination time zone bedtime (**Table 9.3**).

The required light level can be measured with a digital camera (operating in manual mode). When set for ISO50, a camera meter reading of f:5.6 at 1/60 s indicates the brightness comparable to 11,000 lux. Short wavelength light exposure (blue, green), even if less intense, may be more effective than white light.

Wearing an eye shield on the plane or sitting by an unshaded window at the appropriate times can contribute toward circadian clock resetting during the journey. Enhancing destination time–appropriate sleep with ear plugs or playing soothing sounds on an audio device may help. On arrival, sunglasses are best not worn when it is necessary to get exposed to bright light, and it is advisable to be outdoors when possible. Wear a visor and/or sunglasses when outdoors in bright light that is not at an appropriate time. When indoors, the window curtains should be open and bright room lights should be kept on during the period of phototherapy. Indoor light is much dimmer than required, but it can shift the circadian clock.

Often travel schedules and conditions may prevent a traveler from using scheduled exposures to bright light to facilitate an adaptation to the new time zone.

Jet Lag Diet and Other Remedies

The Argonne National Laboratory Jet Lag Diet tries to reset the circadian rhythm by alternative feasting and fasting, beginning 3 days before departure, and by timing the consumption of high-protein breakfasts and lunches, high-carbohydrate dinners, and caffeine. There is one study using it to advantage in US soldiers traversing nine time zones. Websites calculate this timed diet, and there are apps for smartphones to help adjust to the new time zone. Slow-release caffeine in a dose of 300 mg taken in the morning on arrival appears to have benefits to increase wakefulness. This may be more beneficial than 2 cups of espresso coffee. Other remedies, such as "anti-jet lag" pills sold over the counter or plant products, are of questionable efficacy.

In summary, jet lag is unavoidable; there are substantial individual variations in symptoms; and there are many ways of minimizing its effects.

MOTION SICKNESS

Motion sickness is not a true disease but a normal response to a stimulating situation. It can be induced in anyone with a normally functioning vestibular system, given the right stimulation, but it is not produced by voluntary movement. People who lack vestibular function are immune. Vertigo, by contrast, is a sensation of movement without the stimulation of activity or sequelae of motion.

Motion sickness as a generic term includes sea sickness, motor vehicle sickness, air sickness, and other disorders such as ski sickness. One study collected data from 20,029 passengers on ferries on sea routes across the English Channel, Irish Sea, and North Sea and found that more than one-third of passengers reported some symptoms of motion sickness, and 7% vomited. The incidence of illness was greater in females than males, and there was a slight decline in incidence with age. Those who traveled frequently reported less motion sickness; this was presumed due to either habituation or self-selection.

Overpacked buses on winding mountain roads are common sites where adventure travelers experience motion sickness symptoms directly, while others may find it induced by a camel ride. Ski sickness is a special form of motion sickness produced by unusual and contradictory sensory information among the visual, vestibular, and somatosensory systems and develops while performing winding turns on uneven ground, with insufficient visual control, especially on foggy or white-out days with reduced visibility.

Women may be twice as susceptible to motion sickness as men, and more so toward the end of menstruation. Pregnant women are at an increased risk of motion sickness. Those with a history of migraine may be more at risk. A past history of motion sickness is strongly predictive of future problems. Children experience more symptoms and tend to outgrow them. Youth who engage in proprioceptive physical and sporting activities at an early age may be less susceptible. Retention of adaptation to motion sickness stimuli is retained for at least 1 month.

Nausea is a common presentation of motion sickness and may be preceded by pallor and cold sweats; eventually, vomiting occurs. Sufferers may express a desire for cool, fresh air, although ambient air temperature is not found to influence susceptibility. Hypersalivation, yawning, hyperventilation, and frontal headache are reported. Drowsiness, lethargy, inhibition of gastric motility, and loss of performance proficiency are the secondary symptoms of motion sickness. Diminished gastric motility reduces the absorption of oral drugs. Lethargy may take longer to resolve once the stimulus is gone. Altitude sickness should be included in the differential diagnosis.

Prevention

Studies on motion sickness use self-reports or laboratory experiments simulating space sickness or extreme sea conditions. Advice is based on such limited information and anecdotal reports. General advice includes resting before the anticipated motion and beginning with an empty stomach. Generally, high-sodium foods, as well as those that are calorie dense or high in protein or fat, including cheese and milk products, may be more associated with symptoms, as is an increased frequency of eating. One study suggested a pleasant taste of food was more important than composition, although liquid high-protein, low-carbohydrate consumption produced less cardiac vagal tone. An ear plug in the nondominant side has been reported to help minimize symptoms.

Seeking a place in the vehicle where motion is least, sitting in a semi-reclining position, and minimizing head motion, as well as looking at the horizon, help. In a car, sit in the front seat and look out the front window at distant scenery. On an airplane, consider a window seat over the wings, and look out of the windows. For the susceptible on a ship, choose a cabin on a middle deck near the waterline. If lacking other modalities for an individual highly susceptible to seasickness, consider advising the use of a blindfold when inside a room on a ship. Symptoms of motion sickness may decrease with prolonged exposure to changing vestibular and optokinetic stimuli, for example, developing one's "sea legs."

TABLE 9.4 Medication for Motion Sickness

Drug	Adult Dose	Side Effects
Granisetron (Kytril®)	2 mg p.o. single dose	
Ondansetron (Zofran®)	8 mg p.o. single dose	
Phenytoin (Dilantin®)	200 mg p.o. single dose	
Cyclizine (Marezine®)	50 mg p.o. q4-6 h	Minimal sedation
Dimenhydrinate (Dramamine®)	50 mg p.o. q4-6 h	Sedation
Meclizine (Bonine®, Antivert®)	25-50 mg p.o. q6-12 h	Mild sedation
Promethazine (Phenergan®)	25 mg p.o., p.r. q8-12 h	Moderate sedation
Scopolamine patch (Transderm Scop®)	¼–1 patch applied to bare skin q72 h	Dry mouth, blurry vision
Scopolamine hydrobromide	0.4 mg p.o. q6 h	Dry mouth, blurry vision
Ephedrine sulfate	25 mg p.o. q6-12 h	Cardiovascular (counteracts sedation)
Pseudoephedrine	30-60 mg p.o. q6-12 h	Cardiovascular (counteracts sedation)
Dextroamphetamine (Dexedrine®)	5 mg p.o. q a.m.	Cardiovascular (counteracts sedation)

Those who are particularly susceptible to motion sickness could undergo desensitization through exercise procedures. Search the Web for "motion sickness desensitization" to find resources. Physical therapists specializing in vestibular disorders could be consulted. However, behavior modification techniques, including cognitive therapy, biofeedback, and desensitization therapy, require frequent exposure to motion over a considerable time to be effective.

Antihistamines

Several pharmaceuticals are useful to alleviate the symptoms of motion sickness, especially if they are started prophylactically before severe symptoms are manifest (**Table 9.4**). H1 receptor antihistamines useful for motion sickness include cyclizine, dimenhydrinate, and meclizine. Cyclizine is thought to affect gastric dysrhythmias; dimenhydrinate may work as a sedative; and meclizine affects the vestibular system. Common side effects include sedation and a dry mouth. These antihistamines are available without a prescription in the United States, but availability elsewhere should be determined. Caution is advised for use by the elderly, as these drugs may cause confusion and hallucinations, and caution is advised for use in pregnant women, although the common antihistamines are considered category B.

Scopolamine Patch

Scopolamine (an anticholinergic), when administered in the form of a transcutaneous drug patch, is widely used for prevention of motion sickness. The 0.5-mg patch is placed behind the ear, where skin permeability is highest, providing therapeutic levels of scopolamine for up to 3 days. The patch should be applied 4-8 hours before exposure to motion and must be worn for as long as the stimulus is present. Initial enthusiasm for this drug has waned because side effects are unpleasant, efficacy is disputed, and there is concern that the patch may decrease adaptation to motion sickness. Side effects include dry mouth (50%), blurred vision (25%), and occasional anisocoria. Lower doses (cutting the patch into halves or quarters) is not approved according to the package insert but may provide adequate protection and lessened side effects in some people. Scopolamine should not be used in those with conditions such as prostatic hypertrophy and glaucoma.

Scopolamine may cause drowsiness, impaired short-term memory, toxic psychosis (hallucinations, confusion, disorientation, confabulation), acute angle-closure glaucoma, or

urinary retention in susceptible people, especially the elderly. Side effects can become more severe after a long period of use. Anticholinergic intoxication can result after ≥16 days, especially if an allergic skin reaction develops where the adhesive patch is applied, thus allowing greater absorption of the drug. Withdrawal symptoms, including hypersalivation and increased gastrointestinal motility, are possible with prolonged use. Physiologic chemical dependency has been reported. Consider advising use of the patch for long journeys (≥3 days) in those who have a history of severe motion sickness. Oral scopolamine (0.4 mg) can be used for faster onset and shorter duration of drug activity.

Sympathomimetics

Sympathomimetics can potentiate the prophylactic effects of scopolamine and antihistamines and tend to counteract the sedation caused by these drugs. Ephedrine and dextroamphetamine are effective, useful additions for prophylaxis of motion sickness when either scopolamine or an antihistamine does not work well enough (**Table 9.4**) or there is an intense stimulus for <6 hours. Pseudoephedrine could be used in this case as well. The combination of promethazine plus dextroamphetamine has been found to have limited impact on psychomotor performance and to not increase sleeplessness. Sympathomimetics should not be prescribed for patients with cardiovascular risk factors or disease because the drugs are associated with palpitations, tachycardia, and elevation of blood pressure. Patients also need to be warned about possible central nervous system (e.g., restlessness, dizziness, tremor) and gastrointestinal (e.g., anorexia, dry mouth, change in bowel habits) side effects.

Phenytoin

Phenytoin and other anticonvulsants prevent motion sickness but are not widely used. One study found that a therapeutic dose of phenytoin was four times more efficacious than any other single pharmacologic agent in delaying onset of artificially induced motion sickness; it was twice as effective as a scopolamine/dextroamphetamine combination. The dosage used was 15 mg/kg per day given in divided doses at 4-hour intervals over a 20-hour period before the experiment. A blood level of 9 μg/mL appears to be protective. This regimen could have useful clinical applications, as phenytoin in this study did not produce sedation or any decrease in performance.

Other Drugs

Promethazine (a phenothiazine derivative) is used orally to prevent motion sickness. Drugs used to protect against radiation-induced nausea and vomiting, such as granisetron and ondansetron, have been used by pilots flying jet planes where psychomotor performance was maintained, although laboratory studies dispute their ability to influence motion sickness symptoms. Other drugs that have been proposed on the basis of anecdotal or small study reports include nifedipine (calcium channel blocker), triptan serotonin agonists (rizatriptan) in those with a history of migraine headaches, and selegiline (phenethylamine derivative). Ginger has been reported as a remedy for seasickness; however, studies have yielded conflicting results, perhaps owing to variation in the type of preparation used and the experimental design.

Anecdotal reports and small studies support the use of acupressure at the "P6 point" with a commercial device (Accu-Band, ReliefBand, Traveleeze, or Sea-Band) to prevent motion sickness.

Treatment

There are no controlled studies of different drugs to treat large patient populations experiencing the symptoms of motion sickness. Most reports use a variety of experimental situations to induce motion sickness in the subjects. Generally, intramuscular doses of prochlorperazine or trimethobenzamide are effective in treating nausea and vomiting. Rectal suppositories of prochlorperazine or trimethobenzamide may be effective, but onset of action is slower because of unpredictable absorption through the rectal mucosa. In one study of motion sickness in weightless conditions simulating outer space, 50 mg of promethazine or

0.5 mg of intramuscular (i.m.) scopolamine were reported to be effective, but 25 mg of promethazine or 50 mg of dimenhydrinate were not. Caution is necessary in using these agents for treatment if they have already been administered for prophylaxis.

For treatment of the secondary symptoms, IM ephedrine (25 mg) or dimenhydrinate (50 mg) are effective for dizziness, and parenteral metoclopramide (5 mg) aids in gastric emptying.

Nonpharmaceutical treatment of motion sickness may be attempted by putting the afflicted into the driver's seat, the cockpit pilot's chair, or other appropriate active-engaging positions. Keep the head and chest balanced over the hips. Controlled breathing and other behavioral measures may help. Do not read. Debilitated seasick persons need a safety harness if there is a risk they may be washed overboard. Those at bed rest should be supine, face up, head still, eyes closed, and secure in a well-ventilated area.

COLD EXPOSURE

Exposure to cold conditions is more common in travelers and residents due to increasing numbers pursuing recreation in cold environments and from disasters occurring to residents in such places.

Temperature homeostasis is problematic in the cold because humans lose heat easier than they retain it. Humans are relatively incapable of increasing metabolic rate when the temperature drops. The heat balance equation sums conductive, convective, radiative, and evaporative heat exchange. Body fat and skin thickness may protect from cold injury because of low thermal conductivity. About 40% of heat loss from a naked human is mediated by convection at an ambient temperature of 29° C (84° F) and a wind velocity of 0.9 m/s (2 mph). Radiant heat loss under the same conditions accounts for 45% of the total. Evaporative heat loss is the remainder, with half of that coming from the respiratory tract.

Peripheral thermal sensors to cold are found immediately beneath the epidermis, whereas warm receptors are deeper in the dermis and are 10 times more sensitive when there is rapid temperature change. They are not uniformly distributed, and there are many more cold receptors. The forehead, for example, has many more cold sensors than the lower leg. The upper torso has the greatest cold receptor density and sensitivity, with the lower torso, arms, and legs having similar numbers but with much lower sensitivity to cold. Measuring the core temperature assesses the overall status of the regulatory system, but this can be difficult to do in the environmental situations where problems occur. Carry low-reading thermometers in situations where cold conditions prevail. Electronic devices may be easier to read quickly by a stressed, cold operator. The debate on which site to monitor temperature is academic.

The major involuntary response to cold is to vasoconstrict the peripheral circulation and to contract muscles in shivering. Active exercise will increase body temperature, which will plateau 30-40 min after initiation, with higher levels resulting in higher core temperature increases. But even when exercising at 50% of maximal aerobic capacity, the increase is only 1° C. Aging results in a weakening of the vasoconstrictor response to cold, and older individuals do not shiver as much. (Sweating in response to passive heating is also lessened with aging.) Cyclical hormonal changes in women impact these responses. During pregnancy, the thermoregulatory system is very sensitive to heat produced by exercise, with steady-state core temperature during exercise dropping, possibly lessening thermal stress on the fetus.

Thermoregulation is affected by chronic exposure to very cold environments and by chronic exercise in both warm and cold situations, so an acclimated individual can work with less increase in core temperature. Ethnic groups who traditionally lived in warm environments may be more at risk of cold injury, as they appear to have less heat output or cold-induced vasodilation.

There may be some acclimatization to cold, with a cold-induced vasodilatation, or "hunting response," in some individuals. However, for most people, wearing appropriate clothing, wearing mittens rather than gloves, having chemical hand warmers, avoiding

extreme conditions, keeping the feet and hands dry, avoiding direct finger contact with cold surfaces, eating plenty, and maintaining sufficient hydration are important steps to prevent cold injury. Drinking cold water from a stream is more efficient than eating snow. Getting out of the wind can decrease convective exchange, as can putting on dry clothes. Moist or wet skin increases heat loss. To keep the skin dry and limit heat loss, Arctic explorers did not wash. Dressing in multiple layers gives better insulation through greater air trapping. Wearing a base layer of clothing made of materials with wicking properties helps keep the skin dry. A helpful mnemonic referring to clothing is "**COLD**": **C**lean, **O**pen with exercise to limit sweating, **L**oose layers to retain heat, and **D**ry to limit conductive heat loss.

Donning an extra layer, exercising, or getting into a warm shelter require conscious decisions that may be impaired by alcohol and other drugs as well as by mental illness. Alcohol offers no protection and increases heat loss. Some drugs inhibit shivering. Underlying conditions that predispose to cold injury include malnutrition, poverty, stress, and exertion. Individuals with peripheral neuropathy may lack nociception and not vasoconstrict appropriately.

The type and duration of cold contact determines the extent of injury and is compounded by wind chill. Exposed head and neck areas can suffer high heat loss, as these areas do not vasoconstrict effectively. Fatigue and apathy increase the risk of cold injury. A dissociative state resulting from cold can limit self-help. In cold injury situations those who maintain cognitive abilities must help those who have become limited.

Nonfreezing Cold Injuries

Nonfreezing cold injuries include pernio and trench foot (immersion foot). Frostbite represents localized freezing, while systemic injury is termed "hypothermia."

Pernio

Pernio, a prolonged chilblain, can result from water and winter activities following consecutive days of wearing wet boot liners and socks and having continually wet feet. Cold and moisture produce swelling and tenderness, but after prolonged exposure, usually 12 hours or more, deep pain and disabling tissue sensitivity as well as eschar formation can result. These changes follow sympathetic nerve instability and vascular hypersensitivity to cold that produces a vasculitis. Prevention depends on maintaining awareness, keeping the feet dry, changing socks, and checking the feet daily. Treatment includes drying and massage as well as active gentle warming and elevation. Chronic sequelae can occur.

Trench Foot

Trench foot has its origins in the military where troops stood for days in water-filled trenches. Today it can be seen in adventure travelers who become cold, wet, exhausted, and dehydrated and are unable to care for themselves, who go to bed with wet feet, or who continue to put on wet socks and tight boots day after day, leading to peripheral vasoconstriction and tissue ischemia. It can happen in warm water as well as cold and is seen in homeless populations. There is a pre-hyperemic phase followed by a hyperemic period lasting weeks with considerable pain and deep aching during which time victims cannot tolerate even light pressure on their feet. Vascular injury produces very reactive blood vessels, so the limb can easily blanch on elevation and become deep purple-red when lowered, manifesting a peripheral vasoneuropathy. Initial management is the same as for pernio, but pain relief is often problematic.

Freezing Injuries

Freezing injuries when mild are termed "frostnip," and when severe, "frostbite." They are relatively common in very cold environments as well as in major cities during the winter, including cities in the United States and Canada. With dogged persistence in cold, high, and hazardous environments, severe freezing injuries are a predictable risk among adventure travelers. Often the victims lack proper equipment but persist in the circumstances rather than retreat. Urban freezing injuries can result from poverty and the inability to protect

oneself from the environment, which can occur because of equipment breakdown or from a lack of community responses.

When onset of frostbite is considered, giving a dose of aspirin or ibuprofen to enhance circulation has been suggested. Or the foot or hand may be placed in a companion's armpit or groin for 10 minutes. At altitude give oxygen if available. Prevent hypothermia. Treatment for frostbite can be delayed until the victim has been evacuated and will no longer need the affected extremity for rescue efforts. Once a foot has become frozen, using it until it can be actively rewarmed is preferable to the possibility of warming attempts followed by refreezing. Fingers are more problematic, as they become useless with frostbite. Rapid thawing for all frostbite injuries is by immersion in water between 40 and 42° C. The actual temperature is crucial for maximal tissue survival. Aggressive pain control is required. Topical treatment to blisters is with aloe vera, but whether or not to debride is controversial. Subsequent treatment strategies over the last 40 years have focused on expectant observation as viable tissue becomes demarcated. Recent strategies have attempted to use systemic and topical agents, as in burn care, and contemporary methods such as intravenous radioisotope scanning, angiography, duplex imaging, or digital plethysmography to assess tissue viability before various medical interventions such as thrombolysis or amputation are considered.

Hypothermia

Hypothermia results when there is decreased heat production, increased heat loss, or impaired thermoregulation. Thermogenesis is decreased in the very young and the elderly. Older individuals tend to have impaired heat conservation. Ataxia or subtle signs in early stages of hypothermia may be missed by companions.

Diagnosis of hypothermia may be difficult in the environmental conditions encountered during travel because of survival exigencies. Hypothermia reflects body heat loss with a core body temperature of <35° C. Moderate hypothermia occurs below 32° C and is severe below 28° C. Shivering is lost in moderate hypothermia, which is accompanied by hypoventilation, hypotension, bradycardia, and hemoconcentration. Moderate hypothermia requires active external and non-invasive rewarming.

Severe hypothermia presents with pulseless apnea. The victim may appear lifeless, but the advanced cardiac life support adage that "you are not dead until you are warm and dead" applies, unless it can be determined that death occurred before cooling, such as by asphyxia or lethal injury. Gentle handling of the comatose victim is required because of sensitivity to the development of cardiac arrhythmias. The task is to rescue, examine, insulate, and transport, using whatever skills and facilities are available. There is no standard approach. No predictive scale, such as the Glasgow Coma Scale, has been validated, and no controlled studies exist. Positive predictors of survival include rapid cooling rate, presence of ventricular fibrillation during cardiac arrest, and narcotic or ethanol intoxication. The greatest risks to survival may be asphyxia, slow rate of cooling, invasive rewarming, asystole, and development of acute respiratory distress syndrome.

Guiding principles for initial treatment of hypothermia in the field include gently removing clothing and drying the patient, while keeping him or her horizontal to minimize orthostatic hypotension from autonomic dysfunction. Avoid exertion, which could result in a further decline in core temperature (afterdrop), and do not massage the extremities, which suppresses shivering and increases skin vasodilatation, which also can enhance core temperature afterdrop. Mildly hypothermic conscious victims can be given warm sweet fluids to drink. Stabilize injuries, administer warmed intravenous fluids if possible, attempt active rewarming with heated inhalation, and promote truncal warming via hot water bottles applied to the axillae and groin. Oxygenation is critical. Insulate with whatever can be improvised. Conditions in remote circumstances may require prolonged rescue efforts where little actual rewarming may take place, but further heat loss should be thwarted.

Once in a clinical setting, external rewarming with radiant heat, hot water bottles, heating pads and blankets, forced hot air, and warm water immersion can be used. Core rewarming with heated inhaled gases, heated IV infusions, and lavage of internal cavities with warm

fluids can be considered, as can the use of extracorporeal circulation. Active methods can be used in the elderly in whom mild hypothermia does not respond to passive efforts. The rate of active rewarming is debatable, but hourly rates exceeding 0.5° C do not seem to increase mortality. Efforts to treat cardiac arrest may not be successful until considerable rewarming has occurred. Rapid cooling of young victims, such as in sudden cold drowning, can be followed by full recovery without long-term sequelae.

HEAT ILLNESS

The human body is only about 25% efficient, meaning most of our metabolic energy production is added to the body as heat. Despite being better able to tolerate heat than cold, humans continue to die in considerable numbers during heat waves around the world. In the adult, basal heat production is about 60-70 kcal/h; excitement, fear, certain drugs, catecholamines, and thyroid hormones can increase heat production moderately, and physical work can produce up to 1000 kcal/h of thermal energy.

To diffuse excess heat, skin vasodilates and blood shifts from the splanchnic circulation to the skin. Sweat forms, then skin cools as the sweat vaporizes, thus keeping a temperature gradient between heated blood and the skin and promoting heat loss. If evaporation cannot occur because the air surrounding the skin is already saturated, sweat continues to form, but heat loss is curtailed. In these situations, extreme heat production (i.e., work) must be limited.

Children are more at risk of developing heat illness than adults because they are less efficient thermoregulators: they have a lower rate of sweating and a higher set point at which sweating starts. Children produce more metabolic heat per unit weight for a given workload and have a comparatively lower cardiac output than adults. Their larger surface area in proportion to body weight can result in greater heat absorption from the environment. Furthermore, they acclimatize more slowly than adults, and so need to reduce the intensity of activities in a hot environment for a longer period.

Serious heat illness is preventable. Chronic stress, fear, and low socioeconomic status can predispose to heat illness, as was well demonstrated in the 1995 heat wave in Chicago, with the greatest risk among African-American inhabitants residing in city slums, who lacked access to transportation and were socially isolated. Hypertension was the commonest co-morbidity in a study of survivors who suffered substantial mortality within a year. Hispanics were less affected because of their social cohesion. During the 1980 heat wave in St. Louis and Kansas City, the rates of those affected were six times higher in the lowest socioeconomic status compared with the highest socioeconomic status. Poorer people can be expected to have more complications from the stress of heat illness, including more opportunistic infections and prolonged recovery. Climate change is likely to increase exposure to heat illness, especially for the poor.

Organizers of events taking place in hot weather have a responsibility to prevent heat illness. Plenty of hydration stations, with trained spotters for potential victims, must be provided. A response system to deal with potential problems must be set up. It is appropriate to cancel events when there is a significant risk of serious heat illness from environmental factors, such as when the wet-bulb globe temperature (WBGT) is above 25° C. Runners with a fast finishing pace are especially at risk. There are numerous anecdotes of world-class athletes, as well as high school football players, who have died as a result of heat illness.

Communities will need to mobilize for the coming heat waves that are predicted as a part of global warming. The challenge is to recognize increased risk for those living in the relative poverty and social isolation conditions that often co-exist. Often those who died did not seek help or were incapable of following advice that was offered. During the heat wave in France in 2003, the highest mortality risk was in cities, especially in Paris among isolated elderly living in top-story single rooms. That air conditioners are not commonly used in France added to the impact of this tragedy. During heat waves, consider promoting visits to air-conditioned malls for the isolated, relatively poor individuals at highest risk. Providing air-conditioned shelters and lowering energy costs during hot periods are other

measures to consider as public health efforts. The Occupational Health and Safety Administration has a Heat Safety Tool app for smartphones to monitor risk levels.

Acclimatization

Acclimatization is the process whereby the body adapts to a hot environment. With repeated exposures to heat stress, for any given work load, the cardiac output increases; there is expansion of circulating blood volume; and metabolic adaptations occur in skeletal muscle. Sweat production increases, and to conserve blood volume, the sodium concentration in sweat decreases. As a result, there is less heat production for a given amount of work, but increased sweating increases the tendency to dehydration. Each liter of water lost through sweating raises rectal temperature 0.3° C, speeds heart rate 8 beats/min, and decreases cardiac output 1 L/min.

Acclimatization does not occur without work or exercise that elevates body temperature; the cardiovascular system must be capable of responding to these increased demands. A gradual increase in the time and intensity of physical exertion over 8-10 days is advised for those who will be active in the heat. It takes 2 weeks or more for maximal acclimatization.

Those in excellent physical condition are better able to tolerate the heat. The obese are at an increased risk of heat illness. Those with a febrile or gastrointestinal illness are at greater risk. The drugs described under heat stroke predispose to heat illness. Motor vehicle driver vigilance and other measures of performance are diminished under heat stress.

The best indication of the heat-acclimatized state is the capability to sweat profusely during heat stress. Prudent advice is to wear clothing that is lightweight and loose fitting, with only one layer of absorbent material. As much skin as possible should be exposed to facilitate sweat evaporation.

Water consumption should increase, since hypohydration is a major contributor to difficulty adapting to heat stress. Voluntary drinking replaces about two-thirds of the body water lost as sweat. Individuals commonly dehydrate 2–6% of their body weight during hot-weather exercise, even when fluids are available. Drinking on schedule makes better sense: 1 L 2 hours before, a ½ L 15 minutes before, and 250 mL every 15 minutes during the practice or game. Weight and urine should be monitored; urine should be copious, clear, and pale. Sports drinks containing electrolytes and carbohydrates offer no advantage over cold water in maintaining plasma volume or electrolyte concentrations during exercise. Carbohydrate solutions may offer an advantage in endurance activities. Alcohol and caffeine dehydrate.

Acclimatization to heat will be less efficient in those with lower socioeconomic position, so such people and communities will be more vulnerable to heat illness, as has been well demonstrated in the United States. There is a moral hazard in not looking out for the common good, as was evidenced in heat waves and other disasters.

Environmental Heat Illness

Heat illness is a continuum from subtle impairment in performance or heat stress to lethal heat stroke. Specific entities are described in this section.

Heat Syncope

Heat syncope includes orthostatic symptoms or fainting occurring in a person who has not undergone heat acclimatization and who is exposed to a high environmental temperature. It results because not enough salt and water have been retained and is more common in those with heart disease and those taking diuretics. The risk of heat syncope disappears as acclimatization occurs.

Heat Edema

Heat edema is seen during acclimatization and is caused by salt and water retention resulting from aldosterone production. It is seen more often in women; salt supplementation can be a precipitating factor. Heat edema vanishes as one is fully acclimatized.

Heat Tetany

Heat tetany results from hyperventilation on exposure to hot air, leading to respiratory alkalosis, paresthesias, and occasionally frank tetany.

Heat Cramps

Heat cramps are painful muscle contractions occurring in workers or athletes; they are associated with hyponatremia caused by fluid replacement of profuse sweat with free water but not salt. Typically, the victims are acclimatized, exercising, and requiring copious sweat production to control temperature. The muscles involved are those being exercised, and symptoms tend to occur toward the end of the activity. Cramps last a few minutes and disappear spontaneously. A hot environment for the exercise is not mandatory. Salt replacement is important at the first sign of premonitory muscle twitching.

Heat Exhaustion

Heat exhaustion results from body water loss and electrolyte depletion. It is common during exercise and work during heat waves but can occur as a result of heavy sweating while undergoing intense exercise in temperate climates. Typically, postural hypotension develops immediately on termination of exercise in the heat by unacclimatized persons. Although there is a continuum, patients tend to suffer from one of two categories: water depletion or salt depletion.

Early warning signs include a flushed face, hyperventilation, headache, dizziness, nausea, arm tingling, piloerection, paradoxical chilliness, incoordination, and confusion. Pain may not be present and there may be euphoria.

The water-depletion variety can occur in the very young or very old who experience dehydration but cannot act on their thirst to replenish their water losses. Soldiers, laborers in the desert, boiler-room workers, and athletes who ingest salt without adequate water are also commonly afflicted. Symptoms include thirst, fatigue, dysphoria, and impaired judgment. Examination shows dehydration and an elevated body temperature. Left untreated, heat stroke can result as the body temperature rises.

The salt-depletion variety is seen in those who replace fluid losses with water without salt; they do not experience thirst as a predominant symptom. Unlike heat cramps, salt depletion heat exhaustion tends to occur in unacclimatized individuals who exhibit systemic symptoms. Symptoms can include myalgias, nausea, vomiting, and diarrhea, as well as weakness, fatigue, and headache. Hypotension and tachycardia are seen; the body temperature is not elevated unless dehydration results from the vomiting. Replacement of sodium chloride results in rapid improvement.

Confusion is an early sign of heat injury. Delirium has been reported. Commonly, individuals with heat exhaustion suffer from both water and salt depletion; they exhibit a variety of symptoms and are often misdiagnosed as having a viral syndrome. Giving aspirin to exercising patients who develop heat exhaustion can paradoxically increase body temperature.

Treatment of Heat Exhaustion

When treating heat exhaustion, if hypernatremia and scant concentrated urine are present, one should assume water deficiency, calculate the water deficit, and treat. Water should be given orally if the patient is conscious and not vomiting. If shock is present, the patient should first be treated with plasma-expanding fluids; otherwise, replace volume with 5% dextrose intravenously (IV) until the serum sodium has fallen to near normal, and then add hypotonic saline to the infusion. Half the water deficit should be replaced over 3-6 hours. In children, give half normal saline initially until urine output is established, then decrease to quarter normal saline along with 5% dextrose. Hydration and electrolyte balance should be corrected to normal over at least 48 hours in children to avoid seizures. Severe prolonged central nervous system (CNS) symptoms may take days to resolve. Those presenting with primary salt depletion respond quickly to salty oral fluids or normal saline IV if they cannot drink.

Heat Stroke

Heat stroke results from a failure of the thermoregulatory mechanisms to meet heat stress; extreme elevations of body temperature occur, as well as end-organ dysfunction and damage. Risk factors in healthy individuals include environmental extremes (e.g., Hajj in Saudi Arabia, where up to 1000 cases per day may occur with a mortality rate around 50%), salt and water depletion, infection, fever after immunization, lack of acclimatization, obesity, fatigue, and consumption of drugs that suppress sweating (e.g., anticholinergics, drugs for Parkinson disease, phenothiazines, and antihistamines). Other drugs known to predispose to heat stroke include diuretics, which cause salt and water depletion; tricyclics, which increase heat production; butyrophenones, which disturb hypothalamic regulation of temperature and the ability to recognize thirst; and sympathomimetics, which increase psychomotor activity.

Health conditions that are risk factors for the development of heat stroke include those compromising cardiovascular function, diabetes mellitus, hyperthyroidism, potassium deficiency, and alcoholism. Other conditions that result in impaired sweat production are also associated with heat stroke and include prickly heat (miliaria), healed extensive thermal burns, scleroderma, and congestive heart failure.

Relative poverty, advanced age, and lacking an air conditioner or the resources to run it have been found to be major risk factors in US cities during heat waves.

Classic Heat Stroke

Classic heat stroke occurs in sedentary individuals exposed to several days of environmental heat stress in whom thermoregulatory mechanisms stop functioning. It can occur at lower temperatures if the relative humidity is high. The skin is hot, dry, and flushed. Hyperthermia is invariably present, with a body temperature above 40.6° C (105° F), with some CNS disturbance present. Confusion may be the earliest sign, with an inability of victims to recognize their own illness. Eventually, coma may ensue. A respiratory alkalosis is usually present. Pre-existing organic disease increases mortality.

Exertional Heat Stroke

Exertional heat stroke is found in males performing heavy muscular exercise on warm, humid days. In the United States, it is seen in competitive long-distance runners, football players (>75 fatalities per year in the United States among amateurs), and military recruits. Victims are volume depleted and exhibit neurologic symptoms such as strange behavior, confusion, or even coma. Relative bradycardia may be seen in highly conditioned athletes with this disorder. Sweating persists in more than half the cases. These individuals present with a relatively cool, clammy skin, but with an extremely elevated core temperature (up to 44.5° C). However, by the time the individual is seen, his temperature may have dropped to the more typical febrile range but with all the other complications present as a result of widespread organ damage: rhabdomyolysis disseminated intravascular coagulation, lactic acidosis, hyperuricemia, and hypokalemia. A moderate to severe metabolic acidosis is seen. Few cases are seen in women. Patients who recover from heat stroke are more susceptible to recurrent attacks.

Differential Diagnosis of Heat Stroke in Travelers

Be sure to check temperatures of potential heat illness victims when they are first evaluated in the field. Infections, including cerebral falciparum malaria, can occur in environments similar to those where heat stroke occurs. Encephalitis, meningitis, and typhoid fever can present with a picture similar to heat stroke. Shaking chills suggest that fever is due to an infectious etiology. Drug-induced heat illness, especially anticholinergic poisoning, may be difficult to diagnose. Cocaine, methamphetamine, and other sympathomimetic agents may be involved. Sweating suggests that anticholinergic poisoning is not present. Over half of heat stroke patients have constricted, pinpoint pupils, which also militate against anticholinergic poisoning.

Treatment of Heat Stroke

Treatment of heat stroke is a medical emergency, and delay in cooling is the single most important factor leading to death. Begin cooling at the site of collapse by disrobing, fanning, and bathing the skin with cool water. Monitor rectal temperature every 5-10 min during resuscitation. The duration of hyperthermia is the most important factor affecting survival. Reasons for delay include failure to make the diagnosis and lack of facilities to rapidly cool. Mortality is still high, at close to 10%.

Cool the Patient

The victim should be removed from the hot environment, and aggressive cooling techniques improvised depending on what is available. The classic treatment is to immerse the patient in a tub of ice water and massage the skin briskly. Although cold-induced vasoconstriction can theoretically impair heat loss, this method has been used successfully. No other technique yet proposed claims better results. Tepid or tap water may be as good as ice water for cooling and may be more comfortable. Alternatively, the patient can be wetted down with water and rubbed briskly with ice bags, keeping a large fan blowing on the patient, which may be better tolerated than ice-water immersion in confused individuals. If the ice supply is limited, it should be applied to the head, neck, abdomen, axillae, and groin. Special beds (physiologic body-cooling unit or the King Saud University cooling bed) have been constructed to allow the spraying of pressurized water at 15° C on the nude body; they were designed for use in countries where heat stroke occurs to many on pilgrimage. In evacuation, consider an open vehicle and mist sprays. No pharmacologic agent has been shown to help, including dantrolene, although antipyretic agents have not been evaluated.

Support the Vital Signs

A heat stroke patient should be transported to a medical facility with rapid correction of fluid and electrolyte abnormalities; hypoglycemia, if present, should be treated. Diazepam (IV) should be given for seizures, severe cramping, or shivering, which impair cooling. Consider gastric, rectal, thoracic, bladder, or peritoneal lavage cooling. Central venous monitoring can guide fluid therapy and avoid fluid overload. If the victim is comatose, endotracheal intubation is recommended.

Prevention of Heat Stroke

Heat stroke can be prevented by encouraging people to drink enough fluids (especially important for children, elderly people, and those with debilitating conditions), adjusting dosages of drugs affecting fluid balance and thermoregulation in travelers to warm climates, and limiting activity during heat extremes. Exercise in the heat should be commensurate with fitness level, with the hottest hours of the day avoided and rest periods taken. Those engaged in strenuous activity during hot weather need to drink more frequently and copiously than thirst dictates. Consuming 400-500 mL of water before exertion, and then a cup or more (300 mL) of water for every 20 min of exercise in the heat should be encouraged. Splashing or spraying the skin with water intermittently can promote evaporative cooling, and access to air-conditioned spaces is essential. Salt tablet consumption is not routinely recommended.

If someone feels ill or "overstressed" during exercise, he or she should stop and seek a cool area. This is especially important for driven individuals in peer-group situations. After a febrile illness, individuals should be especially cautious of further heat exposure.

FURTHER READING

Jet lag: an app for light exposure time to help with jet lag: <http://entrain.math.lsa.umich.edu>

Heat illness: OHSA website on heat illness in outdoor workers where you can download a Heat Safety Tool app: <https://www.osha.gov/SLTC/heatillness/index.html>

Motion sickness: motion sickness site with much useful information: <http://www.dizziness-and-balance.com/disorders/central/motion.htm>

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