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Welcome to the second issue of a magazine that is intended to both educate and entertain the many millions of Americans who suffer from migraine. As a physician and fellow migraineur who has treated thousands of migraine patients, conducted research in the field and assisted in the development of virtually every new treatment for headache since the emergence of sumatriptan (Imitrex) in the late '80s, I have a particular allegiance to our readership.

Migraine is a decidedly odd malady. Although rarely life-threatening, it is frequently life-altering. Migraine imposes a tremendous physical, financial and psychosocial burden upon our society, and for those of us afflicted it may persist, throughout most of our lives.

While common, the disorder is poorly understood both by the public and by healthcare providers, a situation that results all too often in a failure to seek medical care or medical mismanagement and consequent patient frustration when such care is sought.

In this and subsequent issues we will attempt to dispel the many myths that surround migraine, acquaint you with the tremendous strides that have been made in understanding and treating the disorder and, most important, offer some guidance as to how we migraineurs may enjoy life more fully and control our headache disorder more effectively.

John F. Rothrock, MD
Editor in Chief

Cynthia Andress pauses on the Shirley Lake Trail. She is a mother, a full time student, an active triathlete and a migraineur.

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SO YOU WANT TO TAKE YOUR MIGRAINE... ON VACATION!

Follow this advice and you will increase your chances of enjoying a pleasant and headache-free vacation.

Speaking to her physician, LM, a 35-year-old female migraineur, reports:

“We live in Phoenix, and our summers extend well into October. This Fall my husband and I are planning to take two weeks off in September to go to Mallorca, but instead of looking forward to our vacation I’m dreading it. We went to Paris a few years ago for what was supposed to be a second honeymoon, and I just fell apart. I was exhausted the whole time, but I couldn’t sleep. Despite the great food everywhere around me, I had no appetite. And my migraines went ballistic! To my husband’s disgust, I wound up spending most of the so-called vacation in bed (alone) trying to deal with my headache and wishing I was home. He barely spoke to me on the flight back to Arizona.

We’ve been saving up all year for this big trip to Spain, but because of my migraine I’m afraid it’s just going to be a big waste of money.”

Extended travel can exact a physical toll on anyone, but long flights and a sudden introduction to an unfamiliar environment may make the experience particularly challenging for the migraineur. No sane migraine sufferer wants to experience an extended flare-up of headache, and that such misery could occur in the midst of a long-anticipated vacation is, to say the least, discouraging.

To make matters worse, the acutely suffering migraineur often must bear an additional burden: the disappointment experienced by one’s traveling companion. Especially if they have no personal experience with migraine, even the most compassionate spouse, significant other, relative or friend may find it difficult to deal gracefully with the unappealing behavior provoked by your acute migraine. After all, you don’t have a fever. You don’t look that sick. Why can’t you go sailing; walk with me on the beach; stop for a poolside cocktail; have a romantic tryst in our room before dinner at the restaurant our friends told us about? In short, why can’t you do all those things we spent so much time planning to enjoy? Why are you ruining our vacation!

One needn’t travel thousands of miles to the Balearic islands of Spain; even far less ambitious vacations have the potential to unhinge migraine. The alterations in one’s usual eating, drinking and sleeping habits can wreak havoc, leading the poor migraineur to wonder why he or she didn’t opt instead to spend that hard-earned money on a bathroom renovation.

Try following the advice offered here, and you will increase your chances of enjoying a pleasant, headache-free vacation.

AIRLINE TRAVEL

Travel Stress: The circumstances of extended air travel possess a multitude of potential migraine triggers. Whatever the setting, migraineurs inevitably report stress to be the most common stimulus.
MIGRAINE TREATMENT

Medications: When it comes to your usual medications (including contraceptives), don’t get caught short while on vacation. Well before you leave, make sure you have enough of those medications to last for the time you’re away... especially whatever you normally take for acute migraine treatment. And if in the recent past you’ve had attacks of severe migraine that failed to respond to your usual self-administered therapy and required a trip to the doctor’s office or an emergency room, it’s a good idea to bring with you a written statement by your physician that briefly summarizes your medical history, your migraine history in particular and the medications that have been required to treat your migraine. Any migraineur who has suffered through the experience will tell you that to seek treatment for acute headache at an unfamiliar medical facility can be frustrating and even humiliating. To do so bearing a statement from your regular physician may go a long ways towards lessening the hassle.

JET LAG

The circadian rhythm refers to our brain’s inclination to bring various biologic functions into synchrony with the earth’s daily rotation. The misery we term “jet lag” is a symptom complex that results when this internal clock remains stubbornly set to the time back home over the days that immediately follow the brain’s sudden transport to its new destination.

How much one is affected depends in part on the biologic characteristics of the individual traveler’s brain, and this (unfortunately) is where migraine and jet lag intersect. The same genetically derived
SYNCHRONIZE YOUR BIOLOGIC CLOCK WITH YOUR DESTINATION

Sensitivity of the migrainous brain that can produce visual aura or attacks of inescapable head pain also increases the predisposition to jet lag. Migraine thrives on change, be it the monthly change in levels of sex hormones experienced by menstruating females, a change in barometric pressure...or a sudden change in time zones.

The symptoms of jet lag occur one to two days after travel across at least two time zones, and jet lag tends to be more severe with travel eastward compared with westward. The most common symptoms are insomnia (coupled with daytime sleepiness), a pervasive sense of fatigue, loss of appetite, constipation, mental "fogginess" and impaired physical performance.

Great, you say. Jet lag may be a major buzz-kill, but I really want to go to Spain, and I don't have the time to cruise slowly across the ocean to get there. What can I do?

Without any specific treatment, your circadian rhythm will adjust to your destination time at the rate of about one time zone per day for eastward travel and 1.5 time zones per day for westward travel. Along with the total distance traveled, the severity of jet lag is influenced by:

- the direction of travel (again, eastward travel is more difficult to adapt to than westward travel)
- your ability to sleep during travel

For those who are sufficiently motivated, treatment of jet lag involves accelerating one's adjustment to the new time zone with strategically timed exposure to bright light and the use of melatonin. Put simply, you are trying to help synchronize your biologic clock with that of your destination. Making this effort, along with symptomatic measures intended to help alleviate symptoms such as insomnia and daytime sleepiness, can go a long ways towards easing the transition from home to vacation.

<table>
<thead>
<tr>
<th>TIPS FOR MANAGING JET LAG</th>
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<tr>
<td><strong>Traveling Eastward</strong></td>
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<tr>
<td><strong>Before Travel</strong></td>
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<tr>
<td>• Starting 3 days before departure, move your bedtime and wake time 30 minutes earlier each day</td>
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<tr>
<td>• During this shift, avoid light in the evenings (including electronic light) and seek bright light for the 1st 2-3 hours in the mornings</td>
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<td><strong>During Travel</strong></td>
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<tr>
<td>• Set your watch to your destination's time</td>
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<td>• With watch set to destination's time, avoid early morning exposure to bright light (eg, use sunglasses if cabin lights are on; keep windows covered)</td>
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<tr>
<td>• With watch set to destination's time, maximize late morning and early afternoon exposure to bright light</td>
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<td>• Try to sleep during destination nighttime; if unable to sleep, wear dark glasses or sleep mask to reduce light exposure (especially during the second half of the night)</td>
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<td>• Do not use sedative medication during your flight</td>
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<tr>
<td><strong>Upon Arrival</strong></td>
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<tr>
<td>• Avoid early morning bright light</td>
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<tr>
<td>• Get lots of late morning and early afternoon bright light</td>
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<tr>
<td>• On day of arrival and for up to 5 days thereafter, take melatonin 3 milligrams at desired destination bedtime</td>
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<tr>
<td>• For daytime sleepiness: short naps (&lt;45 mins) at least 8 hours before desired bedtime</td>
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| **Traveling Westward**     |
| **Before Travel**          |
| • Starting 3 days before departure, move your bedtime and wake time 30 minutes later each day |
| • During this shift, avoid bright light the first few hours after awakening and seek light in the evenings |
| **During Travel**           |
| • Set your watch to your destination's time |
| • With watch set to your destination's time, avoid bright light (including electronics) during your destination's nighttime. Use dark glasses if cabin lights are on |
| • Try to stay awake until desired destination bedtime |
| • Try to sleep during destination nighttime; if unable to sleep, wear dark glasses or sleep mask to minimize light exposure |
| • Do not use sedative medication during your flight |
| **Upon Arrival**            |
| • Get lots of late afternoon and evening bright light |
| • Avoid exposure to bright lights during your destination's nighttime |
| • Melatonin is not likely to be helpful |
| • Try to stay up to desired destination bedtime. Avoid nodding off early in the evening |
| • Staying asleep may be difficult for the first few days; avoid electronics during nighttime awakenings |
| • For daytime sleepiness: short naps (<45 mins) at least 8 hours before desired bedtime |
WHAT IS MIGRAINE?

Migraine actively afflicts over 10% of the general population-surprisingly few can offer an accurate answer to the simple question: What is migraine?

Although migraine actively afflicts approximately 38 million Americans—well over 10% of the general population—and every day is the subject of countless conversations, publications, web sites, advertisements and jokes, surprisingly few people (including medical providers) can offer an accurate answer to the simple question: What is migraine?

The term “migraine” is easy enough to define. If you’ve had 5 or more attacks of unprovoked headache (not the headache of a tequila hangover) that lasted 4 to 72 hours, was severe enough to inhibit or even prohibit your routine daily activities, was accompanied by nausea or light/sound sensitivity and could not be attributed to another medical disorder…you are a migraineur.

The headache of migraine is not always severe, throbbing or one-sided

Note that this definition does not require the headache to be throbbing or lateralized to one side of the head. Although such clinical features are common in migraine, they are far from invariable. Plenty of migraineurs have headache pain that is “all over the head”, constant/non-throbbing or both.

Nor does the diagnosis of migraine require the occurrence of aura symptoms (eg, visual “stars”, “flashes”, “zig-zags” or blind spots). Only 20-25% of migraineurs ever experience aura, and in that subgroup there are relatively few who experience aura with each and every migraine attack.

Finally, the headache of migraine is not always severe, and some migraine attacks may involve no headache whatsoever (eg, migraineous aura without headache). With many episodes of migraine the headache may be mild in intensity, lack any associated nausea or light/sound sensitivity and symptomatically resemble tension-type headache more than what we usually think of as migraine.

This tendency to characterize only severe headaches as “migraine” can complicate the medical provider’s attempt to accurately determine a migraine patient’s total headache burden, the key to developing an appropriate treatment strategy. When questioned regarding their headache frequency, migraine patients often base their estimate only on those headaches that are severe and incapacitating. In doing so they may fail to include headaches that are non-disabling but nonetheless decrease work productivity and quality of life. Reluctant to overstate the impact migraine is having on their lives, patients discount those days when they manage to “carry on” despite a headache…but can neither work at full-speed nor take any real pleasure in a social event they otherwise would enjoy.

Again, when migraine patients underestimate their headache burden, this works against the provider’s effort to provide them with an effective management plan. However severe the headaches involved, a migraine patient who is experiencing only 3 “headache days” per month requires a very different treatment approach than the patient who not only averages 3 days of incapacitating headache each month but also is experiencing daily or near daily headache pain of mild to moderate intensity.

WHAT CAUSES MIGRAINE?

For many years it was believed that migraine attacks arose from changes in the blood vessels which supply the head and brain. Aura (when it occurred) was attributed to constriction of arteries, with the neurologic symptoms of aura reflecting decreased blood flow to retinal or brain tissue. The throbbing, sickening pain of migraine in turn was attributed to a compensatory dilation of those and other vessels.
We now believe that migraine is genetic in origin and that the disorder represents a genetically-induced hypersensitivity involving neurons (brain cells) located within the central nervous system. If a genetically primed neuron is triggered by a change in the external environment (e.g., a sudden drop in barometric pressure) or internal environment (e.g., a sudden drop in estrogen level), that neuron may depolarize (discharge electrically) and, by triggering its neighboring neurons to join in, induce the pathways in the brain that normally conduct head pain to awaken and produce the familiar symptoms of a migraine attack.

The biologic circuitry of migraine is illustrated below. Under normal conditions, a painful stimulus produced by, say, trauma or meningitis, activates head pain receptors located on blood vessels (A) within a membrane (the dura) that lines the brain. Those receptors generate a pain signal that is transmitted by the trigeminal nerve (B) to the trigeminal nucleus caudalis (C), a cluster of neurons located within the brain stem. The trigeminal nucleus caudalis (TNC), acting as a relay station, passes the pain signal upward to the brain itself, and at that point there is conscious awareness of headache.

In migraine, the normal flow of head pain signaling is reversed. In response to a change in the internal or external environment, genetically hypersensitive neurons located in the visual cortex of the brain (1) fire off electrically, generating a pain signal that travels brain-to-dura, against the normal flow of head pain conduction: downward to the TNC in the brain stem and out the trigeminal nerve to the blood vessels within the dura. When the signal reaches the junction between the nerve and blood vessel, it stimulates the release of proteins (2) such as CGRP (calcitonin gene related peptide) that in turn cause the vessel to dilate and to leak other proteins that promote inflammation. That inflammatory response further stimulates the already sensitized trigeminal nerve endings, producing another pain signal that bounces back (B) to the brain in the normal direction of sensory flow (dura>brain).

Thus a migraine attack can be thought of as a physiologic “ping pong match”, with pain signal flowing simultaneously in opposite directions, “inside>out” (brain>dura) and “outside>in” (dura>brain). Each signal reinforces the other, with the signal amplifying as the transmission pathway becomes increasingly sensitized. The individual suffering the acute migraine attack correspondingly experiences a progressively more severe headache. This biologic and clinical process will persist until the underlying pain signaling system spontaneously becomes inactive or the afflicted individual takes action to terminate the attack (e.g., goes to sleep or takes a medication such as a triptan).

If this system of migrainous head pain transmission becomes chronically sensitized, the migraineur will begin to experience more frequent episodes of head pain. In the worse case scenario, episodic migraine may “transform” into chronic migraine, and the migraineur may suffer a constant headache of relatively low intensity with superimposed attacks of more severe head pain. In such cases a course of preventive (prophylactic) therapy may be required to stabilize the transmission system (see How can migraine be treated? later in this article).

In their physiologic origins and their treatment, migraine and epilepsy are biologic 1st cousins. Both conditions involve brains that contain abnormally sensitive neurons, and in both the source of this sensitivity may be genetic. Migraine and epilepsy are “bi-directionally co-morbid” (i.e., if one has migraine, he or she is more likely to have epilepsy than normally would be expected…and vice versa). Further cementing this relationship is the fact that several of our best medications for migraine prevention were first developed to treat epilepsy (e.g., divalproex sodium/Depakote and topiramate/Topamax, Trokendi).

In short, while changes in the caliber and permeability of cranial blood vessels may play an important secondary role in generating migrainous symptoms, migraine is a primary brain disorder.
HOW TO TREAT MIGRAINE

Given what we now know about its cause, effective treatment of migraine must necessarily involve stabilization of this genetically primed brain and nervous system pathway for head pain transmission that has become acutely or chronically sensitized.

When we speak of acute (or symptomatic) migraine treatment, we are referring to measures one may take during the time of a headache that are intended to terminate that headache and associated symptoms such as nausea and light sensitivity.

Preventive (or prophylactic) treatment refers to measures used on a chronic basis to reduce headache burden.

Reducing chronic stress, good sleep hygiene and avoidance of obvious migraine triggers may do as much as any prescription therapy to reduce migraine attack frequency.

There are many ways to skin the migraine cat, both for acute/symptomatic treatment and for chronic/prophylactic therapy. While non-prescription (OTC=“over the counter”) and prescription medications are often of great value in controlling migraine, there are other ways to treat the disorder that do not involve administering a pill, nasal spray or injection. Regular aerobic exercise, other measures known to reduce chronic stress (egs, yoga, meditation), good sleep hygiene and avoidance of obvious migraine triggers may do much as any prescription therapy to reduce migraine attack frequency and overall headache burden. Especially when utilized early, aerobic exercise, application of heat or cold to the head and neck areas, drinking a caffeinated beverage or just briefly taking a break and relaxing may terminate an acute attack.

ACUTE MIGRAINE TREATMENT

If medication is used to treat acute migraine headache, several important caveats should be considered.

- **Match medication(s) to headache intensity**
  For example, oral triptans generally work best for headaches of mild to moderate intensity, but injectable sumatriptan is more effective for “rescue” from a severe migrainous headache. You probably need to keep multiple pharmacologic “weapons” on hand to treat the varying intensities of migraine headache you experience.

- **Treat early**
  2-3 aspirin taken with a caffeinated beverage early in an attack may be more effective than a narcotic taken when the headache has become well-established and severe.

- **Administer an adequate dose**
  For example, OTC ibuprofen is available in a 200 milligram (mg) strength and naproxen sodium in a 220 mg strength. Both drugs can be quite effective for early treatment of acute migraine headache, but “migraine doses” are generally 600-800 mg for ibuprofen and at least 440 mg for naproxen sodium; with either, co-administer caffeine (see below)

- **Consider the route of drug administration**
  Acute migraine attacks are accompanied by gastroparesis, meaning that the stomach's usual motility is reduced to the point that it may not pass orally administered medications that “drop in” on to the small intestine where they would otherwise be absorbed, enter the blood stream and speed their way to their intended targets to relieve your acute migraine head pain. Erratic gastrointestinal (GI) absorption of oral medications for the treatment of acute migraine may at least partially account for the therapeutic inconsistency many individuals experience with their use.

  To some extent the problem with gastroparesis/impaired GI absorption may be circumvented by administering the oral medication with a caffeinated beverage or by taking a compound medication that contains caffeine (eg, Excedrin). Some prescription medications for acute migraine (eg, Treximet) are formulated so as to speed up their exit from the stomach and subsequent absorption into the bloodstream.

  If your migraine headache is accompanied by nausea and vomiting, then an orally administered medication obviously is a loser. At such times you can resort to acute migraine medication that is administered intranasally or by subcutaneous injection.

PREVENTIVE (PROPHYLACTIC) MIGRAINE THERAPY

There are also important caveats to keep in mind if preventive therapy is prescribed to chronically stabilize the biologic migraine pathway and thus reduce headache burden.

- **Take your preventive medication as prescribed**
  Skipping doses of an orally administered medication for migraine prevention or
taking a lower (or higher) dose than prescribed may work against you, preventing any positive treatment response, encouraging side effects and even serving to increase your headache burden. If you are receiving Botox injections for suppression of chronic migraine, the intervals between treatments typically should not exceed 12 weeks; extending those intervals beyond 12 weeks may give the migraine pathways a chance to “recover” and become re-sensitized, and before you know it you’ll be back to your miserable pre-Botox baseline.

- **There’s no guarantee of success.**
  No one preventive therapy is effective for all migraineurs, and your friend’s or relative’s remarkably positive response to a given therapy does not ensure you will have the same experience. In addition, all allopathic therapies for migraine prevention have potential side effects, and one patient may enjoy a wonderful therapeutic response to a particular treatment whereas the next will suffer a bag-full of annoying side effects and absolutely no reduction in headache burden. Unfortunately, medical providers currently lack much in the way of a means to pre-dict which patient will respond well versus poorly to a particular therapy, and both acute and preventive migraine treatment thus remains a process of educated “trial and error”.

- **Continue treatment for an adequate duration**
  Don’t expect immediate success. Some oral medications for migraine prevention require a gradual upward escalation of their doses to reach what is required for an optimal therapeutic response, a process that may take weeks to accomplish. Even if such escalation is unnecessary and you get take a therapeutic dose from the get-go, it may take a month or more to determine whether the medication is going to be effective for you. Unfortunately, it’s during those first few weeks of treatment with an oral prevention therapy that side effects from the drug tend to be most prominent. It’s discouraging to be experiencing side effects but no benefit, but stick with the treatment if possible. Talk it over with your provider. Sometimes a temporary reduction in dose will help you through the rough patch. If you are receiving Botox injections for migraine prevention, remember that many patients with chronic migraine do not begin to experience any reduction in headache burden until after the second set of injections. Don’t give up after only one treatment.

- **Treat break-through headaches aggressively**
  Seldom is a migraine prevention therapy so effective that it will completely prevent all headaches from occurring. If you have an acute headache despite prevention therapy, treat that headache! Use the same strategy outlined earlier under Acute migraine treatment.

- **Don’t stick with a loser**
  If you’ve given the prevention therapy a real chance; if you’ve taken an adequate dose for an adequate duration or, in the case of Botox, if you’ve received at least two treatments separated in time by no more than 12 weeks; and if you still are stuck with a substantial headache burden that is eroding your quality of life… *do something!* Perhaps you need a higher dose. Perhaps a different dosing regimen (say, twice daily rather than at bedtime only). Perhaps a different therapy altogether. Whatever. If you’re not making progress, it’s time for a change.

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### CGRP: WHY ALL THE FUSS?

Calcitonin Gene-Related Peptide (CGRP), a protein which is the most potent naturally occurring dilator of blood vessels in the human body, plays a vital role in the circuitry which produces migraine headache.

Point-to-point communication within our nervous systems relies on electrochemical transmission: a electrical signal passes down a conducting “wire” (ie, a nerve), and when that signal reaches its target at the nerve ending, a chemical neurotransmitter is released to connect with a receptor located on the nerve’s target. In the case of migraine, the “wire” is the trigeminal nerve, the chemical neurotransmitter is CGRP and the target is a head pain receptor located on a dural blood vessel.

The CGRP antagonists are experimental large molecule monoclonal antibodies or small molecules that either block CGRP directly or block its receptor. Now under investigation for their safety and effectiveness in treating acute migraine and preventing headache in episodic or chronic migraine, they are highly selective agents that prevent closure of the migraine circuit at a key point in the pathway that generates head pain.

Five different pharmaceutical companies (Alder, Allergan, Amgen, Lilly and Teva) are racing to bring their CGRP antagonists to market for general clinical use. According to the antagonist involved, administration may be oral, subcutaneous or intravenous. The results from research conducted to date have been highly promising, and the CGRP antagonists may well represent the next great breakthrough in migraine therapeutics.
Stress, Migraine and Cannibalism in the Sierra Nevada

No matter the individual’s race, gender, ethnicity or culture, stress is the most common trigger for acute headache reported by migraineurs. Paradoxically, a sudden release from stress also may produce an acute attack of migraine (eg, the dreaded “vacation migraine”), whereas a chronic reduction in stress may result in a dramatic decline in one’s migraine burden.

In the Winter 2016-17 issue of Migraineur we presented the case of Ulysses S. Grant, military hero, 18th U.S. President and “celebrity migraineur”. In April 1865, despite having cornered Robert E. Lee’s dwindling Confederate army at Appomattox, Grant feared Lee would once again conjure up the trickery required to elude his grasp. Racked by anxiety, Grant developed an acute migraine headache that by April 8 was peaking in intensity. In his memoirs he wrote, “I was suffering very severely with a sick headache...I spent the night in bathing my feet in hot water and mustard, and putting mustard plasters on my wrists and the back part of my neck.” Despite these efforts, his “sick headache was still present on the morning of the 9th.

Then arrived a note from Lee indicating his willingness to surrender. Decades later Grant recollected that “the instant I saw the contents of the note, I was cured.”

Other “celebrity migraineurs” less well known to history than Grant have experienced this same stress/release migraine phenomenon. One particularly vivid example of the effect of stress on migraine comes from Margaret Reed, a survivor of the infamous “Donner party”.

In 1846, desiring a better life for his family and specifically concerned by the frequent, debilitating migraines suffered by his wife, James Reed used what remained to him after declaring bankruptcy to obtain the wagon, oxen and supplies required for the long emigration to California. On April 14, accompanied by eight wagons belonging to a number of other families (the large Donner clan amongst them), James, Margaret and their four children began the exodus from Illinois. Passing through Springfield on the 15th, James was unable to persuade his old and trusted friend, Abraham Lincoln, to join them.

Margaret’s migraine flared near the northeastern border of present-day Utah, as the company’s wagons lumbered down a section of trail lined by 300 Sioux warriors on horseback. Attracted by the Reed’s 13-year-old daughter, Virginia, the Sioux offered buffalo robes, “pretty beaded moccasins” and ropes made of grass in exchange for the young girl and her pony. Only after her husband tactfully managed to convey that Virginia was not available did Margaret’s “crippling migraine” subside.

Delayed by a variety of circumstances, near what is now Wyoming the large band that had traveled so far together divided into two separate groups. Fearing that they might otherwise fail to clear the Sierra Nevada range before winter’s onset, one group opted to take “Hasting’s Cutoff”, a little-used new detour that led south of Salt Lake and across the great salt desert before rejoining the standard California Trail. The other group opted for the longer but proven route that arced northward far above the Lake and then southwest into Nevada.

James Reed spoke strongly in favor of the detour and the time it would save. Ironically, those who traveled with him...
on this shorter route—those known to history as the “Donner Party”—would become trapped in the mountain snow. Many would perish.

The detour proved to be a bust. Time was passing, autumn was approaching and the Donner Party grew ever more aware of their slow progress. Nerves frayed, tempers erupted and Margaret’s migraine blossomed. In a bizarre incident along the alkaline waters of the Humboldt River, James was banished from the Party after killing a young teamster. Knowing how inadequate was the emigrants’ remaining store of provisions, he set out for Sutter’s Fort on the far side of the Sierras to seek much-needed supplies.

With James Reed now exiled, the Party continued on, and after 68 excruciating days on the Cutoff they finally rejoined the established California Trail. They rested (too long) in a large meadow near present-day Reno before moving into the mountains. On October 30 they made camp in a “pretty little valley” in the High Sierras just five miles from Truckee (now Donner) Lake. That night it snowed 8 inches.

With her mother buried by the trail back in Kansas and her husband absent and quite possibly dead, Margaret Reed fought alone to keep her children alive.

It was only the beginning. By the following afternoon the snow was axle-deep and the wagons useless. For eight consecutive days the snow continued to fall, and by the time the weather briefly cleared it was obvious to all that the pass was closed. They would be waiting out the winter by the frozen lake. Without rescue, starvation seemed a certainty.

The weeks passed. With her mother buried by the trail back in Kansas and her husband absent and quite possibly dead, Margaret Reed fought alone to keep her children alive. Despite her growing weakness and incessant migraine, she purchased four oxen from the other emigrants and slaughtered them in the snow. When that meat was gone, she deftly slit the throat of the family’s beloved pet dog and wept as she cut up his body. Determined that her children not starve, on January 4 she set out to cross the mountains, organize a relief party and bring back food. After 5 days and 4 nights in the snow and freezing cold she was forced to turn back. Even in her desperation, however, Margaret Reed did not resort to cannibalization. While written accounts of the emigrants’ camp at the lake describe “hair, bones, skulls and the fragments of half-consumed limbs”, children with blood-stained faces devouring human liver and heart, and human flesh boiling in a large iron kettle, the Reeds are considered to be the only family of the Donner Party not to have consumed other humans.

And yet all the Reeds survived. Margaret and James were reunited on February 28, as a rescue party heading westward with the wife in tow met an eastward-bound rescue party led by the husband. On March 1 Margaret suffered an especially severe migraine attack, and the relief party bearing her consequently stopped for a day of rest. After so much stress for so long, the sudden release must have been overwhelming.

Of the 87 members of the Donner Party, 41 perished. Of the 35 who died in the camp by the lake or attempting to cross the mountains, 18 were cannibalized. As for the Reeds, James Reed subsequently made his fortune and eventually became one of California’s leading citizens. His wife, Margaret, is said to have led a peaceful life. Peaceful…and migraine-free.
MANAGING YOUR MIGRAINE

Here is information that may help you on your journey towards “headache free or nearly so”

1 Migraine Tip of the Month: Beverage Awareness

If you enjoy an occasional glass of wine, beer or cocktail and have found that ingestion of an alcoholic beverage frequently will precipitate a migraine attack...well, I'm sorry. Having this tendency myself, I can empathize.

Short of absolute sobriety what can one do to overcome this unfortunate stimulus>response relationship?

First, recognize that as a general rule it is the aromatic alcoholic beverages that possess the greatest potential for triggering a migraine attack. This is unfortunate. If you are a particular fan of red wine, aged scotch or bourbon, liqueurs/aperitifs (egs, amaretto, ouzo) or dark beer: watch out! Champagne, too, can be a killer. For better or worse, the odorless or near-odorless alcoholic beverages tend to be less potent triggers. There is an old saying amongst those who seem to know: “Vodka is the drink of migraineurs.” Whatever alcoholic beverage you choose to drink, be sure to chase it with copious amounts of water. This will help you avoid both a migraine and the embarrassing tendency to become inebriated after even a modest amount of alcohol intake (another unfortunate fact of life for many migraineurs).

Finally, as if the “alcohol as migraine trigger” issue weren't bad enough, remember that migraineurs tend to be more prone to hangovers than those without migraine who drink an equivalent amount of alcohol. Take pains to hydrate well both while you are imbibing your alcoholic drink of choice and during the hours afterwards.

THE FIRST STEP OF YOUR JOURNEY BEGINS HERE

2 Migraine Treatment of the Month: Caffeine!

Chronic over-consumption of caffeine can aggravate chronic migraine by predisposing to yet more headache, and for some relatively few and unlucky migraineurs
ingestion of caffeine may trigger an acute migraine attack. For the majority of migraine sufferers, however, caffeine can serve as a surprisingly effective therapeutic weapon for dealing with acute migrainous headache.

Why? For one thing, acute migraine induces gastroparesis (paralysis of the stomach”). If the stomach’s typical motility is absent, any oral medication that “drops in” may simply lie there instead of moving on to the small intestine to be absorbed into the bloodstream and transported to target receptors.

...wash down oral medication intended for acute migraine treatment with your favorite caffeinated beverage.

To circumvent this gastroparesis problem one can use a drug that rapidly exits the stomach despite its immobility (eg, Treximet), co-administer an oral medication that promotes stomach motility (eg, metoclopramide) or simply give up on the oral route and administer the acute migraine therapy intranasally or via injection. A cheap and simple alternative to these options: wash down oral medication intended for acute migraine treatment with your favorite caffeinated beverage.

Aside from its effect of speeding up the absorption of whatever oral medication is taken with it, caffeine also may have a direct effect on reducing acute migraine headache. In migraine's circuitry caffeine inhibits a key step in head pain signaling, and many migraineurs have discovered that a timely cup of coffee or can of soda will terminate acute migraine.

For the majority of migraineurs, then, here is your “caffeine prescription”:

1) Avoid chronic overuse of caffeine, but...
2) In the setting of acute migraine headache, administer your oral “rescue” medication with a caffeinated beverage.
3) Eliminate caffeine from your diet if you wish, but do so knowing that the value of any “food elimination diet” for migraine treatment remains an object of considerable controversy.

Migraine Myth of the Month

Myth: Migraine is caused by... stress, hormones, weather changes, sinus disease, problems with the temporomandibular joint (TMJ), “evil” humors, etc.

Reality: Migraine results from a genetically sensitized brain whose “primed” neurons may respond to a variety of external environmental stimuli (egs, barometric pressure changes, bright sunshine) or internal stimuli (fluctuations in the levels of sex hormones) by discharging electrically and igniting an attack of acute migraine. The stimulus is the match, the genetically sensitive brain is the fuse and the head pain signaling pathway of the nervous system is the stick of dynamite.

Not infrequently a patient will assure her provider that she has “red wine headaches, rainy weather headaches, headaches with my periods and stress headaches...but not migraine”. What this patient in fact is saying is that she has a migrainous brain that responds to a variety of common triggers by producing a migraine attack. It is her genes and her brain that cause her to have the disorder we identify as “migraine”. It is the stress, red wine, menses, etc that triggers migraine. Similarly, migraine may be aggravated by pain elsewhere in the body (especially in structures supplied by nerves that are part of the migraine circuitry: the neck, jaw, sinuses).

No matter the gender, race, ethnicity or country of origin, the most common migraine trigger reported by migraineurs is stress (Andress-Rothrock D, King W, Rothrock J. An analysis of migraine triggers in a clinic-based population. *Headache* 2010;50:1366). Ironically, a sudden release from stress also can precipitate a migraine attack, a fact of migraine life that puzzles and frustrates many of those thus afflicted. The genetically migrainous brain is highly reactive to change, be it “bad” change (a nasty run-in with your boss) or “good” (sleeping in on Saturday morning after a sleep-deprived week). [See “Celebrity Migraine” p9]

Wait a minute, you say. If migraine is genetic, why is it that no one else in my family but me has ever had a problem with migraine?

One possibility: you’re mistaken. If providers ask patients with migraine whether a 1st degree relative (mother, father, sister, brother) has migraine, about 50% respond “yes”. If clinical investigators directly interview those family members, about 90% respond “yes”.

Another possibility: in the generation preceding yours, there was very little clinical expression of the genetic predisposition to migraine in the affected individual(s). A red wine headache here. A menstrual headache there. Not much.

A final possibility: during your own embryogenesis, within your mother’s uterus, your DNA underwent a subtle mutation, and-voila!-you, like an Abraham of headache, created your very own new line of migraine. Congratulations?
Marilyn, a thirty-seven year old journalist living in Alexandria, Virginia writes:

“I’ve had it! While I’ve had migraines here and there at other times, since 8th grade I’ve had a severe migraine, lasting for days, every month when I have my period. For years, at least a fifth of my life has been consumed by these menstrual headaches. My mother had the same problem, and her migraines ended completely after menopause. Same thing with her mother. And my older sister.

I say, enough is enough. Why put up with this for another 10 year? I’ve had my children. I’m ready for a hysterectomy...and an end to these monthly migraines.”

Prior to puberty migraine is slightly more common in males, but from puberty onward the headache disorder is notable for its preponderance in females. By young adulthood the female:male ratio for migraine prevalence is 3:1, and only with the onset of menopause does this gender-related difference begin to diminish.

Migraine’s tendency to affect women more than men has been attributed primarily to the influence that female sex hormones exert upon the biologic circuitry that generates migrainous symptoms. Put simply, one of the key contributors to migraine’s preponderance in females is the fluctuations in estrogen levels that occur throughout much of a woman’s lifetime.

One of the most common clinical manifestations of this hormonal influence is migraine’s tendency to worsen just before and during menses. In “pure” menstrual migraine attacks occur only in conjunction with the menses. More commonly, actively cycling females with menstrually-related migraine (MRM) have attacks temporally related to menses and also at other times of the month.

A majority of actively cycling female migraineurs-up to 70%-report worsening of their migraine in association with menses. As a general rule, the headaches of MRM tend to be longer in duration and less responsive to acute treatment than headaches that occur at other times of the month; for some women, the menstrual week is marked by one long, continuous migraine. Once the MRM boulder starts rolling down the hill, its biologic momentum accelerates, making it hard to stop. Key to optimal treatment of MRM is stopping the migraine before it gets a chance to start.

Effective management of MRM typically involves the use of a calendar and headache diary. Tracking your migraine and menstrual cycle will help you identify when migraine occurs in relation to onset of flow. If there is a distinct pattern-say, migraine attacks usually begin the day prior to flow onset-and your cycles are regular and predictable, then anticipatory “mini-prophylaxis” can be utilized. A day or two prior to the anticipated onset of your MRM you can begin any one of a number of medications intended to prevent menstrual headache and continue that medication for the next 5 to 7 days, your “high risk” (for migraine) time. Medications commonly used for MRM mini-prophylaxis include magnesium oxide (400 milligrams (mg) once or twice daily-no prescription required), naproxen sodium (550-660 mg twice daily-available via prescription or over-the-counter) and frovatriptan (Frova; 2.5 mg twice daily-prescription only).

Strangely, some women find that successful prevention of MRM with their mini-prophylaxis therapy may simply shift the prolonged migraine they avoided to the week following the end of menses.

The same acute migraine treatments you use at other times of the month may be effective for MRM as well. Any of the 7 currently available oral triptans, Treximet (an oral compound containing brand Imitrex and naproxen sodium), intranasal zolmitriptan (Zomig), “exhalant” sumatriptan (Onzeta), various of the oral non-steroidal anti-inflammatory drugs (NSAIDs: egs, aspirin, naproxen sodium, ibuprofen, Cambia) and subcutaneously injected sumatriptan are reasonable options.

While elimination of menses may also eliminate MRM, DO NOT seek surgical menopause simply as a treatment for MRM. To their dismay, many women with MRM find that even following removal of their ovaries (and a consequent end to the cyclic fluctuations in levels of sex hormones) they continue to experience monthly episodes of week-long migraine that mimic their pre-surgical menstrual headaches. It’s as if the brain has become “hard-wired” to MRM, and the “hard disc” will continue to signal cyclical periods of migraine headache even in the absence of peripheral stimuli (ie, estrogen shifts).

There are ways to suppress menses that are reversible and far less invasive than surgical resection of the ovaries. You can take an active oral contraceptive throughout the month, skipping the week of differently-colored inactive pills. You can use an estrogen-secreting IUD for contraception. You can become pregnant! Any of these may lead to cessation of MRM (definitely so in the case of pregnancy).
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