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NOVEMBER 27, 2011

Psychologists chase down sleep demons

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What do Moby Dick, the Salem witch trials and alien abductions all have in common? They all circle back to sleep paralysis.

Less than 8 percent of the general population experiences sleep paralysis, but it is more frequent in two groups -- students and psychiatric patients -- according to a new study by psychologists at Penn State and the University of Pennsylvania.

Sleep paralysis is defined as "a discrete period of time during which voluntary muscle movement is inhibited, yet ocular and respiratory movements are intact," the researchers state in the current issue of Sleep Medicine Reviews. Hallucinations may also be present in these transitions to or from sleep.

Alien abductions and incubi and succubi, as well as other demons that attack while people are asleep, are implicated as different cultural interpretations of sleep paralysis. The Salem witch trials are now thought possibly to involve the townspeople experiencing sleep paralysis. And in the 19th-century novel Moby Dick, the main character Ishmael experiences an episode of sleep paralysis in the form of a malevolent presence in the room.

Brian A. Sharpless, clinical assistant professor of psychology and assistant director of the psychological clinic at Penn State, noted that some people who experience these episodes may regularly try to avoid going to sleep because of the unpleasant sensations they experience. But other people enjoy the sensations they feel during sleep paralysis.

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BLOG ARCHIVE

► 2012 (102)

▼ 2011 (85)

► December (3)

▼ November (12)

► Nov 28 (1)

▼ Nov 27 (1)

Psychologists chase down sleep demons

► Nov 25 (1)

► Nov 23 (1)

► Nov 19 (1)

► Nov 16 (1)

► Nov 14 (1)

► Nov 10 (1)

► Nov 08 (1)

► Nov 07 (1)

► Nov 04 (1)

► Nov 03 (1)

"I realized that there were no real sleep paralysis prevalence rates available that were based on large and diverse samples," Sharpless said. "So I combined data from my previous study with 34 other studies in order to determine how common it was in different groups." MHC CEUs

He looked at a total of 35 published studies from the past 50 years to find lifetime sleep paralysis rates. These studies surveyed a total of 36,533 people. Overall he found that about one-fifth of these people experienced an episode at least once. Frequency of sleep paralysis ranged from once in a lifetime to every night.

When looking at specific groups, 28 percent of students reported experiencing sleep paralysis, while nearly 32 percent of psychiatric patients reported experiencing at least one episode. People with panic disorder were even more likely to experience sleep paralysis, and almost 35 percent of those surveyed reported experiencing these episodes. Sleep paralysis also appears to be more common in non-Caucasians.

"Sleep paralysis should be assessed more regularly and uniformly in order to determine its impact on individual functioning and better articulate its relation to other psychiatric and medical conditions," said Sharpless.

He looked at a broad range of samples, and papers were included from many different countries.

People experience three basic types of hallucinations during sleep paralysis -- the presence of an intruder, pressure on the chest sometimes accompanied by physical and/or sexual assault experiences and levitation or out-of-body experiences.

Up to this point there has been little research conducted on how to alleviate sleep paralysis or whether or not people experience episodes throughout their lives.

"I want to better understand how sleep paralysis affects people, as opposed to simply knowing that they experience it," said Sharpless. "I want to see how it impacts their lives." Sharpless hopes to look at relationships between sleep paralysis and post-traumatic stress disorder in the future.

This research was supported in part by the National Institute of Mental Health.

Also working on this research was Jacques P. Barber, professor of psychiatry, University of Pennsylvania.

Posted by Continuing Education Resources at 9:02 PM

Labels: alien, mhc ceu's, mhc ceus, mhc continuing education, sleep

► October (6)
► September (6)
► August (2)
► July (1)
► June (5)
► May (10)
► March (4)
► February (9)
► January (27)

► 2010 (209)
► 2009 (4)
► 2008 (6)

PSYCHOLOGY LINKS

- Dr. Deb

ABOUT ME

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Alien Abduction

Wikipedia Articles

Contents

Articles

References

Article Licenses

Sleep paralysis

Sleep paralysis is a phenomenon in which people, either when falling asleep or wakening, temporarily experience a sense of inability to move, similar to when an arm or leg *goes to sleep*, but not associated with numbness. More formally, it is a transition state between wakefulness and rest characterized by complete muscle atonia (muscle weakness). It can occur at sleep onset or when awakening. It is believed a result of disrupted REM sleep, which is normally characterized by complete muscle atonia that prevents individuals from acting out their dreams. Sleep paralysis has been linked to disorders such as narcolepsy, migraines, anxiety disorders, and obstructive sleep apnea; however, it can also occur in isolation.[1][2] When linked to another disorder, sleep paralysis commonly occurs in association with the neuromuscular disorder narcolepsy.[2]

Classification

The Nightmare, by Henry Fuseli (1781) is thought to be one of the classic depictions of sleep paralysis perceived as a demonic visitation.

The two major classifications of sleep paralysis are *isolated sleep paralysis (ISP)* and *recurrent isolated sleep paralysis (RISP).* Of these two types ISP is much more common than RISP.[2] ISP episodes are infrequent and of short duration, approximately one minute. Sleep paralysis may only occur once in an individual's lifetime.[2] As the name suggests, recurrent isolated sleep paralysis is a chronic condition. The individual suffers from frequent episodes throughout their lifetime.[2] One of the major differences between ISP and RISP is duration. RISP episodes can last for up to an hour or longer, and have a much higher occurrence of perceived out of body experiences—while ISP episodes are generally short (usually no longer than one minute) and are typically associated with the intruder and incubus hallucinations. ISP episodes can, however, persist for up to half an hour.[2] With RISP the individual can also suffer back to back episodes of sleep paralysis in the same night while this is unlikely in individuals who suffer from ISP.[2]

It can be difficult to differentiate between cataplexy brought on by narcolepsy and true sleep paralysis, because the two phenomena are physically indistinguishable.[2] The best way to differentiate between the two is to note when the attacks occur most often. Narcolepsy attacks are more common when the individual is falling asleep; ISP and RISP attacks are more common on awakening.[3]

Prevalence

Isolated sleep paralysis is commonly seen in patients that have been diagnosed with narcolepsy. Approximately 30-50% of people that have been diagnosed with narcolepsy have experienced sleep paralysis as an auxiliary symptom.[1][4] The prevalence of sleep paralysis in the general population is approximately 6.2%. A majority of the individuals that have experienced sleep paralysis have sporadic episodes that occur once a month to once a year. Only 3% of individuals experiencing sleep paralysis that is not associated with a neuromuscular disorder have nightly episodes, as mentioned earlier, these individuals are diagnosed as having RISP.[1] Sleep paralysis is just as common for males as it is for females, however, different age groups have been found to be more susceptible to developing isolated sleep paralysis. Approximately 36% of the general population that experiences isolated sleep

paralysis is likely to develop it between 25 and 44 years of age.[1]

Pathophysiology

The pathophysiology of sleep paralysis has not been concretely identified, although there are several theories about what causes an individual to develop sleep paralysis. The first of these stems from the understanding that sleep paralysis is a parasomnia resulting from inappropriate overlap of the REM and waking stages of sleep.[5] Polysomnographic studies found that individuals with sleep paralysis had shorter REM sleep latencies than normal along with shortened NREM and REM sleep cycles, and fragmentation of REM sleep.[6] This study supports the observation that disturbance of regular sleeping patterns can instigate an episode of sleep paralysis, because fragmentation of REM sleep commonly occurs when sleep patterns are disrupted and has now been seen in combination with sleep paralysis.[6]

Another major theory is that the neural bodies that regulate sleep are out of balance in such a way that allows for the different sleep states to overlap.[7] In this case, cholinergic sleep on neural populations are hyper activated and the serotonergic sleep off neural populations are under-activated.[7] As a result the cells capable of sending the signals that would allow for complete arousal from the sleep state, the serotonergic neural populations, have difficulty in overcoming the signals sent by the cells that keep the brain in the sleep state.[7] Normally during REM sleep the threshold for a stimulus capable of causing arousal is greatly elevated; however, in individuals with SP there is almost no blocking of exogenous stimuli, which means it is much easier for the individual to be aroused by a stimulus.[7] There may also be a problem with the regulation of melatonin, which under normal circumstances regulates the serotonergic neural populations.[2] Melatonin is typically at its lowest point during REM sleep.[2] Inhibition of melatonin at an inappropriate time would make it impossible for the sleep off neural populations to depolarize when presented with a stimulus that would normally lead to complete arousal.[2] This could explain why the REM and waking stages of sleep overlap during sleep paralysis, and definitely explains the muscle paralysis experienced on awakening.[2] If the effects of the sleep on neural populations cannot be counteracted, we retain characteristics of the REM stage of sleep once we have awoken.

Research has found a genetic component in sleep paralysis.[8] The characteristic fragmentation of REM sleep, hypnopompic, and hypnagogic hallucinations have a heritable component in other parasomnias, which lends credence to the idea that sleep paralysis is also genetic.[9] Twin studies have shown that if one twin of a monozygotic pair experiences sleep paralysis that other twin is very likely to experience it as well.[9] The identification of a genetic component means that there is some sort of disruption of function at the physiological level. Further studies must be conducted to determine whether there is a mistake in the signaling pathway for arousal as suggested by the first theory presented, or whether the regulation of melatonin or the neural populations themselves have been disrupted.

Signs and symptoms

Physiologically, sleep paralysis is closely related to REM atonia, the paralysis that occurs as a natural part of REM (rapid eye movement) sleep. Sleep paralysis occurs either when falling asleep, or when awakening. When it occurs upon falling asleep, the person remains aware while the body shuts down for REM sleep, and it is called hypnagogic or predormital sleep paralysis. When it occurs upon awakening, the person becomes aware before the REM cycle is complete, and it is called hypnopompic or postdormital.[10] The paralysis can last from several seconds to several minutes, with some rare cases being hours, "by which the individual may experience panic symptoms"[11] (described below). As the correlation with REM sleep suggests, the paralysis is not entirely complete; use of EOG traces shows that eye movement is still possible during such episodes, however, the individual experiencing sleep paralysis is unable to speak.[12]

Le Cauchemar (*The Nightmare*), by Eugène Thivier (1894)

Hypnagogic and hypnopompic hallucinations are symptoms commonly experienced during episodes of sleep paralysis. Some scientists have proposed this condition as an explanation for reports of alien abductions and ghostly encounters.[13] A study by Susan Blackmore and Marcus Cox (the Blackmore-Cox study) of the University of the West of England supports the suggestion that reports of alien abductions are related to sleep paralysis rather than to temporal lobe lability.[14] There are three main types of these hallucinations that can be linked to pathologic neurophysiology.[7] These include the belief that there is an intruder in the room, the incubus, and vestibular motor sensations.[15]

Many people that experience sleep paralysis are struck with a deep sense of terror, because they sense a menacing presence in the room while paralyzed—hereafter referred to as *the intruder*. This phenomenon is believed to be the result of a hyper vigilant state created in the midbrain.[7] More specifically, the emergency response activates in the brain when individuals wake up paralyzed and feel vulnerable to attack.[15] This helplessness can intensify the effects of the threat response well above the level typical to normal dreams; this could explain why hallucinations during sleep paralysis are so vivid.[15] Normally the threat activated vigilance system is a protective mechanism the body uses to differentiate between dangerous situations and determine whether the fear response is appropriate.[15] This threat vigilance system is evolutionarily biased to interpret ambiguous stimuli as dangerous, because "erring on the side of caution" increases survival chances.[15] This could explain why those who experience sleep paralysis generally believe the presence they sense is evil.[15] The amygdala is heavily involved in the threat activation response mechanism, which is implicated in both intruder and incubus SP hallucinations.[3] The specific pathway the threat-activated vigilance system acts through is not perfectly understood. It is believed that either the thalamus receives sensory information and sends it on the amygdala, which regulates emotional experience—or that the amygdaloid complex, anterior cingulate, and the structures in the pontine tegmentum interact to create the hallucination.[7] It is also highly possible that SP hallucinations could result from a combination of these. The anterior cingulate has an extensive array of cortical connections to other cortical area, which lets it integrate the different sensations and emotions we experience.[7] The amygdaloid complex helps us interpret emotional experience and act appropriately.[16] Most importantly, it helps us direct our attention to the most pertinent stimuli in a potentially dangerous situation and act appropriately.[16] Proper amygdaloid complex function requires input from the thalamus. This creates a thalamoamygdala pathway capable of bypassing intense scrutiny of incoming stimuli, which allows for quick responses in a potentially life-threatening situation.[7][16]

Typically these pathways let us quickly disregard non-threatening situations. In sleep paralysis, however, these pathways become over-excited and move into a state of hypervigilance where the mind perceives every external stimuli as a threat. The individual can create endogenous stimuli that contribute to the perceived threat.[7] A similar process occurs in the incubus hallucination, with slight variations.

The incubus hallucination is associated with the subject's belief that an intruder is attempting to suffocate them, usually by strangulation.[15] It is believed that the incubus hallucination is a combination of the threat vigilance activation system and the muscle paralysis associated with sleep paralysis that removes voluntary control of breathing.[15] Several features of REM breathing patterns exacerbate the feeling of suffocation.[15] These include shallow rapid breathing, hypercapnia, and slight blockage of the airway, a symptom prevalent in sleep apnea patients.[7] Attempts at breathing deeply fail, and give the individual a sense of resistance—which the

threat-activated vigilance system interprets as someone sitting on their chest, suffocating them.[7] The sensation of entrapment causes a feedback loop that involves the threat-activated vigilance system: fear of suffocation increases as a result of continued helplessness, which makes the individual struggle to end the SP episode.[15] The intruder and incubus hallucinations highly correlate with one another—and moderately correlate with the third type of hallucination, vestibular-motor hallucination, also known as out-of-body experiences.[15]

The third hallucination type differs from the other two in that it involves the brainstem, cerebellar, and cortical vestibular centers—not the threat activation vigilance system.[3] Under normal conditions, medial and vestibular nuclei, cortical, thalamic, and cerebellar centers coordinate things such as head and eye movement, and orientation in space.[7] In sleep paralysis, these mechanisms—which usually coordinate body movement and provide information on body position—activate and, because there is no actual movement, become confused and induce a floating sensation.[15] The vestibular nuclei in particular has been identified as being closely related to dreaming during the REM stage of sleep.[7] Unlike the other two types of hallucinations, vestibular-motor experiences arise from completely endogenous sources of stimuli.[15]

Diagnosis

Sleep paralysis is mainly diagnosed by ruling out other potential sleep disorders that could account for the feelings of paralysis.[5] The main disorder that is checked for is narcolepsy due to the high prevalence of narcolepsy in conjunction with sleep paralysis. The availability of a genetic test for narcolepsy makes this an easy disorder to rule out.[9] Once all other conditions have been ruled out, the description that the patient gives of their episode is compared to the typical experiences of sleep paralysis that have been well documented.[5] If the two descriptions match and no other sleep disorder can account for the symptoms, the patient is diagnosed with sleep paralysis.[5]

Prevention

Several circumstances have been identified that are associated with an increased risk of sleep paralysis. These include insomnia and sleep deprivation, an erratic sleep schedule, sleeping in the supine position, stress, overuse of stimulants, physical fatigue, as well as certain medications that are used to treat ADHD.[2] It is also believed that there may be a genetic component in the development of RISP due to a high concurrent incidence of sleep paralysis in monozygotic twins.[9] Sleeping in the supine position has been found to be an especially prominent instigator of sleep paralysis.[17]

Sleeping in the supine position is believed to make the sleeper more vulnerable to episodes of sleep paralysis because in this sleeping position it is possible for the soft palate to collapse and obstruct the airway.[17] This is a possibility regardless of whether the individual has been diagnosed with sleep apnea or not. There may also be a greater rate of microarousals while sleeping in the supine position because there is a greater amount of pressure being exerted on the lungs by gravity when lying in the supine position.[17]

While many factors can increase risk for ISP or RISP, they can be avoided with minor lifestyle changes.[5] By maintaining a regular sleep schedule and observing good sleep hygiene, one can reduce chances of sleep paralysis. It helps subjects to reduce the intake of stimulants and stress in daily life by taking up a hobby or seeing a trained psychologist who can suggest coping mechanisms for stress. However, some cases of ISP and RISP involve a genetic factor—which means some people may find sleep paralysis unavoidable.

Treatment

Treatment starts with patient education about sleep stages and muscle atonia associated with REM sleep. Patients should be evaluated for narcolepsy if symptoms persist.[18] The safest treatment for sleep paralysis is for people to adopt healthier sleeping habits. However, in serious cases more clinical treatments are available. The most commonly used drugs are tricyclic antidepressants and selective serotonin reuptake inhibitors (SSRI's).[19] Despite the fact that these treatments are prescribed for serious cases of RISP, it is important to note that these drugs are not effective for everyone. There is currently no drug that has been found to completely interrupt episodes of sleep paralysis a majority of the time.[19]

Prognosis

Sleep paralysis poses no serious health risk to those that experience it, despite the fact that it can be an intensely terrifying experience. SUNDS is a deadly disorder believed to be related to sleep paralysis, however, they are still considered completely separate disorders, so sleep paralysis sufferers should not be alarmed.[2]

History

The original definition of sleep paralysis was codified by Samuel Johnson in his *A Dictionary of the English Language* as *nightmare*, a term that evolved into our modern definition. Such sleep paralysis was widely considered to be the work of demons, and more specifically incubi, which were thought to sit on the chests of sleepers. In Old English the name for these beings was *mare* or *mære* (from a proto-Germanic **marōn*, cf. Old Norse *mara*), hence comes the *mare* part in *nightmare*. The word might be etymologically cognate to Greek *Marōn* (in the Odyssey) and Sanskrit *Māra*.

Various forms of magic and spiritual possession were also advanced as causes. In nineteenth century Europe, the vagaries of diet were thought to be responsible. For example, in Charles Dickens's *A Christmas Carol*, Ebenezer Scrooge attributes the ghost he sees to "... an undigested bit of beef, a blot of mustard, a crumb of cheese, a fragment of an underdone potato..." In a similar vein, the *Household Cyclopedia* (1881) offers the following advice about nightmares:

> "Great attention is to be paid to regularity and choice of diet. Intemperance of every kind is hurtful, but nothing is more productive of this disease than drinking bad wine. Of eatables those which are most prejudicial are all fat and greasy meats and pastry... Moderate exercise contributes in a superior degree to promote the digestion of food and prevent flatulence; those, however, who are necessarily confined to a sedentary occupation, should particularly avoid applying themselves to study or bodily labor immediately after eating... Going to bed before the usual hour is a frequent cause of night-mare, as it either occasions the patient to sleep too long or to lie long awake in the night. Passing a whole night or part of a night without rest likewise gives birth to the disease, as it occasions the patient, on the succeeding night, to sleep too soundly. Indulging in sleep too late in the morning, is an almost certain method to bring on the paroxysm, and the more frequently it returns, the greater strength it acquires; the propensity to sleep at this time is almost irresistible."[20]

Folklore

- In Finnish and Swedish folklore, sleep paralysis is caused by a mare, a supernatural creature related to incubi and succubi. The mare is a damned woman, who is cursed and her body is carried mysteriously during sleep and without her noticing. In this state, she visits villagers to sit on their rib cages while they are asleep, causing them to experience nightmares. The Swedish film *Marianne* examines the folklore surrounding sleep paralysis.[21]
- Folk belief in Newfoundland, South Carolina and Georgia describe the negative figure of the hag who leaves her physical body at night, and sits on the chest of her victim. The victim usually wakes with a feeling of terror, has difficulty breathing because of a perceived heavy invisible weight on his or her chest, and is unable to move i.e., experiences sleep paralysis. This nightmare experience is described as being "hag-ridden" in the Gullah lore. The "Old Hag" was a nightmare spirit in British and also Anglophone North American folklore.
- In Fiji, the experience is interpreted as *kana tevoro*, being "eaten" by a demon. In many cases the demon can be the spirit of a recently dead relative who has come back for some unfinished business, or has come to communicate some important news to the living. Often persons sleeping near the afflicted person say *kania, kania*, "eat! eat!" in an attempt to prolong the possession for a chance to converse with the dead relative or spirit and seek answers as to why he or she has come back. The person waking up from the experience is often asked to immediately curse or chase the spirit of the dead relative, which sometimes involves literally speaking to the spirit and telling him or her to go away or using expletives.
- In Nigeria, "ISP appears to be far more common and recurrent among people of African descent than among whites or Nigerian Africans,"[11] and is often referred to within African communities as "the Devil on your back."[22][23][24]
- In Turkey sleep paralysis is called *karabasan*, and is similar to other stories of demonic visitation during sleep. A demon, commonly known as a *djinn* (*cin* in Turkish), comes to the victim's room, holds him or her down hard enough not to allow any kind of movement, and starts to strangle the person. To get rid of the demonic creature, one needs to pray to Allah with certain lines from the Qur'an.
- In Thailand it is believed that sleep paralysis and discomfort is caused by a ghost of the Thai folklore known as *Phi Am* (Thai: ผีอำ).[25] Some people claim that this spirit may even cause bruises.[26] Stories about this spirit are common in Thai comics.[27]
- In the Southern states of the United States, elders refer to it as the "witch riding your back."

Around the world

Complete references to many cultures are given in the References section

East Asia

- In Chinese culture, sleep paralysis is widely known as "鬼壓身/鬼压身" (pinyin: guǐ yā shēn) or "鬼壓床/鬼压床" (pinyin: guǐ yā chuáng), which literally translate into "ghost pressing on body" or "ghost pressing on bed." A more modern term is "夢魘/梦魇" (pinyin: mèng yǎn).
- In Japanese culture, sleep paralysis is referred to as *kanashibari* (金縛り), literally "bound or fastened in metal," from "kane" (metal) and "shibaru" (to bind, to tie, to fasten). This term is occasionally used by English speaking authors to refer to the phenomenon both in academic papers and in pop psych literature.[28]
- In Korean culture, sleep paralysis is called *gawi nulim* (Hangul: 가위눌림), literally, "being pressed down by a ghost." It is often associated with a belief that a ghost or spirit is lying on top of or pressing down on the sufferer.
- In Mongolian culture, nightmares in general as well as sleep paralysis is referred to by the verb-phrase *khar darakh* (written kara darahu), meaning "to be pressed by the Black" or "when the Dark presses." *Kara* means black, and may refer to the dark side personified. *Kharin buu* means "shaman of the black" (shamans of the dark side only survive in far-northern Mongolia), while *tsaghaan zugiin buu* means "shaman of the white direction"

(referring to shamans who only invoke benevolent spirits). Compare 'karabasan' (the dark presser) in Turkish, which may date from pre-Islamic times when the Turks had the same religion and mythology as the Mongols. See Mythology of the Turkic and Mongolian peoples and Tengriism.

South-East Asia

- In Cambodian, Lao, and Thai culture, sleep paralysis is called *phĭi am* (Thai pronunciation: [pʰǐi.ʔam], Lao pronunciation: [pʰǐi.ʔàm]) and *khmout sukkhot*. It is described as an event in which the person is sleeping and dreams that one or more ghostly figures are nearby or even holding him or her down. The sufferer is unable to move or make any noises. This is not to be confused with *pee khao* and *khmout jool*, ghost possession.
- In Hmong culture, sleep paralysis is understood to be caused by a nocturnal pressing spirit, *dab tsog*. *Dab tsog* attacks "sleepers" by sitting on their chests, sometimes attempting to strangle them. Some believe that *dab tsog* is responsible for sudden unexpected nocturnal death syndrome (SUNDS), which claimed the lives of over 100 Southeast Asian immigrants in the late 1970s and early 1980s. Adler (2011) offers a biocultural perspective on sleep paralysis and the sudden deaths. She suggests that an interplay between the Brugada syndrome (a genetic cardiac disorder) and the traditional meaning of a *dab tsog* attack are at the heart of the sudden deaths.[29]
- In Vietnamese culture, sleep paralysis is called *ma đè*, meaning "held down by a ghost," or *bóng đè*, meaning "held down by a shadow."
- In Philippine culture, *bangungut* has traditionally been attributed to nightmares.[30] People who claim to survive such nightmares report symptoms of sleep paralysis.
- In New Guinea, people refer to this phenomenon as *Suk Ninmyo*, believed to originate from sacred trees that use human essence to sustain its life. The trees are said to feed on human essence during night as to not disturb the human's daily life, but sometimes people wake unnaturally during the feeding, resulting in the paralysis.
- In Malay of Malay Peninsula, sleep paralysis is known as *kena tindih* (or *ketindihan* in Indonesia), which means "being pressed."[31] Incidents are commonly considered to be the work of a malign agency; occurring in what are explained as blind spots in the field of vision, they are reported as demonic figures.

South Asia

- In Pakistan, sleep paralysis is considered to be an encounter with Shaitan (Urdu: شیطان) (Satan), evil jinns or demons who have taken over one's body. Like Iran, this ghoul is known as 'bakhtak' (Urdu: بختک) or 'ifrit'. It is also assumed that it is caused by the black magic performed by enemies and jealous persons. People, especially children and young girls, wear Ta'wiz (Urdu: تعویز) (Amulet) to ward off evil eye. Spells, incantations and curses could also result in ghouls haunting a person. Some homes and places are also believed to be haunted by evil ghosts, satanic or other supernatural beings and they could haunt people living there especially during the night. Muslim holy persons (Imams, Maulvis, Sufis, Mullahs, Faqirs) perform exorcism on individuals who are believed to be possessed. The homes, houses, buildings and grounds are blessed and consecrated by Mullahs or Imams by reciting Qur'an and Adhan (Urdu: أذان), the Islamic call to prayer, recited by the muezzin.
- In Bangladesh, the phenomenon of sleep paralysis is referred to as *boba* meaning "speechless" for the reason that people can't move their body and can't talk even if they want to.
- In Tamil Nadu and Sri Lankan Tamil culture, this particular phenomenon is referred to as 'Amuku Be' or 'Amuku Pei' meaning "the ghost that forces one down."
- In Nepal, especially Newari culture it is also known as 'Khyaak' a ghost-like figure believed to reside in the darkness under the staircases of a house.

Middle-East, Western and Central Asia

- In Arabic Culture, sleep paralysis is often referred to as 'Kaboos' (Arabic: كابوس), literally "pressers" or 'Ja-thoom' (Arabic: جاثوم) literally "What sits heavily on something," though the term *kaboos* can also refer to any bad dream. In folklore across Arab countries, the 'kaboos' is believed to be a *shayṭān* or a *'ifrīt* that sits, heavily, on people's chests.
- In Turkish culture, sleep paralysis is often referred to as "karabasan" ("The dark presser/assailer"). It is believed to be a creature that attacks people in their sleep, pressing on their chest and stealing their breath. However, folk legends do not provide a reason why the devil or ifrit does that.
- In Persian culture it is known as 'bakhtak' (Persian: بختک), which is a ghost-like spear chucker creature that sits on the dreamer's chest, making breathing hard for him/her.

Africa

- In African culture, isolated sleep paralysis is commonly referred to as "the witch riding your back."[22][23]
- Ogun Oru is a traditional explanation for nocturnal disturbances among the Yoruba of Southwest Nigeria; ogun oru (nocturnal warfare) involves an acute night-time disturbance that is culturally attributed to demonic infiltration of the body and psyche during dreaming. Ogun oru is characterized by its occurrence, a female preponderance, the perception of an underlying feud between the sufferer's earthly spouse and a 'spiritual' spouse, and the event of bewitchment through eating while dreaming. The condition is believed to be treatable through Christian prayers or elaborate traditional rituals designed to exorcise the imbibed demonic elements.[32]
- In Zimbabwean Shona culture the word Madzikirira is used to refer something really pressing one down. This mostly refers to the spiritual world in which some spirit—especially an evil one—tries to use its victim for some evil purpose. The people believe that witches can only be people of close relations to be effective, and hence a witches often try to use one's spirit to bewitch one's relatives.
- In Ethiopian culture the word 'dukak' (ዱካክ - Amharic) is used, which is believed to be an evil spirit that possesses people during their sleep. Some people believe this experience is linked to use of Khat (ጫት 'Chat' - Amharic). Khat users experience sleep paralysis when suddenly quitting chewing Khat after use for a long time. In Amharic, the official language of Ethiopia, the word 'dukak' taken out of the context of Khat withdrawal related sleep paralysis, means depression. The evil spirit 'dukak' is an anthropomorphism (anthropomorphic personification) of the depression that often results from the act of quitting chewing Khat. 'Dukak' often appears in hallucinations of the quitters and metes out punishments to its victims for offending him by quitting. The punishments are often in the form of implausible physical punishments (e.g., the 'dukak' puts the victim in a bottle and shakes the bottle vigorously) or outrageous tasks the victim must perform (e.g., swallow a bag of gravel).[33]
- In Swahili speaking East Africa, it is known as *jinamizi*, which refers to a creature sitting on one's chest making it difficult for him/her to breathe. It is attributed to result from a person sleeping on his back. Most people also recall being strangled by this 'creature'. People generally survive these 'attacks'

Europe

- In Finnish folk culture sleep paralysis is called *unihalvaus* (dream paralysis), but the Finnish word for nightmare, *painajainen*, is believed to originally have meant sleep paralysis, as *painajainen* translates to *pusher* or *presser*.
- In Hungarian folk culture sleep paralysis is called *lidércnyomás* (*lidérc pressing*) and can be attributed to a number of supernatural entities like *lidérc* (wraith), *boszorkány* (witch), *tündér* (fairy) or *ördögszerető* (demon lover).[34] The word *boszorkány* itself stems from the Turkish root *bas-*, meaning "to press."[35]
- In Iceland folk culture sleep paralysis is generally called having a *Mara*. A goblin or a succubus (since it is generally female) believed to cause nightmares (the origin of the word 'Nightmare' itself is derived from an English cognate of her name). Other European cultures share variants of the same folklore, calling her under different names; Proto-Germanic: *marōn*; Old English: *mære*; German: *Mahr*; Dutch: *nachtmerrie*; Icelandic, Old

Norse, Faroese, and Swedish: *mara*; Danish: *mare*; Norwegian: *mare*; Old Irish: *morrigain*; Croatian, Bosnian, Serbian, Slovene: *môra*; Bulgarian, Polish: *mara*; French: *cauchemar*; Romanian: *moroi*; Czech: *můra*; Slovak: *mora*. The origin of the belief itself is much older, back to the reconstructed Proto Indo-European root *mora-*, an incubus, from the root *mer-* "to rub away" or "to harm."

- In Malta, folk culture attributes a sleep paralysis incident to an attack by the *Haddiela,* who is the wife of the *Hares*, an entity in Maltese folk culture that haunts the individual in ways similar to a poltergeist. As believed in folk culture, to get rid of the Haddiela, one must place a piece of silverware or a knife under the pillow prior to sleep.
- In Greece and Cyprus, it is believed that sleep paralysis occurs when a ghost-like creature or Demon named Mora, Vrachnas or Varypnas (Greek: Μόρα, Βραχνάς, Βαρυπνάς) tries to steal the victim's speech or sits on the victim's chest causing asphyxiation.
- In Catalan legend and popular culture, the *Pesanta* is an enormous dog (or sometimes a cat) that goes into people's houses in the night and puts itself on their chests making it difficult for them to breathe and causing them the most horrible nightmares. The Pesanta is black and hairy, with steel paws, but with holes so it can't take anything.

Americas

- During the Salem witch trials several people reported nighttime attacks by various alleged witches, including Bridget Bishop, that may have been caused by sleep paralysis.[36]
- In Mexico, it is believed that this is caused by the spirit of a dead person. This ghost lies down upon the body of the sleeper, rendering him unable to move. People refer to this as "subirse el muerto" (dead person on you).[37]
- In many parts of the Southern United States, the phenomenon is known as a *hag*, and the event is said to portend an approaching tragedy or accident.
- In Newfoundland, it is known as the 'Old Hag'.[38] In island folklore, the Hag can be summoned to attack a third party, like a curse. In his 1982 book, *The Terror that Comes in the Night*, David J. Hufford writes that in local culture the way to call the Hag is to recite the Lord's Prayer backwards.
- In contemporary western culture the phenomenon of supernatural assault are thought to be the work of what are known as shadow people. Victims report primarily three different entities, a man with a hat, the old hag noted above, and a hooded figure.[39] A flood of calls took place on the popular radio show *Coast to Coast AM*, hosted by George Noory, when the subject was introduced. Sleep paralysis is known to involve a component of hallucination in 20% of the cases, which may explain these sightings. It is also believed that the phenomenon of reported alien abduction is caused by sleep paralysis where the hallucination of aliens has been generated by 20th and 21st century science fiction.[40]
- Several studies show that African-Americans may be predisposed to isolated sleep paralysis—known in folklore as "the witch is riding you" or "the haint is riding you."[24] Other studies show that African-Americans who experience frequent episodes of isolated sleep paralysis, i.e., reporting having one or more sleep paralysis episodes per month coined as "sleep paralysis disorder," were predisposed to panic attacks.[41] This finding has been replicated by other independent researchers.[42][43]

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External links

- Sleep information and links (http://www.stanford.edu/~dement/paralysis.html) from Stanford University
- Sleep Paralysis and Associated Hypnagogic and Hypnopompic Experiences (http://www.arts.uwaterloo.ca/~acheyne/S_P.html) from University of Waterloo
- Waking Up to Sleep Paralysis (http://www.csicop.org/doubtandabout/sleep/)
- Sleep Paralysis Visions: Demons, Succubi, and the Archetypal Mind (http://www.realitysandwich.com/sleep_paralysis) by Ryan Hurd

Alien abduction

The terms **alien abduction** or **abduction phenomenon** describe "subjectively real memories of being taken secretly against one's will by apparently nonhuman entities and subjected to complex physical and psychological procedures".[1] People claiming to have been abducted are usually called "abductees" or "experiencers". Typical claims involve being subjected to a forced medical examination that emphasizes their reproductive system.[2] Abductees sometimes claim to have been warned against environmental abuse and the dangers of nuclear weapons.[3] While many of these claimed encounters are described as terrifying, some have been viewed as pleasurable or transformative.

Due to a lack of any substantial physical evidence, most scientists and mental health professionals dismiss the phenomenon as "deception, suggestibility (fantasy-proneness, hypnotizability, false-memory syndrome), personality, sleep phenomena, psychopathology, psychodynamics [and] environmental factors".[4] Skeptic Robert Sheaffer also sees similarity between the aliens depicted in early science fiction films, in particular, *Invaders From Mars*, and those reported to have actually abducted people.[5]

The first alien abduction claim to be widely publicized was the Betty and Barney Hill abduction in 1961.[6] Reports of the abduction phenomenon have been made around the world, but are most common in English speaking

countries, especially the United States.[5] The contents of the abduction narrative often seem to vary with the home culture of the alleged abductee.[5]

Alien abductions have been the subject of conspiracy theories and science fiction storylines (notably *The X-Files*) that have speculated on stealth technology required if the phenomenon were real, the motivations for secrecy, and that alien implants could be a possible form of physical evidence.

Overview

CUFOS Definition of an Abductee[7]
A person must be taken:
• Against his or her will • From terrestrial surroundings • By non-human beings.
The beings must take the person to:
• An enclosed place • Not terrestrial in appearance • Assumed or known to be an alien spacecraft by the witness.
In this place, the person must either:
• Be subjected to an examination, • Engage in communication (verbal or telepathic), • Or both.
These experiences may be remembered:
• Consciously • Or through methods of focused concentration such as hypnosis.

Mainstream scientists reject claims that the phenomenon literally occurs as reported. However, there is little doubt that many apparently stable persons who report alien abductions believe their experiences were real.[8] As reported in the *Harvard University Gazette* in 1992, Dr. John Edward Mack investigated over 800 claimed abductees and "spent countless therapeutic hours with these individuals only to find that what struck him was the 'ordinariness' of the population, including a restaurant owner, several secretaries, a prison guard, college students, a university administrator, and several homemakers ... 'The majority of abductees do not appear to be deluded, confabulating, lying, self-dramatizing, or suffering from a clear mental illness,' he maintained."[9] "While psychopathology is indicated in some isolated alien abduction cases," Stanley Krippner et al. confirmed, "assessment by both clinical examination and standardized tests has shown that, as a group, abduction experients are not different from the general population in term of psychopathology prevalence."[10] Other experts who have argued that abductees' mental health is no better or worse than average include psychologists John Wilson and Rima Laibow, and psychotherapist David Gotlib.[11]

Some abduction reports are quite detailed. An entire subculture has developed around the subject, with support groups and a detailed mythos explaining the reasons for abductions: The various aliens (Greys, Reptilians, "Nordics" and so on) are said to have specific roles, origins, and motivations. Abduction claimants do not always attempt to explain the phenomenon, but some take independent research interest in it themselves and explain the lack of greater awareness of alien abduction as the result of either extraterrestrial or governmental interest in cover-up.

History

As noted below, the Antonio Villas Boas case (1957) and the Hill abduction (1961) were the first cases of UFO abduction to earn widespread attention.

Though these two cases are sometimes viewed as the earliest abductions, skeptic Peter Rogerson[12] notes this assertion is incorrect: the Hill and Boas abductions, he contends, were only the first "canonical" abduction cases, establishing a template that later abductees and researchers would refine but rarely deviate from. Additionally, Rogerson notes purported abductions were cited contemporaneously at least as early as 1954, and that "the growth of the abduction stories is a far more tangled affair than the 'entirely unpredisposed' official history would have us believe." (The phrase "entirely unpredisposed" appeared in folklorist Thomas E. Bullard's study of alien abduction; he argued that alien abductions as reported in the 1970s and 1980s had little precedent in folklore or fiction.)

Paleo-abductions

While "alien abduction" did not achieve widespread attention until the 1960s, there were many similar stories circulating decades earlier. These early abduction-like accounts have been dubbed "paleo-abductions" by UFO researcher Jerome Clark.[13]

- In a 1897 edition of the Stockton, California *Daily Mail*, Colonel H. G. Shaw claimed he and a friend were harassed by three tall, slender humanoids whose bodies were covered with a fine, downy hair who tried to kidnap the pair.[13]
- Rogerson writes that the 1955 publication of Harold T. Wilkins's *Flying Saucers Uncensored* declared that Karl Hunrath and Wilbur Wilkinson, who had claimed they were contacted by aliens, had disappeared under mysterious circumstances; Wilkins reported speculation that the duo were the victims of "alleged abduction by flying saucers".[12]

Contactees

The UFO contactees of the 1950s claimed to have contacted aliens, and the substance of contactee narratives is often regarded as quite different from alien abduction accounts.

Two landmark cases

An early alien abduction claim occurred in the mid-1950s with the Antonio Villas Boas case, which did not receive much attention until several years later.

Widespread publicity was generated by the Betty and Barney Hill abduction case of 1961, culminating in a made-for-television film broadcast in 1975 (starring James Earl Jones and Estelle Parsons) dramatizing the events. The Hill incident was probably the prototypical abduction case and was perhaps the first in which the claimant described beings that later became widely known as the Greys and in which the beings were said to explicitly identify an extraterrestrial origin.

If fictional sources such as science fiction movies and pulps are taken into consideration, the phenomena might be traced back to the 1930s.

Later developments

Dr. Ronald Sprinkle (University of Wyoming psychologist) became interested in the abduction phenomenon in the 1960s. For some years, he was probably the only academic figure devoting any time to studying or researching abduction accounts. Sprinkle became convinced of the phenomenon's actuality, and was perhaps the first to suggest a link between abductions and cattle mutilation. Eventually Sprinkle came to believe that he had been abducted by aliens in his youth; he was forced from his job in 1989. (Bryan, 145fn)

Budd Hopkins—a painter, sculptor and raconteur by profession—had been interested in UFOs for some years. In the 1970s he became interested in abduction reports, and began using hypnosis to extract more details of dimly remembered events. Hopkins soon became a figurehead of the growing abductee subculture. (Schnabel 1994)

The 1980s brought a major degree of mainstream attention to the subject. Works by Budd Hopkins, Whitley Strieber, David M. Jacobs and John Edward Mack presented alien abduction as a genuine phenomenon. (Schnabel 1994)

Also of note in the 1980s was the publication of folklorist Dr. Thomas E. Bullard's comparative analysis of nearly 300 alleged abductees. The mid and late 1980s saw the involvement of two esteemed academic figures: Harvard psychiatrist John Mack and historian David M. Jacobs.

With Hopkins, Jacobs and Mack, several shifts occurred in the nature of the abduction narratives. There had been earlier abduction reports (the Hills being the best known), but they were believed to be few and far between, and saw rather little attention from ufology (and even less attention from mainstream professionals or academics). Jacobs and Hopkins argued that alien abduction was far more common than earlier suspected; they estimate that tens of thousands (or more) North Americans had been taken by unexplained beings. (Schnabel 1994)

Furthermore, Jacobs and Hopkins argued that there was an elaborate scheme underway, that the aliens were attempting a program to create human–alien hybrids, though the motives for this scheme were unknown. There were anecdotal reports of phantom pregnancy related to UFO encounters at least as early as the 1960s, but Budd Hopkins and especially David M. Jacobs were instrumental in popularizing the idea of widespread, systematic interbreeding efforts on the part of the alien intruders. Despite the relative paucity of corroborative evidence, Jacobs presents this scenario as not only plausible, but self-evident. Hopkins and Jacobs have also been criticized for selective citation of abductee interviews, favoring those that support their hypothesis of extraterrestrial intervention.

The involvement of Jacobs and Mack marked something of a sea change in the abduction studies. Their efforts were controversial (both men saw some degree of damage to their professional reputations), but to other observers, Jacobs and Mack brought a degree of respectability to the subject.

John Mack

Matheson writes that "if Jacobs's credentials were impressive," then those of Harvard psychiatrist John Edward Mack might seem "impeccable" in comparison. (Matheson, 251) Mack was a well known, highly esteemed psychiatrist, author of over 150 scientific articles and winner of the Pulitzer Prize for his biography of T. E. Lawrence. Mack became interested in the phenomenon in the late 1980s, interviewing over 800 people, and eventually writing two books on the subject.

In June 1992, Mack co-organized a five-day conference at MIT to discuss and debate the abduction phenomenon.[14] The conference attracted a wide range of professionals, representing a variety of perspectives. (In response to this conference, Mack and Jacobs were awarded an Ig Nobel Prize in 1993).

Writer C. D. Bryan attended the conference, initially intending to gather information for a short humorous article for The New Yorker. While attending the conference, however, Bryan's view of the subject changed, and he wrote a serious, open-minded book on the phenomenon, additionally interviewing many abductees, skeptics, and proponents.

Abductees

The precise number of alleged abductees is uncertain. One of the earliest studies of abductions found 1,700 claimants, while contested surveys argued that 5–6 percent of the general population might have been abducted.[4]

As a category, some studies show that abductees have psychological characteristics that render their testimony suspect.[5] Dr. Elizabeth Slater conducted a blind study of nine abduction claimants and found them to be prone to "mildly paranoid thinking," nightmares and having a weak sexual identity.[5]

According to Yvonne Smith, some alleged abductees test positive for lupus, despite not showing any symptoms.[15]

Paranormal

Alleged abductees are seen by many pro-abduction researchers to have a higher incidence of non-abduction related paranormal events and abilities.[16] Following an abduction experience, these paranormal abilities and occurrences sometimes seem to become more pronounced.[16] According to investigator Benton Jamison, abduction experiencers who report UFO sightings that should have been, but are not, reported by independent corroborating witnesses often seem to "be 'psychic personalities' in the sense of Jan Ehrenwald."[16]

Demographics

In a study investigating the motivations of the alleged abductors, Jenny Randles found that in each of the 4 cases out of 50 total where the experiencer was over 40 years of age or more, they were rejected by the aliens for "what they (the experiencers) usually inferred to be a medical reason."[17] Randles concludes "[T]he abduction is essentially a young person's experience."[17] Given the reproductive focus of the alleged abductions it is not surprising that one man reported being rejected because he had undergone a vasectomy.[18] It could also be partially because people over the age of 40 are less likely to have "hormonic" or reproductive activity going on.

Although abduction and other UFO-related reports are usually made by adults, sometimes young children report similar experiences.[19] These child-reports often feature very specific details in common with reports of abduction made by adults, including the cirumstances, narrative, entities and aftermaths of the alleged occurrences.[19] Often these young abductees have family members who have reported having abduction experiences.[19] Family involvement in the military, or a residence near a military base is also common amongst child abduction claimants.[19]

The abduction narrative

Although different cases vary in detail (sometimes significantly), some UFO researchers, such as folklorist Thomas E. Bullard[20] argue that there is a broad, fairly consistent sequence and description of events that make up the typical "close encounter of the fourth kind" (a popular but unofficial designation building on Dr. J. Allen Hynek's classifying terminology). Though the features outlined below are often reported, there is some disagreement as to exactly how often they actually occur.

Bullard argues most abduction accounts feature the following events. They generally follow the sequence noted below, though not all abductions feature all the events:

1. **Capture**. The abductee is forcibly taken from terrestrial surroundings to an apparent alien space craft.
2. **Examination and Procedures**. Invasive physiological and psychological procedures, and on occasion simulated behavioral situations, training & testing, or sexual liaisons.
3. **Conference**. The abductors communicate with the abductee or direct them to interact with specific individuals for some purpose.
4. **Tour**. The abductees are given a tour of their captors' vessel, though this is disputed by some researchers who consider this definition a confabulation of intent when just apparently being taken around to multiple places inside the ship.
5. **Loss of Time**. Abductees often rapidly forget the majority of their experience, either as a result of fear, medical intervention, or both.
6. **Return**. The abductees are returned to earth, occasionally in a different location from where they were allegedly taken or with new injuries or disheveled clothing.
7. **Theophany**. Coinciding with their immediate return, abductee may have a profound sense of love, a high, or "mystical experience", accompanied by a feeling of oneness with God, the universe, or their abductors. Whether this is the result of a metaphysical change, Stockholm Syndrome, or prior medical tampering is often not scrutinized by the abductees at the time.
8. **Aftermath**. The abductee must cope with the psychological, physical, and social effects of the experience.

When describing the "abduction scenario", David M. Jacobs says:

> The entire abduction event is precisely orchestrated. All the procedures are predetermined. There is no standing around and deciding what to do next. The beings are task-oriented and there is no indication whatsoever that we have been able to find of any aspect of their lives outside of performing the abduction procedures.[21]

Capture

Abduction claimants report unusual feelings preceding the onset of an abduction experience.[22] These feelings manifest as a compulsive desire to be at a certain place at a certain time or as expectations that something "familiar yet unknown," will soon occur.[22] Abductees also report feeling severe, undirected anxiety at this point even though nothing unusual has actually occurred yet.[22] This period of foreboding can last for up to several days before the abduction actually takes place or be completely absent.[22]

Eventually, the experiencer will undergo an apparent "shift" into an altered state of consciousness.[22] British abduction researchers have called this change in consciousness "the Oz Factor." External sounds cease to have any significance to the experiencer and fall out of perception.[22] They report feeling introspective and unusually calm.[22] This stage marks a transition from normal activity to a state of "limited self-willed mobility."[22] As consciousness shifts one or more lights are alleged to appear, occasionally accompanied by a strange mist.[22] The source and nature of the lights differ by report, sometimes the light emanates from a source outside the house (presumably the abductors' UFO), sometimes the lights are in the bedroom with the experiencer and transform into alien figures.[22]

As the alleged abduction proceeds, claimants say they will walk or be levitated into an alien craft, often through solid objects like walls or a window.[22] Alternatively, they may experience rising through a tunnel with or without the abductors accompanying them into the awaiting craft.[22]

Examination

The examination phase of the so-called "abduction narrative" is characterized by the performance of medical procedures and examinations by apparently alien beings against or irrespective of the will of the experiencer. Such procedures often focus on sex and reproductive biology. However, the literature holds reports of a wide variety of procedures allegedly performed by the beings. The entity that appears to be in charge of the operation is often taller than the others involved.[2][23]

Miller notes different areas of emphasis between human medicine and what is allegedly being practiced by the abductors.[2] The abductors' areas of interest appear to be the cranium (see below), nervous system, skin, reproductive system, and to a lesser degree, the joints.[2] Systems given less attention than a human doctor would, or omitted entirely include cardiovascular system, the respiratory system below the pharynx and the lymphatic system.[2] The abductors also appear to ignore the upper region of the abdomen in favor of the lower one.[2]

There are also differences in procedure as well as emphasis between human medicine and that claimed to be practiced by the entities. The abductors do not appear to wear gloves during the "examination."[2] Other constants of terrestrial medicine like pills and tablets are missing from abduction narratives although sometimes abductees are asked to drink liquids.[2] Injections also seem to be rare and IVs are almost completely absent.[2] Dr. Miller says he's never heard an abductee claim to have a tongue depressor used on them.[2]

Subsequent abduction procedures

After the so-called medical exam, the alleged abductees often report other procedures being performed with the entities.[21] Common among these post-examination procedures are what abduction researchers refer to as imaging, envisioning, staging, and testing.[21]

"Imaging" procedures consist of an abductee being made to view screens displaying images and scenes that appear to be specially chosen with the intent to provoke certain emotional responses in the abductee.[21] "Envisioning" is a similar procedure, with the primary difference being that the images being viewed, rather than being on a screen, actually seem to be projected into the experiencer's mind.[21] "Staging" procedures have the abductee playing a more active role, according to reports containing this element.[21] It shares vivid hallucination-like mental visualization with the envisioning procedures, but during staging the abductee interacts with the illusionary scenario like a role player or an actor.[21]

"Testing" marks something of a departure from the above procedures in that it lacks the emotional analysis feature.[21] During testing the experiencer is placed in front of a complicated electronic device and is instructed to operate it.[21] The experiencer is often confused, saying that they do not know how to operate it.[21] However, when they actually set about performing the task, the abductee will find that they do, in fact, know how to operate the machine.[21]

Child presentation

Abductees of all ages and genders sometimes report being subjected to a "child presentation."[21] As its name implies, the child presentation involves the abduction claimant being shown a "child."[21] Often the children appear to be neither human, nor the same species as the abductors.[21] Instead, the child will almost always share characteristics of *both* species.[21] These children are labeled by experiencers as hybrids between humans and their abductors, usually Greys.

Unlike Budd Hopkins and David Jacobs, folklorist Thomas E. Bullard could not identify a child presentation phase in the abduction narrative, even after undertaking a study of 300 abduction reports.[18] Bullard says that the child presentation "seems to be an innovation in the story"[18] and that "no clear antecedents" to descriptions of the child presentation phase exists before its popularization by Hopkins and Jacobs.[18]

Less common elements

Bullard also studied the 300 reports of alien abduction in an attempt to observe the less prominent aspects of the claims.[3] He notes the emergence of four general categories of events that recur regularly, although not as frequently as stereotypical happenings like the medical examination. These four types of events are:[3]

1. The conference
2. The tour
3. The journey
4. Theophany

Chronologically within abduction reports these rarer episodes tend to happen in the order listed, between the medical examination and the return.[3]

After allegedly displaying cold callous disregard towards the abduction experiencers, sometimes the entities will change drastically in behavior once the initial medical exam is completed.[3] They become more relaxed and hospitable towards their captive and lead him or her away from the site of the examination.[3] The entities then hold a **conference** with the experiencer, wherein they discuss things relevant to the abduction phenomenon.[3] Bullard notes five general categories of discussion that occur during the conference "phase" of reported abduction narratives: An interrogation session, explanatory segment, task assignment, warnings, and prophecies.[3]

Tours of the abductors' craft are a rare but recurring feature of the abduction narrative.[3] The tour seems to be given by the alleged abductors as a courtesy in response to the harshness and physical rigors of the forced medical examination.[3] Sometimes the abductee report traveling on a "**journey**" to orbit around Earth or to what appear to be other planets.[3] Some abductees find that the experience is terrifying, particularly if the aliens are of a more fearsome species, or if the abductee was subjected to extensive probing and medical testing.

Return

Eventually the abductors will return the abductees to *terra firma*, usually to exactly the same location and circumstances they were in before being taken.[24] Usually, explicit memories of the abduction experience will not be present, and the abductee will realize they have experienced "missing time" upon checking a timepiece.[24]

Sometimes the alleged abductors appear to make mistakes when returning their captives.[24] Famed UFO researcher Budd Hopkins has joked about "the cosmic application of Murphy's Law" in response to this observation.[24] Hopkins has estimated that these "errors" accompany 4–5 percent of abduction reports.[24] One type of common apparent mistake made by the abductors is failing to return the experiencer to the same spot that they were taken from initially.[24] This can be as simple as a different room in the same house, or abductees can even find themselves outside and all the doors of the house are locked from the inside.[24]

Realization event

Physician and abduction researcher John G. Miller sees significance in the reason a person would come to see themselves as being a victim of the abduction phenomenon.[25] He terms the insight or development leading to this shift in identity from non-abductee to abductee the "realization event."[25] The realization event is often a single, memorable experience, but Miller reports that not all abductees experience it as a distinct episode.[25] Either way, the realization event can be thought of as the "clinical horizon" of the abduction experience.[25]

Trauma and recovery

Most people alleging alien abductions report invasive examinations of their bodies[26] and some ascribe psychological trauma to their experiences.[27] Alleged abductees claim their memories of the abduction events have caused posttraumatic stress disorder (PTSD). "Post abduction syndrome" is a term used by abductees to describe the effects of abduction, though it is not recognized by any professional treatment organizations.[27] The difference between PAS and PTSD is described as the recurrence of the phenomenon and the inability to identify when the disorder started; furthermore, the medical community considers PTSD to be a severe and debilitating ailment whereas "PAS" has been promoted only by fringe researchers.

Support groups

Support groups for people who believed they were abducted began appearing in the mid-1980s. These groups appear throughout the United States, Canada and Australia. Their members are primarily white married females with some college education.[28]

Therapeutical Hypnosis

Many alien abductees recall much of their abduction(s) through hypnosis.[29] Because of this, it is claimed by some skeptics that the vast majority of evidence for alien abduction is based on memories 'recovered' through hypnosis. Due to the extensive use of hypnosis, the abduction narratives are frequently explained by skeptics as false memories and suggestions by the hypnotherapist.[30] Alleged abductees seek out hypnotherapists to try to resolve issues such as missing time or unexplained physical symptoms such as muscle pain or headaches. This usually involves two phases, an information gathering stage, in which the hypnotherapist asks about unexplained illnesses or unusual phenomena

during the patients lives (caused by or distortions of the alleged abduction), followed by hypnosis and guided imagery to facilitate recall. The information gathering enhances the likelihood that the events discussed will be incorporated into later abduction "memories".[31] Seven steps are hypothesized to lead to the development of false memories:[30]

1. A person is predisposed to accept the idea that certain puzzling or inexplicable experiences might be telltale signs of UFO abduction.
2. The person seeks out a therapist, whom he or she views as an authority and who is, at the very least, receptive to this explanation and has some prior familiarity with UFO abduction reports.
3. Alternatively, the therapist frames the puzzling experiences in terms of an abduction narrative.
4. Alternative explanations of the experiences are not explored.
5. There is increasing commitment to the abduction explanation and increasing anxiety reduction associated with ambiguity reduction.
6. The therapist legitimates or ratifies the abductee's experience, which constitutes additional positive reinforcement.
7. The client adopts the role of the "victim" or abductee, which becomes integrated into the psychotherapy and the client's view of self.

Perspectives

There have been a variety of explanations offered for abduction phenomena, ranging from sharply skeptical appraisals, to uncritical acceptance of all abductee claims, to the demonological, to everything in between.

Some have elected not to try explaining things, instead noting similarities to other phenomena, or simply documenting the development of the alien abduction phenomenon.

Others are intrigued by the entire phenomenon, but hesitate in making any definitive conclusions. The late Harvard psychiatrist John Mack concluded, "The furthest you can go at this point is to say *there's an authentic mystery here.* And that is, I think, as far as anyone *ought* to go." (emphasis as in original) (Bryan, 269)

Putting aside the question of whether abduction reports are literally and objectively "real", literature professor Terry Matheson argues that their popularity and their intriguing appeal are easily understood. Tales of abduction "are intrinsically absorbing; it is hard to imagine a more vivid description of human powerlessness." After experiencing the frisson of delightful terror one may feel from reading ghost stories or watching horror movies, Matheson notes that people "can return to the safe world of their homes, secure in the knowledge that the phenomenon in question cannot follow. But as the abduction myth has stated almost from the outset, there is no avoiding alien abductors." (Matheson, 297)

Matheson writes that when compared to the earlier contactee reports, abduction accounts are distinguished by their "relative sophistication and subtlety, which enabled them to enjoy an immediately more favorable reception from the public."

Skeptical perspectives

Skeptical perspectives assert that reports of people being kidnapped and subjected to forced medical examinations by extraterrestrial creatures do not occur literally as reported. Although being only one of many competing explanations for the phenomenon, it is the only one that is widely accepted by mainstream scientists and historians.

Various hypotheses have been proposed by skeptics to explain reports without the need to invoke non-parsimonious concepts such as intelligent extraterrestrial life forms. These hypotheses usually center on known psychological processes that can produce subjective experiences similar to those reported in abduction claims. Skeptics are also likely to critically examine abduction claims for evidence of hoaxing or influence from popular culture sources such as science fiction. One example of a comprehensive, skeptical analysis that focuses on the effects of mass marketing

is art historian John F. Moffitt's 2003 book *Picturing Extraterrestrials: Alien Images in Modern Mass Culture*[32]

Examples

- According to Brian Dunning proposed psychological alternative explanations of the abduction phenomenon have included hallucination, temporary schizophrenia, epileptic seizures and parasomnia—near-sleep mental states (hypnogogic states, night terrors and sleep paralysis). Sleep paralysis in particular is often accompanied by hallucinations and peculiar sensation of malevolent or neutral presence of "something," though usually people experiencing it do not interpret that "something" as aliens.[33]
- It is possible that some alleged abductees may experience spontaneous lucid dreams. It was proven by the OOBE Research Center during their mass alien abduction experiment (Los Angeles, USA, October 2011).[34]
- It is possible that some alleged abductees may be mentally unstable or under the influence of recreational drugs.
- In *The Demon-Haunted World* astronomer Carl Sagan points out that the alien abduction experience is remarkably similar to tales of demon abduction common throughout history. "There is no spaceship in these stories. But most of the central elements of the alien abduction account are present, including sexually obsessive non-humans who live in the sky, walk through walls, communicate telepathically, and perform breeding experiments on the human species. Unless we believe that demons really exist, how can we understand so strange a belief system, embraced by the whole Western world (including those considered the wisest among us), reinforced by personal experience in every generation, and taught by Church and State? Is there any real alternative besides a shared delusion based on common brain wiring and chemistry?" (Sagan 1996 124)
- It has also been noted that Terence McKenna described seeing "Machine Elves" while experimenting with Dimethyltryptamine (also known as DMT). In a 1988 study conducted at UNM, psychiatrist Rick Strassman found that approximately 20 percent of volunteers injected with high doses of DMT had experiences identical to purported Alien Abductions.[35]

Paranormal and conspiratorial

- Some have argued that alien abduction is a literal phenomenon: extraterrestrials kidnap humans to conduct studies or experiments. This is a well-known popular explanation, but has seen very little support from most mainstream scientists.
- Various authors, including Jacques Vallée and John Mack, have suggested that the dichotomy 'real' versus 'imaginary' may be too simplistic; that a proper understanding of this complex phenomenon may require a reevaluation of our concept of the nature of reality.

Testimonials

Abduction researcher Brian Thompson claims that a nurse acquaintance of his reported that during 1957 in Cincinnati she encountered a 3-foot-tall (0.91 m) praying mantis-like entity two days after a V-shaped UFO sighting.[23] This mantis-like creature is reminiscent of the insectoid-type entity reported in some abduction accounts.[23] He related this report to fellow researcher Leonard Stringfield.[23] Stringfield told him of two cases he had in his files where separate witnesses reported identical circumstances in the same place and year.[23]

While some corroborated accounts seem to support the literal reality of the abduction experience, others seem to support a psychological explanation for the phenomenon's origins. Jenny Randles and Keith Basterfield both noted at the 1992 MIT alien abduction conference that of the five cases they knew of where an abduction researcher was present at the onset of an abduction experience, the experiencer "didn't physically go anywhere."[36]

Brazilian researcher Gilda Moura reported on a similar case, the Sueli case, from her home country. When psychologist and UFO researcher Don Donderi said that these cases were "evidence of psychological processes" that did not "have anything to do with a physical alien abduction," Moura replied "If the Sueli case is not an abduction, I don't know what is an abduction any more."[36] Gilda Moura noted that in the Brazilian Sueli case during the

abduction UFOs were observed.[36] Later, she claims the experiencer had eye burns, saw lights and there seemed to be residual poltergeist activity.[36]

Attempts at confirmation

It has been argued that if actual "flesh and blood" aliens are abducting humans, there should be some hard evidence that this is occurring.[5] Proponents of the physical reality of the abduction experience have suggested ways that could conceivably confirm abduction reports.

One procedure reported occurring during the alleged exam phase of the experience is the insertion of a long needle-like contraption into a woman's navel.[2] Some have speculated that this could be a form of laparoscopy.[2] If this is true, after the abduction there should be free gas in the female's abdomen, which could be seen on an x-ray.[2] The presence of free gas would be extremely abnormal, and would help substantiate the claim of some sort of procedure being done to her.[2]

Notable abduction claims

- 1956: Elizabeth Klarer (South Africa)
- 1957: Antonio Villas Boas (Brazil)
- 1961: Betty and Barney Hill abduction (USA)[6]
- 1967: Betty Andreasson (USA)
- 1967: Schirmer Abduction (USA)
- 1973: Pascagoula Abduction (USA)
- 1975: Travis Walton (USA)
- 1976: Allagash Abductions (USA)
- 1978: Valentich disappearance (Australia)
- 1978-1981: Pier Fortunato Zanfretta (Italy)
- 1979: Robert Taylor incident (Scotland)
- 1970s–1980s: Whitley Strieber (USA)
- 1997: Kirsan Ilyumzhinov (Russia)[37]

Notable figures

- Brigitte Barclay
- Budd Hopkins
- Linda Moulton Howe
- David Icke
- David M. Jacobs
- John Edward Mack
- Whitley Strieber

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External links

- Alien abduction (http://www.dmoz.org//Society/Paranormal/UFOs/Abduction/) at the Open Directory Project

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Brian Dunning (born 1965) is an American writer and producer who specialises in the debunking of pseudoscience. He has hosted a weekly podcast, *Skeptoid*, since 2006 and has also written articles and books, and produced a TV series on the subject. He was formerly chief technology officer of Buylink Corporation, a large business-to-business network. Skeptoid is the 2012 science winner of the Stitcher podcast awards.[2]

Biography

Dunning was born in 1965 in Santa Monica, California, USA. Between 1983 and 1984 he studied computer science at Brigham Young University; film and television at UCLA from 1985 to 1987 and then fiction writing at UC Irvine between 1988 and 1992.

Buylink

In 1996 he co-founded and was chief technology officer for Buylink Corporation[3] which grew by 1999 into the world's largest business-to-business network with 23,000 members transacting over $5 billion annually. Buylink was funded by Hummer Winblad Venture Partners and Draper Fisher Jurvetson,[4] and Dunning led its team working

with Wells Fargo Bank in the development of the first online payment factoring system, code-named PayLater.[5] In 2000 he lectured on Buylink at the UC Berkeley's Entrepreneur Forum called *Bricks to Clicks in the New Internet Reality*,[6] and in the same year appeared on CNNfn with Rhonda Schaffler's Maverick of the Morning segment to talk about Buylink.[7]

Between 1997 and 2005 he was technical editor for FileMaker Advisor Magazine,[8] and contributing editor of ISO FileMaker Magazine, 1996–2002,[9] winning one of the FileMaker Excellence Awards at the 2001 FileMaker Developers Conference.[10]

Skepticism

Since 2006 Dunning has hosted and produced *Skeptoid: Critical Analysis of Pop Phenomena*,[11] a weekly audio podcast dedicated "to furthering knowledge by blasting away the widespread pseudosciences that infect popular culture, and replacing them with way cooler reality."[12] He is also the author of the book of the same title and a sequel. He produced *Here Be Dragons*, a free 40 minute video introduction to critical thinking intended for general audiences,[13] and received an award from the Portland Humanist Film Festival for this in November 2011.[14] He was also winner of the 2010 Parsec Award for "Best Fact Behind the Fiction Podcast".[15] and in August 2010 received an award recognizing his contributions in the skeptical field, from the Independent Investigations Group (IIG) during its 10th Anniversary Gala.[16]

Dunning also writes articles for Skepticblog.org,[17] and is an executive producer for the network television pilot *The Skeptologists*.[18] He is a member of the National Association of Science Writers,[19] and is the "Chancellor" of the non-accredited "Thunderwood College", a parody of unaccredited institutions of higher learning that offer "degrees" in a variety of subjects.[20]

Skeptoid podcast

Skeptoid is Dunning's weekly podcast. The show follows an audio essay format, and is dedicated to the critical examination of pseudoscience and the paranormal. In May 2012, *Skeptoid* became a California state non-profit corporation.[21]

Along with similarly themed *Point of Inquiry*, *Skepticality: The Official Podcast of Skeptic Magazine*, and *The Skeptic's Guide to the Universe*, it is a Top-50 program on the iTunes (US) Science & Medicine podcast charts.[22] In December 2011, *Skeptoid* claimed to have a weekly average of 174,000 downloads.[23]

Brian Dunning in the giant hamster wheel at the College of Curiosity in 2012, City Museum, St. Louis MO

Each roughly ten-minute *Skeptoid* episode focuses on a single pop culture phenomenon that is pseudoscientific in nature. Episodes usually fall into one of four categories:

- Quackery medical modalities: such as homeopathy, reflexology, detoxification, or chiropractic
- Popular cultural misconceptions: such as organic foods, SUVs, and global warming
- Urban legends: such as crop circles, the Amityville Horror, the Phoenix Lights, or the Philadelphia Experiment
- Religion and mythology: such as creation legends, New Age religions, and concepts of sin

In May 2007, *Skeptoid* was announced as a qualifying media outlet for the James Randi Educational Foundation's "One Million Dollar Paranormal Challenge".[24] Applicants to the challenge must have a presence in popular broadcast media in order to qualify.[25] By passing a simple test of their paranormal ability and having it reported on the *Skeptoid* podcast, applicants can satisfy that primary requirement and then proceed to apply for the Challenge.

Episode 300

In celebration of the 300th episode of the podcast, Dunning, composer Lee Sanders, singer Rachel Bloom, artist Jesse Horn, director Ryan Johnson and sound mixer Bill Simpkins produced the animated musical short, *The Gypsy Queen.* It is the story of a young girl who challenged the Queen and her rats who sold everyone in the village a *uber-scarf* which when worn across the eyes blinds the wearer to all unpleasantness. The villagers, once the uber-scarf was removed discovered they had been scammed, and while blind the rats had emptied the homes of food and valuables. The premier was released at the University of Irvine March 3, 2012. Entertainment that evening featured comedians Penny Chan and Matt Kirshen. Mentalism by Mark Edward, emceed by Emery Emery.[26]

Honors

Skeptoid was a 2009 Podcast Awards finalist in the Education category.[27]

In 2010, *Skeptoid* won the Parsec Award for "Best Fact Behind the Fiction" podcast.[28]

Skeptoid has been praised for "Outstanding Contribution to Science and Skepticism" by the Independent Investigations Group (IIG)[29]

In 2012, "Skeptoid" was nominated for a Stitcher Award in the Best Science Podcast category.[30]

Skeptoid 300th Episode Party - Brian Dunning, Ryan Johnson, Jesse Horn, Lee Sanders and Bill Simpkins in a Q&A discussing the premier of "The Gypsy Queen"

Fraud Indictment

In August 2008, eBay filed suit against Dunning and two other men accusing them of defrauding eBay and eBay affiliates in a cookie stuffing scheme.[31] On June 24, 2010, based on the same allegations and following an investigation by the Federal Bureau of Investigation, a grand jury indicted Dunning on charges of wire fraud.[32][33] According to Dunning "There are several legal reasons that the lawsuit is improper, and we've been fighting it on that basis."[34]

In the media

Dunning on the Discovery Channel *Weird or What?* television shoot, March 17, 2011.

- Dunning was a speaker at the Northeast Conference on Science and Skepticism in 2011.[35]
- Ohlone College, Psychology Club Speaker Series speaks on *Health Scams and Myths*[36]
- Dunning spoke at Google with a presentation of *Sounds From Beyond*.[37] When asked if he sees any positive science trends in the media he answered, "No...I think we have a negative trend, in the realm of rationality and reason it is going to continue to slide. And the reason I think we are going to continue to lose ground as far as capturing the hearts and minds of the general public, is because of the Internet and television, its become so easy to reach so many people with a sensational message. People don't have time to listen to ten things a day, they have time to listen to one thing and it is whatever is easiest. Its the front page of CNN or the first five minutes of Oprah, that's where people get their science information."
- *Sounds From Beyond* lecture for Pixar[38]
- On the Queen Mary BIL conference.[39]
- Chaos Communication Congress: *Here Be Dragons* in Berlin[40]
- At SkeptiCal Conference 2010: lecture on *The Virgin of Guadalupe: A Positive Take*[41]
- Skeptics Guide to the Universe[42] Dunning announces a casting call for *The Skeptologists*, we need "...skeptical experts to jump in the Batmobile racing to the center of the woo to work their skeptical magic on whatever the

topic is that week".

- James Randi Educational Foundation The Amaz!ng Meeting TAM 2012[43] ; TAM 9, 2011; TAM Australia 2011; TAM 8, 2010; TAM 7, 2009; TAM 6, 2008; TAM 5.5, 2008

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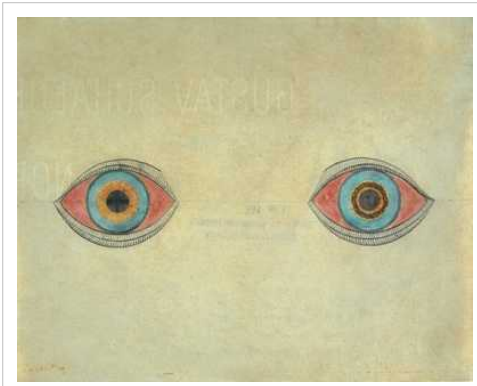
Hallucination

Hallucination	
Classification and external resources	
	
My eyes at the moment of the apparitions by August Natterer.	
ICD-10	R44 [1]
ICD-9	780.1 [2]
DiseasesDB	19769 [3]
MedlinePlus	003258 [4]
MeSH	D006212 [5]

A **hallucination**, in the broadest sense of the word, is a perception in the absence of a stimulus. In a stricter sense, hallucinations are defined as perceptions in a conscious and awake state in the absence of external stimuli which have qualities of real perception, in that they are vivid, substantial, and located in external objective space. The latter definition distinguishes hallucinations from the related phenomena of dreaming, which does not involve wakefulness; illusion, which involves distorted or misinterpreted real perception; imagery, which does not mimic real perception and is under voluntary control; and pseudohallucination, which does not mimic real perception, but is not under voluntary control.[6] Hallucinations also differ from "delusional perceptions", in which a correctly sensed and interpreted stimulus (i.e. a real perception) is given some additional (and typically bizarre) significance.

Hallucinations can occur in any sensory modality — visual, auditory, olfactory, gustatory, tactile, proprioceptive, equilibrioceptive, nociceptive, thermoceptive and chronoceptive.

A mild form of hallucination is known as a *disturbance*, and can occur in any of the senses above. These may be things like seeing movement in peripheral vision, or hearing faint noises and/or voices. Auditory hallucinations are very common in paranoid schizophrenia. They may be benevolent (telling the patient good things about themselves) or malicious, cursing the patient etc. Auditory hallucinations of the malicious type are frequently heard like people talking about the patient behind their back. Like auditory hallucinations, the source of their visual counterpart can also be behind the patient's back. Their visual counterpart is the feeling of being looked-stared at, usually with malicious intent. Frequently, auditory hallucinations and their visual counterpart are experienced by the patient together.

Hypnagogic hallucinations and hypnopompic hallucinations are considered normal phenomena. Hypnagogic hallucinations can occur as one is falling asleep and hypnopompic hallucinations occur when one is waking up.

Hallucinations can be associated with drug use (particularly deliriants), sleep deprivation, psychosis, neurological disorders, and delirium tremens.

Classification

Hallucinations may be manifested in a variety of forms.[7] Various forms of hallucinations affect different senses, sometimes occurring simultaneously, creating multiple sensory hallucinations for those experiencing them.

Auditory

Auditory hallucinations (also known as *paracusia*)[8] are the perception of sound without outside stimulus. Auditory hallucinations can be divided into two categories: elementary and complex. Elementary hallucinations are the perception of sounds such as hissing, whistling, an extended tone, and more. In many cases, tinnitus is an elementary auditory hallucination. However, some people who experience certain types of tinnitus, especially pulsatile tinnitus, are actually hearing the blood rushing through vessels near the ear. Because the auditory stimulus is present in this situation, it does not qualify as a hallucination.

Complex hallucinations are those of voices, music, or other sounds which may or may not be clear, may be familiar or completely unfamiliar, and friendly or aggressive, among other possibilities. Hallucinations of one or more talking voices are particularly associated with psychotic disorders such as schizophrenia, and hold special significance in diagnosing these conditions. However, many people not suffering from diagnosable mental illness may sometimes hear voices as well.[9] One important example to consider when forming a differential diagnosis for a patient with paracusia is lateral temporal lobe epilepsy. Despite the tendency to associate hearing voices, or otherwise hallucinating, and psychosis with schizophrenia or other psychiatric illnesses, it is crucial to take into consideration that even if a person does exhibit psychotic features, they do not necessarily suffer from a psychiatric disorder on its own. Disorders such as Wilson's disease, various endocrinological disorders, numerous metabolic disturbances, multiple sclerosis, systemic lupus erythematosis, porphyria, sarcoidosis, and many others can present with psychosis.

Musical hallucinations are also relatively common in terms of complex auditory hallucinations and may be the result of a wide range of causes ranging from hearing-loss (such as in musical ear syndrome, the auditory version of Charles Bonnet syndrome), lateral temporal lobe epilepsy,[10] arteriovenous malformation,[11] stroke, lesion, abscess, or tumor.[12]

The Hearing Voices Movement is a support and advocacy group for people who hallucinate voices, but do not otherwise show signs of mental illness or impairment.

High caffeine consumption has been linked to an increase in the likelihood of experiencing auditory hallucinations. A study conducted by the La Trobe University School of Psychological Sciences revealed that as few as five cups of coffee a day could trigger the phenomenon.[13]

Command hallucinations

Command hallucinations are hallucinations in the form of commands; they can be auditory or inside of the persons mind and / or consciousness.[14] The contents of the hallucinations can range from the innocuous to commands to cause harm to the self or others.[14] Command hallucinations are often associated with schizophrenia. People experiencing command hallucinations may or may not comply with the hallucinated commands, depending on circumstances. Compliance is more common for non-violent commands.[15]

Command hallucinations are sometimes used in defense of a crime, often homicides. It is essentially a voice one hears and it tells them what to do. "Sometimes they are quite benign directives such as "Stand up." or "Shut the door."[16] Whether it is a command for something simple or something that is a threat, it is still considered a "command hallucination." Some helpful questions that can assist one in figuring out if they may be suffering from this includes: "What are the voices telling you to do?","When did your voices first start telling you to do things?, "Do

you recognize the person who is telling you to harm yourself (others)?", "Do you think you can resist doing what the voices are telling you to do?".[16]

Olfactory

Phantosmia is the phenomenon of smelling odors that aren't really present. The most common odors are unpleasant smells such as rotting flesh, vomit, urine, feces, smoke, or others. Phantosmia often results from damage to the nervous tissue in the olfactory system. The damage can be caused by viral infection, brain tumor, trauma, surgery, and possibly exposure to toxins or drugs.[17] Phantosmia can also be induced by epilepsy affecting the olfactory cortex and is also thought to possibly have psychiatric origins. Phantosmia is different from parosmia, in which a smell is actually present, but perceived differently from its actual smell.

Olfactory hallucinations can also appear in some cases of associative imagination, for example, while watching a romance movie, where the man gifts roses to the woman, the viewer senses the roses' odor (which in fact does not exist).

Olfactory hallucinations have also been reported in migraine, although the frequency of such hallucinations is unclear.[18][19]

Tactile hallucinations

Tactile hallucinations are the illusion of tactile sensory input, simulating various types of pressure to the skin or other organs. One subtype of tactile hallucination, formication, is the sensation of insects crawling underneath the skin and is frequently associated with prolonged cocaine or amphetamine use[20] or with withdrawal from alcohol or benzodiazepines. However, formication may also be the result of normal hormonal changes such as menopause, or disorders such as peripheral neuropathy, high fevers, Lyme disease, skin cancer, and more.[20]

Gustatory

This type of hallucination is the perception of taste without a stimulus. These hallucinations, which are typically strange or unpleasant, are relatively common among individuals who have certain types of focal epilepsy, especially temporal lobe epilepsy. The regions of the brain responsible for gustatory hallucination in this case are the insula and the superior bank of the sylvian fissure.[21][22]

General somatic sensations

General somatic sensations of a hallucinatory nature are experienced when an individual feels that his body is being mutilated i.e. twisted, torn, or disembowelled. Other reported cases are invasion by animals in the person's internal organs such as snakes in the stomach or frogs in the rectum. The general feeling that one's flesh is decomposing is also classified under this type of hallucination.[23]

Cause

Hallucinations can be caused by a number of factors.

Hypnagogic hallucination

These hallucinations occur just before falling asleep, and affect a surprisingly high proportion of the population (in one survey 37% of the respondents experienced them twice a week [24]). The hallucinations can last from seconds to minutes, all the while the subject usually remains aware of the true nature of the images. These may be associated with narcolepsy. Hypnagogic hallucinations are sometimes associated with brainstem abnormalities, but this is rare.[25]

Peduncular hallucinosis

Peduncular means pertaining to the peduncle, which is a neural tract running to and from the pons on the brain stem. These hallucinations usually occur in the evenings, but not during drowsiness, as in the case of hypnagogic hallucination. The subject is usually fully conscious and then can interact with the hallucinatory characters for extended periods of time. As in the case of hypnagogic hallucinations, insight into the nature of the images remains intact. The false images can occur in any part of the visual field, and are rarely polymodal.[25]

Delirium tremens

One of the more enigmatic forms of visual hallucination is the highly variable, possibly polymodal delirium tremens. Individuals suffering from delirium tremens may be agitated and confused, especially in the later stages of this disease. Insight is gradually reduced with the progression of this disorder. Sleep is disturbed and occurs for a shorter period of time, with rapid eye movement sleep.

Parkinson's disease and Lewy body dementia

Parkinson's disease is linked with Lewy body dementia for their similar hallucinatory symptoms. The symptoms strike during the evening in any part of the visual field, and are rarely polymodal. The segue into hallucination may begin with illusions[26] where sensory perception is greatly distorted, but no novel sensory information is present. These typically last for several minutes, during which time the subject may be either conscious and normal or drowsy/inaccessible. Insight into these hallucinations is usually preserved and REM sleep is usually reduced. Parkinson's disease is usually associated with a degraded substantia nigra pars compacta, but recent evidence suggests that PD affects a number of sites in the brain. Some places of noted degradation include the median raphe nuclei, the noradrenergic parts of the locus coeruleus, and the cholinergic neurons in the parabrachial and pedunculopontine nuclei of the tegmentum.[25]

Migraine coma

This type of hallucination is usually experienced during the recovery from a comatose state. The migraine coma can last for up to two days, and a state of depression is sometimes comorbid. The hallucinations occur during states of full consciousness, and insight into the hallucinatory nature of the images is preserved. It has been noted that ataxic lesions accompany the migraine coma.[25]

Charles Bonnet syndrome

Charles Bonnet syndrome is the name given to visual hallucinations experienced by blind patients. The hallucinations can usually be dispersed by opening or closing the eyelids until the visual images disappear. The hallucinations usually occur during the morning or evening, but are not dependent on low light conditions. These prolonged hallucinations usually do not disturb the patients very much, as they are aware that they are hallucinating.[25] A differential diagnosis are opthalmopathic hallucinations.[27]

Focal epilepsy

Visual hallucinations due to focal seizures differ depending on the region of the brain where the seizure occurs. For example, visual hallucinations during occipital lobe seizures are typically visions of brightly colored, geometric shapes that may move across the visual field, multiply, or form concentric rings and generally persist from a few seconds to a few minutes. They are usually unilateral and localized to one part of the visual field on the ipsilateral side of the seizure focus, typically the temporal field. However, unilateral visions moving horizontally across the visual field begin on the contralateral side and move towards the ipsilateral side.[21]

Temporal lobe seizures, on the other hand, can produce complex visual hallucinations of people, scenes, animals, and more as well as distortions of visual perception. Complex hallucinations may appear real or unreal, may or may not be distorted with respect to size, and may seem disturbing or affable, among other variables. One rare but notable type of hallucination is heautoscopy, a hallucination of a mirror image of one's self. These "other selves" may be perfectly still or performing complex tasks, may be an image of a younger self or the present self, and tend to be only briefly present. Complex hallucinations are a relatively uncommon finding in temporal lobe epilepsy patients. Rarely, they may occur during occipital focal seizures or in parietal lobe seizures.[21]

Distortions in visual perception during a temporal lobe seizure may include size distortion (micropsia or macropsia), distorted perception of movement (where moving objects may appear to be moving very slowly or to be perfectly still), a sense that surfaces such as ceilings and even entire horizons are moving farther away in a fashion similar to the dolly zoom effect, and other illusions.[28] Even when consciousness is impaired, insight into the hallucination or illusion is typically preserved.

Drug-induced hallucination

Drug Induced Hallucinations are hallucinations that are caused by the consumption of psychoactive substances such as deliriants. Along with deliriants, psychedelics, certain stimulants and opiates; these are also known to cause hallucinations that can be experienced through both visual and auditory. Some psychedelics such as Lysergic acid diethylamide, cannabis, and psilocybin can cause hallucinations that range from a spectrum of mild to severe and can cause brain damage that can also be a precursor to mental illnesses and psychotic disorders. Opiates are also a popular drug used to induce hallucinations, especially in larger doses. This as well as other drugs that induce hallucinations can cause severe mental and physical damage. Some of the most common opiates are opium are heroin and morphine. Despite the addictive properties of some of these drugs (mainly opiates and mental dependencies in some cannabis users); Other drugs can be used in psychotherapy to treat mental disorders, addiction and even experiments. [29]

Sensory deprivation hallucination

Hallucinations can be caused by sense deprivation when it occurs for prolonged periods of time, and almost always occur in the modality being deprived (visual for blindfolded/darkness, auditory for muffled conditions, etc.)

Experimentally-induced hallucinations

Main article : Hallucinations in the sane

Pathophysiology

VISUAL

Sometimes internal imagery can overwhelm the sensory input from external stimuli when sharing neural pathways, or if indistinct stimuli is perceived and manipulated to match one's expectations or beliefs, especially about the environment. This can result in a hallucination,[30] and this effect is sometimes exploited to form an optical illusion.

There are 3 pathophysiologic mechanisms thought to account for complex visual hallucinations theses mechanisms consist of the following:

The first mechanism involves irritation of cortical centers responsible for visual processing (e.g., seizure activity). The irritation of the primary visual cortex causes simple elementary visual hallucinations.

The second mechanism involves lesions that cause deafferentation of the visual system may lead to cortical release phenomenon, which includes visual hallucination.

The third mechanism is the reticular activating system, which has been linked to the genesis of visual hallucinations.[31]

Some specific classifications include: elementary hallucinations, which may entail flicks, specks, and bars of light (called phosphenes). Closed eye hallucinations in darkness, which are common to psychedelic drugs (i.e., LSD, mescaline). Scenic or "panoramic" hallucinations, which are not superimposed but vividly replace the entire visual field with hallucinatory content similarly to dreams;[32] such scenic hallucinations may occur in epilepsy[33] (in which they are usually stereotyped and experimental[34] in character), hallucinogen use,[32] and more rarely in catatonic schizophrenia[35][36] (cf. oneirophrenia), mania,[37] and brainstem lesions,[38] amongst others.

Another thing that may cause visual hallucinations is prolonged visual deprivation. Which a study was done where 13 healthy people were blindfolded for a period of 5 days and 10 out of the 13 subjects reported visual hallucinations. This finding lends strong support to the idea that the simple loss of normal visual input is sufficient to cause visual hallucinations.[31]

PSYCHODYNAMIC FACTS

Various theories have been put forward to explain the occurrence of hallucinations. When psychodynamic (Freudian) theories were popular in psychology, hallucinations were seen as a projection of unconscious wishes, thoughts and wants. As biological theories have become orthodox, hallucinations are more often thought of (by psychologists at least) as being caused by functional deficits in the brain. With reference to mental illness, the function (or dysfunction) of the neurotransmitters glutamate and dopamine are thought to be particularly important.[39] The Freudian interpretation may have an aspect of truth, as the biological hypothesis explains the physical interactions in the brain, while the Freudian deals with the origin of the theme of the hallucination. Psychological research has argued that hallucinations may result from biases in what are known as metacognitive abilities.[40]

INFORMATION PROCESSING PERSPECTIVE

These are abilities that allow us to monitor or draw inferences from our own internal psychological states (such as intentions, memories, beliefs and thoughts). The ability to discriminate between internal (self-generated) and external (stimuli) sources of information is considered to be an important metacognitive skill, but one which may break down to cause hallucinatory experiences. Projection of an internal state (or a person's own reaction to another's) may arise in the form of hallucinations, especially auditory hallucinations. A recent hypothesis that is gaining acceptance concerns the role of overactive top-down processing, or strong perceptual expectations, that can generate spontaneous perceptual output (that is, hallucination).[41]

Stages of Hallucination

1. Emergence of surprising or warded-off memory or fantasy images[42]
2. Frequent reality checks[42]
3. Last vestige of insight as hallucinations become "real"[42]
4. Fantasy and distortion elaborated upon and confused with actual perception[42]
5. Internal-external boundaries destroyed and possible pantheistic (or personally felt or believed, possibly profound, internal spiritual or religious) experience[42]

BIOLOGICAL PERSPECTIVE

VISUAL The most common modality referred to when people speak of hallucinations. These include the phenomena of seeing things which are not present or visual perception which does not reconcile with the physical, consensus reality. There are many different causes, which have been classed as psychophysiologic (a disturbance of brain structure), psychobiochemical (a disturbance of neurotransmitters), psychodynamic (an emergence of the unconscious into consciousness), and psychological (e.g. meaningful experiences consciousness), this is also the case in Alzheimer's disease. Numerous disorders can involve visual hallucinations, ranging from psychotic disorders to dementia to migraine, but experiencing visual hallucinations does not in itself mean there is necessarily a disorder. Visual hallucinations are associated with organic disorders of the brain and with drug and alcohol related illness,[43] and not typically considered the result of a psychiatric disorder.[44]

SCHIZOPHRENIC HALLUCINATION Hallucinations caused by schizophrenia. Schizophrenia is when one is unable to tell the difference between real and unreal experiences, accompanied by the inability to think logically, have contextually appropriate emotions, and to function in social situations.[45] Scientifically reviewed. 21 October 2012. Web.</ref> It has been found that when one experiences a hallucination induced by Schizophrenia, there are many abnormalities that are going on in the brain; Particularly in the region that processes voices in sounds (for those who experience auditory hallucinations) and visual processing. (visual hallucinations). According to studies and experiments conducted by researchers, it was seen that a possible cause for these hallucinations were abnormalities in gray matter and general functioning that combines interpreting sounds, voices and visuals, as well as regulating emotions.[46]

NEUROANATOMICAL CORRELATES Normal everyday procedures like getting an MRI (Magnetic Resonance Imaging) have been used to find out more about auditory and verbal hallucinations. "Functional magnetic resonance imaging (fMRI) and repetitive transcranial magnetic stimulation (rTMS) were used to explore the pathophysiology of auditory/verbal hallucinations (AVHs)"[47] Throughout the exploring through MRI's of patients,there were "lower levels of hallucination-related activation in Broca's area strongly predicted greater rate of response to left temporoparietal rTMS."[47] What these findings could suggest is that "dominant hemisphere temporoparietal areas are involved in expressing AVHs, with higher levels of coactivation and/or coupling involving inferior frontal regions reinforcing underlying pathophysiology."[47]

Also through fMRI's, it is found that there can be better understandings on why hallucinations happen in the brain, by understanding emotion's and cognition and how it can prompt physical reactions that can help result in a hallucination. It suggests the theory that "motivations in the body and mind can drive us to certain behaviors that we act in, such as survival instinct and intuition" and that they can work in a hand in hand like fashion. It can also be viewed as a symbolic "homeostasis" that can have adverse effects by having these hallucinations and / or mental illnesses. The amygdlada has also been seen to relate to this finding by contributing a "declarative judgement of emotional salience" as well as affecting both "efferent and afferent representational levels of affective autonomic responses in the brain". [48]

PATHOPHYSIOLOGICAL MECHANISMS "The left superior temporal cortex, which supports linguistic functions, has consistently been reported to activate during auditory–verbal hallucinations in schizophrenia patients"[49] The Charles Bonnet Syndrome supports the visual cortex cortex deafferentiation proposal. There is irritation in the visual cortex when hallucination occur, which could suggest why it is reported that images that are not real are seen. Although many sufferers of the Charles Bonnet Syndrome are elderly, it can occur in anyone.[50] The reticular activation system can be used to support the neurotransmitters (dopamine and norepinephrine) effect on hallucinations.

Treatments

There are few treatments for many types of hallucinations. However, for those hallucinations caused by mental disease, a psychologist or psychiatrist should be alerted, and treatment will be based on the observations of those doctors. Antipsychotic and atypical antipsychotic medication may also be utilized to treat the illness if the symptoms are severe and cause significant distress. For other causes of hallucinations there is no factual evidence to support any one treatment is scientifically tested and proven. However, abstaining from hallucinogenic drugs, managing stress levels, living healthily, and getting plenty of sleep can help reduce the prevalence of hallucinations. In all cases of hallucinations, medical attention should be sought out and informed of one's specific symptoms.

Epidemiology

One study from as early as 1895[51] reported that approximately 10% of the population experienced hallucinations. A 1996-1999 survey of over 13,000 people[52] reported a much higher figure, with almost 39% of people reporting hallucinatory experiences, 27% of which were daytime hallucinations, mostly outside the context of illness or drug use. From this survey, olfactory (smell) and gustatory (taste) hallucinations seem the most common in the general population.

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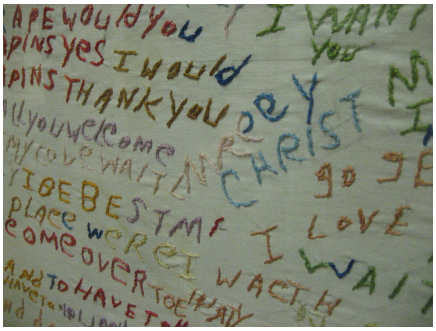
External links

- Hearing Voices Network (http://www.hearing-voices.org/)
- "Anthropology and Hallucinations" (http://www.psychanalyse-paris.com/843-Anthropology-and.html), chapter from *The Making of Religion*
- "The voice inside: A practical guide to coping with hearing voices" (http://www.mind.org.uk/help/medical_and_alternative_care/the_voice_inside_coping_with_hearing_voices)
- Psychology Terms (http://dictionary-psychology.com)

Schizophrenia

Schizophrenia	
Classification and external resources	
 Cloth embroidered by a patient diagnosed with schizophrenia	
ICD-10	F20 [1]
ICD-9	295 [2]
OMIM	181500 [3]
DiseasesDB	11890 [4]
MedlinePlus	000928 [5]
eMedicine	med/2072 [6] emerg/520 [7]
MeSH	*F03.700.750* [8]

Schizophrenia (/ˌskɪtsəˈfrɛniə/ or /ˌskɪtsəˈfriːniə/) is a mental disorder characterized by a breakdown of thought processes and by poor emotional responsiveness.[9] Common symptoms include auditory hallucinations, paranoid or bizarre delusions, or disorganized speech and thinking, and it is accompanied by significant social or occupational dysfunction. The onset of symptoms typically occurs in young adulthood, with a global lifetime prevalence of about 0.3–0.7%.[10] Diagnosis is based on observed behavior and the patient's reported experiences.

Genetics, early environment, neurobiology, and psychological and social processes appear to be important contributory factors; some recreational and prescription drugs appear to cause or worsen symptoms. Current research is focused on the role of neurobiology, although no single isolated organic cause has been found. The many possible combinations of symptoms have triggered debate about whether the diagnosis represents a single disorder or a number of discrete syndromes. Despite the etymology of the term from the Greek roots *skhizein* (*σχίζειν*, "to split") and *phrēn, phren-* (*φρήν, φρεν-*; "mind"), schizophrenia does not imply a "split personality", or "multiple personality disorder" (which is known these days as dissociative identity disorder)—a condition with which it is often confused in public perception.[11] Rather, the term means a "splitting of mental functions", because of the symptomatic presentation of the illness.

The mainstay of treatment is antipsychotic medication, which primarily suppresses dopamine (and sometimes serotonin) receptor activity. Psychotherapy and vocational and social rehabilitation are also important in treatment. In more serious cases—where there is risk to self and others—involuntary hospitalization may be necessary, although hospital stays are now shorter and less frequent than they once were.[12]

The disorder is thought mainly to affect cognition, but it also usually contributes to chronic problems with behavior and emotion. People with schizophrenia are likely to have additional (comorbid) conditions, including major depression and anxiety disorders; the lifetime occurrence of substance abuse is almost 50%.[13] Social problems, such as long-term unemployment, poverty, and homelessness are common. The average life expectancy of people with the disorder is 12 to 15 years less than those without, the result of increased physical health problems and a higher suicide rate (about 5%).[10]

Symptoms

A person diagnosed with schizophrenia may experience hallucinations (most reported are hearing voices), delusions (often bizarre or persecutory in nature), and disorganized thinking and speech. The latter may range from loss of train of thought, to sentences only loosely connected in meaning, to incoherence known as word salad in severe cases. Social withdrawal, sloppiness of dress and hygiene, and loss of motivation and judgment are all common in schizophrenia.[14] There is often an observable pattern of emotional difficulty, for example lack of responsiveness.[15] Impairment in social cognition is associated with schizophrenia,[16] as are symptoms of paranoia; social isolation commonly occurs.[17] Difficulties in working and long-term memory, attention, executive functioning, and speed of processing also commonly occur.[10] In one uncommon subtype, the person may be largely mute, remain motionless in bizarre postures, or exhibit purposeless agitation, all signs of catatonia.[18]

Late adolescence and early adulthood are peak periods for the onset of schizophrenia,[10] critical years in a young adult's social and vocational development.[19] In 40% of men and 23% of women diagnosed with schizophrenia, the condition manifested itself before the age of 19.[20] To minimize the developmental disruption associated with schizophrenia, much work has recently been done to identify and treat the prodromal (pre-onset) phase of the illness, which has been detected up to 30 months before the onset of symptoms.[19] Those who go on to develop schizophrenia may experience transient or self-limiting psychotic symptoms[21] and the non-specific symptoms of social withdrawal, irritability, dysphoria,[22] and clumsiness[23] during the prodromal phase.

Schneiderian classification

In the early 20th century, the psychiatrist Kurt Schneider listed the forms of psychotic symptoms that he thought distinguished schizophrenia from other psychotic disorders. These are called *first-rank symptoms* or Schneider's first-rank symptoms. They include delusions of being controlled by an external force; the belief that thoughts are being inserted into or withdrawn from one's conscious mind; the belief that one's thoughts are being broadcast to other people; and hearing hallucinatory voices that comment on one's thoughts or actions or that have a conversation with other hallucinated voices.[24] Although they have significantly contributed to the current diagnostic criteria, the specificity of first-rank symptoms has been questioned. A review of the diagnostic studies conducted between 1970 and 2005 found that they allow neither a reconfirmation nor a rejection of Schneider's claims, and suggested that first-rank symptoms should be de-emphasized in future revisions of diagnostic systems.[25]

Positive and negative symptoms

Schizophrenia is often described in terms of positive and negative (or deficit) symptoms.[26] Positive symptoms are those that most individuals do not normally experience but are present in people with schizophrenia. They can include delusions, disordered thoughts and speech, and tactile, auditory, visual, olfactory and gustatory hallucinations, typically regarded as manifestations of psychosis.[27] Hallucinations are also typically related to the content of the delusional theme.[28] Positive symptoms generally respond well to medication.[28] Negative symptoms are deficits of normal emotional responses or of other thought processes, and respond less well to medication.[14] They commonly include flat or blunted affect and emotion, poverty of speech (alogia), inability to experience pleasure (anhedonia), lack of desire to form relationships (asociality), and lack of motivation (avolition). Research suggests that negative symptoms contribute more to poor quality of life, functional disability, and the burden on

others than do positive symptoms.[29] People with prominent negative symptoms often have a history of poor adjustment before the onset of illness, and response to medication is often limited.[14][30]

Causes

A combination of genetic and environmental factors play a role in the development of schizophrenia.[10][11] People with a family history of schizophrenia who suffer a transient psychosis have a 20–40% chance of being diagnosed one year later.[31]

Genetic

Estimates of heritability vary because of the difficulty in separating the effects of genetics and the environment.[32] The greatest risk for developing schizophrenia is having a first-degree relative with the disease (risk is 6.5%); more than 40% of monozygotic twins of those with schizophrenia are also affected.[11] It is likely that many genes are involved, each of small effect and unknown transmission and expression.[11] Many possible candidates have been proposed, including specific copy number variations, NOTCH4, and histone protein loci.[33] A number of genome-wide associations such as zinc finger protein 804A have also been linked.[34] There appears to be significant overlap in the genetics of schizophrenia and bipolar disorder.[35]

Assuming a hereditary basis, one question from evolutionary psychology is why genes that increase the likelihood of psychosis evolved, assuming the condition would have been maladaptive from an evolutionary point of view. One idea is that genes are involved in the evolution of language and human nature, but to date such ideas remain little more than hypothetical in nature.[36][37]

Environment

Environmental factors associated with the development of schizophrenia include the living environment, drug use and prenatal stressors.[10] Parenting style seems to have no major effect, although people with supportive parents do better than those with critical or hostile parents.[11] Living in an urban environment during childhood or as an adult has consistently been found to increase the risk of schizophrenia by a factor of two,[10][11] even after taking into account drug use, ethnic group, and size of social group.[38] Other factors that play an important role include social isolation and immigration related to social adversity, racial discrimination, family dysfunction, unemployment, and poor housing conditions.[11][39]

Drug use

Amphetamine, cocaine, and to a lesser extent alcohol, can result in psychosis that presents very similarly to schizophrenia.[11][40] Although not generally believed to be a cause of the illness, people with schizophrenia use nicotine at much greater rates than the general population.[41] About half of those with schizophrenia use drugs or alcohol excessively.[42] Evidence supports a link between earlier onset of psychotic illness and cannabis use; alcohol use is not associated with an earlier onset of psychosis.[43] Other drugs may be used only as coping mechanisms by individuals who have schizophrenia to deal with depression, anxiety, boredom, and loneliness.[42][44] There is evidence that alcohol abuse via a kindling mechanism can occasionally cause the development of a chronic substance induced psychotic disorder, i.e. schizophrenia.[45] A small number of people withdrawing from benzodiazepines experience a severe protracted withdrawal syndrome which can resemble schizophrenia and be misdiagnosed as such.[46] The more often cannabis is abused, the more likely a person is to develop a psychotic illness,[47] with frequent use being correlated with twice the risk of psychosis and schizophrenia.[48] Whether cannabis use is a contributory cause of schizophrenia, rather than a behavior that does not actually cause the disease, remains controversial.[33][49]

Developmental factors

Factors such as hypoxia and infection, or stress and malnutrition in the mother during fetal development, may result in a slight increase in the risk of schizophrenia later in life.[10] People diagnosed with schizophrenia are more likely to have been born in winter or spring (at least in the northern hemisphere), which may be a result of increased rates of viral exposures in utero.[11] This difference is about 5 to 8%.[50]

Mechanisms

A number of attempts have been made to explain the link between altered brain function and schizophrenia.[10] One of the most common is the dopamine hypothesis, which attributes psychosis to the mind's faulty interpretation of the misfiring of dopaminergic neurons.[10]

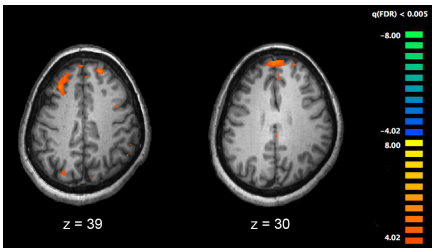
Psychological

Many psychological mechanisms have been implicated in the development and maintenance of schizophrenia. Cognitive biases have been identified in those with the diagnosis or those at risk, especially when under stress or in confusing situations.[51] Some cognitive features may reflect global neurocognitive deficits such as memory loss, while others may be related to particular issues and experiences.[52][53]

Despite a demonstrated appearance of blunted affect, recent findings indicate that many individuals diagnosed with schizophrenia are emotionally responsive, particularly to stressful or negative stimuli, and that such sensitivity may cause vulnerability to symptoms or to the disorder.[54][55] Some evidence suggests that the content of delusional beliefs and psychotic experiences can reflect emotional causes of the disorder, and that how a person interprets such experiences can influence symptomatology.[56][57][58] The use of "safety behaviors" to avoid imagined threats may contribute to the chronicity of delusions.[59] Further evidence for the role of psychological mechanisms comes from the effects of psychotherapies on symptoms of schizophrenia.[60]

Neurological

Schizophrenia is associated with subtle differences in brain structures, found in 40 to 50% of cases, and in brain chemistry during acute psychotic states.[10] Studies using neuropsychological tests and brain imaging technologies such as fMRI and PET to examine functional differences in brain activity have shown that differences seem to most commonly occur in the frontal lobes, hippocampus and temporal lobes.[61] Reductions in brain volume, smaller than those found in Alzheimer's disease, have been reported in areas of the frontal cortex and temporal lobes. It is uncertain whether these volumetric changes are progressive or preexist prior to the onset of the disease.[62] These differences have been linked to the neurocognitive deficits often associated with schizophrenia.[63] Because neural circuits are altered, it has alternatively been suggested that schizophrenia should be thought of as a collection of neurodevelopmental disorders.[64]



Functional magnetic resonance imaging (fMRI) and other brain imaging technologies allow for the study of differences in brain activity in people diagnosed with schizophrenia. The image shows two levels of the brain, with areas that were more active in healthy controls than in schizophrenia patients shown in orange, during an fMRI study of working memory.

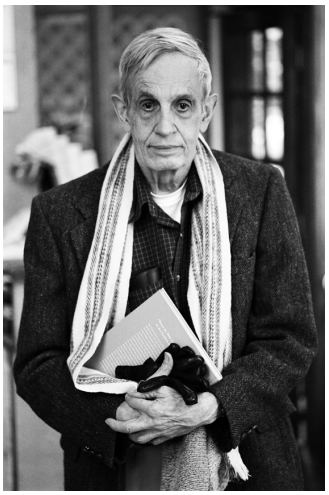
Particular attention has been paid to the function of dopamine in the mesolimbic pathway of the brain. This focus largely resulted from the accidental finding that phenothiazine drugs, which block dopamine function, could reduce psychotic symptoms. It is also supported by the fact that amphetamines, which trigger the release of dopamine, may exacerbate the psychotic symptoms in schizophrenia.[65] The influential dopamine hypothesis of schizophrenia proposed that excessive activation of D_2 receptors was the cause of (the positive symptoms of) schizophrenia. Although postulated for about 20 years based on the D_2 blockade effect common to all antipsychotics, it was not

until the mid-1990s that PET and SPET imaging studies provided supporting evidence. The dopamine hypothesis is now thought to be simplistic, partly because newer antipsychotic medication (atypical antipsychotic medication) can be just as effective as older medication (typical antipsychotic medication), but also affects serotonin function and may have slightly less of a dopamine blocking effect.[66]

Interest has also focused on the neurotransmitter glutamate and the reduced function of the NMDA glutamate receptor in schizophrenia, largely because of the abnormally low levels of glutamate receptors found in the postmortem brains of those diagnosed with schizophrenia,[67] and the discovery that glutamate-blocking drugs such as phencyclidine and ketamine can mimic the symptoms and cognitive problems associated with the condition.[68] Reduced glutamate function is linked to poor performance on tests requiring frontal lobe and hippocampal function, and glutamate can affect dopamine function, both of which have been implicated in schizophrenia, have suggested an important mediating (and possibly causal) role of glutamate pathways in the condition.[69] But positive symptoms fail to respond to glutamatergic medication.[70]

Diagnosis

Schizophrenia is diagnosed based on criteria in either the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders*, version DSM-IV-TR, or the World Health Organization's International Statistical Classification of Diseases and Related Health Problems, the ICD-10.[10] These criteria use the self-reported experiences of the person and reported abnormalities in behavior, followed by a clinical assessment by a mental health professional. Symptoms associated with schizophrenia occur along a continuum in the population and must reach a certain severity before a diagnosis is made.[11] As of 2009 there is no objective test.[10]



John Nash, a U.S. mathematician and joint winner of the 1994 Nobel Prize for Economics, suffered from schizophrenia. His life has been the subject of the 2001 Academy Award-winning film *A Beautiful Mind*.

Criteria

The ICD-10 criteria are typically used in European countries, while the DSM-IV-TR criteria are used in the United States and to varying degrees around the world, and are prevailing in research studies. The ICD-10 criteria put more emphasis on Schneiderian first-rank symptoms. In practice, agreement between the two systems is high.[71]

According to the revised fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR), to be diagnosed with schizophrenia, three diagnostic criteria must be met:[72]

1. Characteristic symptoms: Two or more of the following, each present for much of the time during a one-month period (or less, if symptoms remitted with treatment).
   - Delusions
   - Hallucinations
   - Disorganized speech, which is a manifestation of formal thought disorder
   - Grossly disorganized behavior (e.g. dressing inappropriately, crying frequently) or catatonic behavior
   - Negative symptoms: Blunted affect (lack or decline in emotional response), alogia (lack or decline in speech), or avolition (lack or decline in motivation)

     If the delusions are judged to be bizarre, or hallucinations consist of hearing one voice participating in a running commentary of the patient's actions or of hearing two or more voices conversing with each other, only that symptom is required above. The speech disorganization criterion is only met if it is severe enough to substantially impair communication.

2. Social or occupational dysfunction: For a significant portion of the time since the onset of the disturbance, one or more major areas of functioning such as work, interpersonal relations, or self-care, are markedly below the level achieved prior to the onset.
3. Significant duration: Continuous signs of the disturbance persist for at least six months. This six-month period must include at least one month of symptoms (or less, if symptoms remitted with treatment).

If signs of disturbance are present for more than a month but less than six months, the diagnosis of schizophreniform disorder is applied.[72] Psychotic symptoms lasting less than a month may be diagnosed as brief psychotic disorder, and various conditions may be classed as psychotic disorder not otherwise specified. Schizophrenia cannot be diagnosed if symptoms of mood disorder are substantially present (although schizoaffective disorder could be diagnosed), or if symptoms of pervasive developmental disorder are present unless prominent delusions or hallucinations are also present, or if the symptoms are the direct physiological result of a general medical condition or a substance, such as abuse of a drug or medication.

Subtypes

The DSM-IV-TR contains five sub-classifications of schizophrenia, although the developers of DSM-5 are recommending they be dropped from the new classification:[73][74]

- Paranoid type: Delusions or auditory hallucinations are present, but thought disorder, disorganized behavior, or affective flattening are not. Delusions are persecutory and/or grandiose, but in addition to these, other themes such as jealousy, religiosity, or somatization may also be present. (DSM code 295.3/ICD code F20.0)
- Disorganized type: Named *hebephrenic schizophrenia* in the ICD. Where thought disorder and flat affect are present together. (DSM code 295.1/ICD code F20.1)
- Catatonic type: The subject may be almost immobile or exhibit agitated, purposeless movement. Symptoms can include catatonic stupor and waxy flexibility. (DSM code 295.2/ICD code F20.2)
- Undifferentiated type: Psychotic symptoms are present but the criteria for paranoid, disorganized, or catatonic types have not been met. (DSM code 295.9/ICD code F20.3)
- Residual type: Where positive symptoms are present at a low intensity only. (DSM code 295.6/ICD code F20.5)

The ICD-10 defines two additional subtypes:[74]

- Post-schizophrenic depression: A depressive episode arising in the aftermath of a schizophrenic illness where some low-level schizophrenic symptoms may still be present. (ICD code F20.4)
- Simple schizophrenia: Insidious and progressive development of prominent negative symptoms with no history of psychotic episodes. (ICD code F20.6)

Differential

Psychotic symptoms may be present in several other mental disorders, including bipolar disorder,[75] borderline personality disorder,[76] drug intoxication and drug-induced psychosis. Delusions ("non-bizarre") are also present in delusional disorder, and social withdrawal in social anxiety disorder, avoidant personality disorder and schizotypal personality disorder. Schizophrenia is comorbid with obsessive-compulsive disorder (OCD) considerably more often than could be explained by pure chance, although it can be difficult to distinguish obsessions that occur in OCD from the delusions of schizophrenia.[77]

A more general medical and neurological examination may be needed to rule out medical illnesses which may rarely produce psychotic schizophrenia-like symptoms,[72] such as metabolic disturbance, systemic infection, syphilis, HIV infection, epilepsy, and brain lesions. It may be necessary to rule out a delirium, which can be distinguished by visual hallucinations, acute onset and fluctuating level of consciousness, and indicates an underlying medical illness. Investigations are not generally repeated for relapse unless there is a specific *medical* indication or possible adverse effects from antipsychotic medication.

Prevention

Prevention of schizophrenia is difficult as there are no reliable markers for the later development of the disease.[78] The evidence for the effectiveness of early interventions to prevent schizophrenia is inconclusive.[79] While there is some evidence that early intervention in those with a psychotic episode may improve short term outcomes, there is little benefit from these measures after five years.[10] Attempting to prevent schizophrenia in the prodrome phase is of uncertain benefit and therefore as of 2009 is not recommended.[80]

Management

The primary treatment of schizophrenia is antipsychotic medications, often in combination with psychological and social supports.[10] Hospitalization may occur for severe episodes either voluntarily or (if mental health legislation allows it) involuntarily. Long-term hospitalization is uncommon since deinstitutionalization beginning in the 1950s, although it still occurs.[12] Community support services including drop-in centers, visits by members of a community mental health team, supported employment[81] and support groups are common. Some evidence indicates that regular exercise has a positive effect on the physical and mental health of those with schizophrenia.[82]

Medication

Risperidone (trade name Risperdal) is a common atypical antipsychotic medication.

The first-line psychiatric treatment for schizophrenia is antipsychotic medication,[83] which can reduce the positive symptoms of psychosis in about 7–14 days. Antipsychotics, however, fail to significantly ameliorate the negative symptoms and cognitive dysfunction.[30][84] Long term use decreases the risk of relapse.[85]

The choice of which antipsychotic to use is based on benefits, risks, and costs.[10] It is debatable whether, as a class, typical or atypical antipsychotics are better.[86][87] Both have equal drop-out and symptom relapse rates when typicals are used at low to moderate dosages.[88] There is a good response in 40–50%, a partial response in 30–40%, and treatment resistance (failure of symptoms to respond satisfactorily after six weeks to two or three different antipsychotics) in 20% of people.[30] Clozapine is an effective treatment for those who respond poorly to other drugs, but it has the potentially serious side effect of agranulocytosis (lowered white blood cell count) in less than 4% of patients.[10][11][89]

With respect to side effects typical antipsychotics are associated with a higher rate of extrapyramidal side effects while atypicals are associated with considerable weight gain, diabetes and risk of metabolic syndrome.[88] While atypicals have fewer extrapyramidal side effects these differences are modest.[90] Some atypicals such as quetiapine and risperidone are associated with a higher risk of death compared to the typical antipsychotic perphenazine, while clozapine is associated with the lowest risk of death.[91] It remains unclear whether the newer antipsychotics reduce the chances of developing neuroleptic malignant syndrome, a rare but serious neurological disorder.[92]

For people who are unwilling or unable to take medication regularly, long-acting depot preparations of antipsychotics may be used to achieve control.[93] They reduce the risk of relapse to a greater degree than oral medications.[85] When used in combination with psychosocial interventions they may improve long-term adherence to treatment.[93]

Psychosocial

A number of psychosocial interventions may be useful in the treatment of schizophrenia including: family therapy,[94] assertive community treatment, supported employment, cognitive remediation,[95] skills training, cognitive behavioral therapy (CBT), token economic interventions, and psychosocial interventions for substance use and weight management.[96] Family therapy or education, which addresses the whole family system of an individual, may reduce relapses and hospitalizations.[94] The evidence for CBT's effectiveness in either reducing symptoms or preventing relapse is minimal.[97][98] Art or drama therapy have not been well-researched.[99][100]

Prognosis

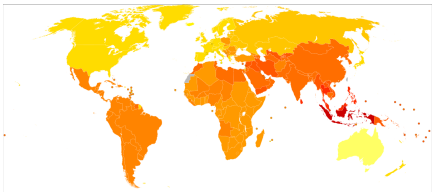
Schizophrenia has great human and economic costs.[10] It results in a decreased life expectancy of 12–15 years, primarily because of its association with obesity, sedentary lifestyles, and smoking, with an increased rate of suicide playing a lesser role.[10] These differences in life expectancy increased between the 1970s and 1990s,[101] and between the 1990s and first decade of the 21st century did not change substantially in a health system with open access to care (Finland).[91]

Schizophrenia is a major cause of disability, with active psychosis ranked as the third-most-disabling condition after quadriplegia and dementia and ahead of paraplegia and blindness.[102] Approximately three-fourths of people with schizophrenia have ongoing disability with relapses[30] and 16.7 million people globally are deemed to have moderate or severe disability from the condition.[103] Some people do recover completely and others function well in society.[104] Most people with schizophrenia live independently with community support.[10] In people with a first episode of psychosis a good long-term outcome occurs in 42%, an intermediate outcome in 35% and a poor outcome in 27%.[105] Outcomes for schizophrenia appear better in the developing than the developed world.[106] These conclusions, however, have been questioned.[107][108]

There is a higher than average suicide rate associated with schizophrenia. This has been cited at 10%, but a more recent analysis of studies and statistics revises the estimate to 4.9%, most often occurring in the period following onset or first hospital admission.[109] Several times more (20 to 40%) attempt suicide at least once.[110][111] There are a variety of risk factors, including male gender, depression, and a high intelligence quotient.[110]

Schizophrenia and smoking have shown a strong association in studies world-wide.[112][113] Use of cigarettes is especially high in individuals diagnosed with schizophrenia, with estimates ranging from 80% to 90% being regular smokers, as compared to 20% of the general population.[113] Those who smoke tend to smoke heavily, and additionally smoke cigarettes with high nicotine content.[111] Some evidence suggests that paranoid schizophrenia may have a better prospect than other types of schizophrenia for independent living and occupational functioning.[114]

Epidemiology

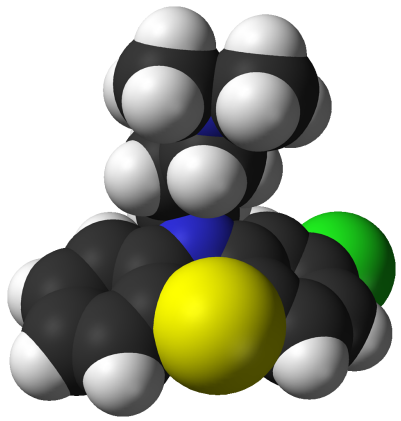


Disability-adjusted life year for schizophrenia per 100,000 inhabitants in 2004.

Schizophrenia affects around 0.3–0.7% of people at some point in their life,[10] or 24 million people worldwide as of 2011.[115] It occurs 1.4 times more frequently in males than females and typically appears earlier in men[11]—the peak ages of onset are 20–28 years for males and 26–32 years for females.[116] Onset in childhood is much rarer,[117] as is onset in middle- or old age.[118] Despite the received wisdom that schizophrenia occurs at similar rates worldwide, its prevalence varies across the world,[119] within countries,[120] and at the local and neighborhood level.[121] It causes approximately 1% of worldwide disability adjusted life years.[11] The rate of schizophrenia varies up to threefold depending on how it is defined.[10]

History

Accounts of a schizophrenia-like syndrome are thought to be rare in the historical record before the 19th century, although reports of irrational, unintelligible, or uncontrolled behavior were common. A detailed case report in 1797 concerning James Tilly Matthews, and accounts by Phillipe Pinel published in 1809, are often regarded as the earliest cases of the illness in the medical and psychiatric literature.[122] The term *dementia praecox* was used in 1891 by Arnold Pick in a case report of a psychotic disorder. In 1893 Emil Kraepelin introduced a broad new distinction in the classification of mental disorders between *dementia praecox* and mood disorder (termed manic depression and including both unipolar and bipolar depression). Kraepelin believed that *dementia praecox* was primarily a disease of the brain,[123] and particularly a form of dementia, distinguished from other forms of dementia such as Alzheimer's disease which typically occur later in life.[124] It is sometimes argued that the use of the term *démence précoce* in 1852 by the French physician Bénédict Morel constitutes the medical discovery of schizophrenia. However this account ignores the fact that there is little to connect Morel's descriptive use of the term and the independent development of the *dementia praecox* disease concept at the end of the nineteenth-century.[125]



Molecule of chlorpromazine (trade name Thorazine), which revolutionized treatment of schizophrenia in the 1950s

The word *schizophrenia*—which translates roughly as "splitting of the mind" and comes from the Greek roots *schizein* (σχίζειν, "to split") and *phrēn*, *phren-* (φρήν, φρεν-, "mind")[126]—was coined by Eugen Bleuler in 1908 and was intended to describe the separation of function between personality, thinking, memory, and perception. Bleuler described the main symptoms as 4 *A*'s: flattened *Affect*, *Autism*, impaired *Association* of ideas and *Ambivalence*.[127] Bleuler realized that the illness was not a dementia, as some of his patients improved rather than deteriorated, and thus proposed the term schizophrenia instead. Treatment was revolutionized in the mid-1950s with the development and introduction of chlorpromazine.[128]

In the early 1970s, the diagnostic criteria for schizophrenia were the subject of a number of controversies which eventually led to the operational criteria used today. It became clear after the 1971 US-UK Diagnostic Study that schizophrenia was diagnosed to a far greater extent in America than in Europe.[129] This was partly due to looser diagnostic criteria in the US, which used the DSM-II manual, contrasting with Europe and its ICD-9. David Rosenhan's 1972 study, published in the journal *Science* under the title "On being sane in insane places", concluded that the diagnosis of schizophrenia in the US was often subjective and unreliable.[130] These were some of the factors leading to the revision not only of the diagnosis of schizophrenia, but the revision of the whole DSM manual, resulting in the publication of the DSM-III in 1980.[131] The term schizophrenia is commonly misunderstood to mean that affected persons have a "split personality". Although some people diagnosed with schizophrenia may hear voices and may experience the voices as distinct personalities, schizophrenia does not involve a person changing among distinct multiple personalities. The confusion arises in part due to the literal interpretation of Bleuler's term schizophrenia (Bleuler originally associated Schizophrenia with dissociation and included split personality in his category of Schizophrenia[132][133]). Dissociative identity disorder (having a "split personality") was also often misdiagnosed as Schizophrenia based on the loose criteria in the DSM-II.[133][134] The first known misuse of the term to mean "split personality" was in an article by the poet T. S. Eliot in 1933.[135]

Society and culture

In 2002 the term for schizophrenia in Japan was changed from *Seishin-Bunretsu-Byō* 精神分裂病 (mind-split-disease) to *Tōgō-shitchō-shō* 統合失調症 (integration disorder) to reduce stigma.[136] The new name was inspired by the biopsychosocial model; it increased the percentage of patients who were informed of the diagnosis from 37% to 70% over three years.[137]

In the United States, the cost of schizophrenia—including direct costs (outpatient, inpatient, drugs, and long-term care) and non-health care costs (law enforcement, reduced workplace productivity, and unemployment)—was estimated to be $62.7 billion in 2002.[138] The book and film *A Beautiful Mind* chronicles the life of John Forbes Nash, a Nobel Prize-winning mathematician who was diagnosed with schizophrenia.

The term schizophrenia was coined by Eugen Bleuler.

Violence

Individuals with severe mental illness including schizophrenia are at a significantly greater risk of being victims of both violent and non-violent crime.[139] On the other hand, schizophrenia has sometimes been associated with a higher rate of violent acts, although this is primarily due to higher rates of drug use.[140] Rates of homicide linked to psychosis are similar to those linked to substance misuse, and parallel the overall rate in a region.[141] What role schizophrenia has on violence independent of drug misuse is controversial, but certain aspects of individual histories or mental states may be factors.[142]

Media coverage relating to schizophrenia tends to revolve around rare but unusual acts of violence. Furthermore, in a large, representative sample from a 1999 study, 12.8% of Americans believed that individuals with schizophrenia were "very likely" to do something violent against others, and 48.1% said that they were "somewhat likely" to. Over 74% said that people with schizophrenia were either "not very able" or "not able at all" to make decisions concerning their treatment, and 70.2% said the same of money management decisions.[143] The perception of individuals with psychosis as violent has more than doubled in prevalence since the 1950s, according to one meta-analysis.[144]

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External links

- Schizophrenia (http://www.dmoz.org/Health/Mental_Health/Disorders/Schizophrenia//) at the Open Directory Project

Epileptic seizure

Epileptic seizure	
Classification and external resources	
ICD-10	G40 [1], P90 [2], R56 [3]
ICD-9	345.9 [4], 780.3 [5]
DiseasesDB	19011 [6]
eMedicine	neuro/694 [7] neuro/415 [8]
MeSH	D012640 [9]

An **epileptic seizure**, occasionally referred to as a **fit**, is defined as a transient symptom of "abnormal excessive or synchronous neuronal activity in the brain".[10] The outward effect can be as dramatic as a wild thrashing movement (tonic-clonic seizure) or as mild as a brief loss of awareness (absence seizure). It can manifest as an alteration in mental state, tonic or clonic movements, convulsions, and various other psychic symptoms (such as déjà vu or jamais vu). Sometimes it is not accompanied by convulsions but a full body "slump", where the person simply will lose body control and slump to the ground. The medical syndrome of recurrent, unprovoked seizures is termed epilepsy, but seizures can occur in people who do not have epilepsy. For more information, see non-epileptic seizure.

About 4% of people will have an unprovoked seizure by the age of 80 and the chance of experiencing a second seizure is between 30% and 50%.[11][12] Treatment may reduce the chance of a second one by as much as half.[12] Most single episode seizures are managed by primary care physicians (emergency or general practitioners), whereas investigation and management of ongoing epilepsy is usually done by neurologists. Difficult-to-manage epilepsy may require consultation with an epileptologist, a neurologist with an interest in epilepsy.

Classification

Clinicians organize different types of seizure according to whether the source of the seizure within the brain is localized (*partial-* or *focal*-onset seizures) or distributed (*generalized* seizures). Partial seizures are further divided on the extent to which consciousness is affected (simple partial seizures and complex partial seizures). If consciousness is unaffected, then it is a *simple partial* seizure; otherwise it is a *complex partial* seizure. A partial seizure may spread within the brain—a process known as *secondary generalization*. Generalized seizures are divided according to the effect on the body, but all involve loss of consciousness. These include absence, myoclonic, clonic, tonic, tonic–clonic, and atonic seizures. A *mixed seizure* is defined as the existence of both generalized and partial seizures in the same patient.[13]

Following standardization proposals published in 1970, terms such as "petit mal", "grand mal", "Jacksonian", "psychomotor", and "temporal-lobe seizure" have fallen into disuse.

Signs and symptoms

The signs and symptoms of seizures vary depending on the type.[14] Seizures may cause involuntary changes in body movement or function, sensation, awareness, or behavior. Seizures are often associated with a sudden and involuntary contraction of a group of muscles and loss of consciousness. However, a seizure can also be as subtle as a fleeting numbness of a part of the body, a brief or long term loss of memory, visual changes, sensing/discharging of an unpleasant odor, a strange epigastric sensation, or a sensation of fear and total state of confusion. A seizure can last from a few seconds to status epilepticus, a continuous group of seizures that is often life-threatening without immediate intervention. Therefore seizures are typically classified as motor, sensory, autonomic, emotional or cognitive. After the active portion of a seizure, there is typically a period referred to as postictal before a normal level of consciousness returns.[14]

In some cases, the full onset of a seizure event is preceded by some of the sensations described above, called vertiginous epilepsy. These sensations can serve as a warning to that a generalized tonic–clonic seizure is about to occur. These warning sensations are cumulatively called an aura and are due to a focal seizure.[14]

Some patients are able to tell when a seizure is about to happen. Some symptoms experienced by the person before a seizure may include dizziness, lightheadedness, tightening of the chest, and some experience things in slow-motion just prior to the seizure. Symptoms experienced by a person during a seizure depend on where in the brain the disturbance in electrical activity occurs. Partial and frontal seizures and focal epileptic discharges tend to happen more during sleep than during wakefulness. In contrast, psychogenic nonepileptic seizures are rare between midnight and 6 a m. and never occur during sleep.[15] Generalized epilepsy but not focal epilepsy is higher in the morning probably reflecting a diurnal variation in cortical excitability.[16] A person having a tonic–clonic seizure may cry out, lose consciousness and fall to the ground, and convulse, often violently. A person having a complex partial seizure may appear confused or dazed and will not be able to respond to questions or direction. Some people have seizures that are not noticeable to others. Sometimes, the only clue that a person is having an absence seizure is rapid blinking, extreme confusion for a few seconds or sometimes into hours.

Causes

Unprovoked seizures are often associated with epilepsy and related seizure disorders.

Causes of *provoked* seizures include:

- dehydration
- sleep deprivation
- cavernoma or cavernous malformation is a treatable medical condition that can cause seizures, headaches, and brain hemorrhages. An MRI can quickly confirm or reject this as a cause.
- arteriovenous malformation (AVM) is a treatable medical condition that can cause seizures, headaches, and brain hemorrhages.
- head injury may cause non-epileptic post-traumatic seizures or post-traumatic epilepsy, in which the seizures chronically recur.
- intoxication with drugs, for example aminophylline or local anaesthetics
- normal doses of certain drugs that lower the seizure threshold, such as tricyclic antidepressants
- infection, such as encephalitis or meningitis
- fever leading to febrile convulsions (but see above)
- metabolic disturbances, such as hypoglycaemia, hyponatremia or hypoxia
- withdrawal from drugs (anticonvulsants, antidepressants, and sedatives such as alcohol, barbiturates, and benzodiazepines,)
- space-occupying lesions in the brain (abscesses, tumors)
- seizures during (or shortly after) pregnancy can be a sign of eclampsia.

- seizures in a person with hydrocephalus may indicate severe shunt failure.
- binaural beat brainwave entrainment may trigger seizures in both epileptics and non-epileptics
- haemorrhagic stroke can occasionally present with seizures, embolic strokes generally do not (though epilepsy is a common later complication); cerebral venous sinus thrombosis, a rare type of stroke, is more likely to be accompanied by seizures than other types of stroke
- multiple sclerosis sufferers may rarely experience seizures
- parasitic infection such as cerebral malaria

Some medications produce an increased risk of seizures and electroconvulsive therapy (ECT) deliberately sets out to induce a seizure for the treatment of major depression. Many seizures have unknown causes.

Seizures which are provoked are not associated with epilepsy, and people who experience such seizures are normally not diagnosed with epilepsy. However, the seizures described above resemble those of epilepsy both outwardly, and on EEG testing.

Seizures can occur after a subject witnesses a traumatic event. This type of seizure is known as a psychogenic non-epileptic seizure and is related to posttraumatic stress disorder.

Diagnosis

Only about 25 percent of people who have a seizure or develop status epilepticus have epilepsy.[17] It is important to distinguish primary epileptic seizures from secondary causes. Blood tests, lumbar puncture or toxicology screening can be helpful in specific circumstances suggestive of an underlying cause like alcohol or benzodiazepine withdrawal, meningitis or drug overdose, but there is insufficient evidence to support their routine use in the work-up of an adult with an apparently unprovoked first seizure.[18] A 2007 review recommends an electroencephalogram and brain imaging with CT scan or MRI scan in the work-up.[19] MRI is more sensitive in a first apparently unprovoked seizure.

Physical examination

Most patients are in a postictal state following a seizure. In this state they are drowsy and often confused. There may be signs of other injuries. A small study found that finding a bite to the side of the tongue was very helpful when present: while only a quarter of those with seizures had such a bite (sensitivity of 24%), the finding was very specific for seizures, with only 1% due to other causes (specificity of 99%).[20]

Serum prolactin level

Two meta-analyses have quantified the role of an elevated serum prolactin. The first meta-analysis found that[21]: "If a serum prolactin concentration is greater than three times the baseline when taken within one hour of syncope, then in the absence of test "modifiers":

1. the patient is nine times more likely to have suffered a GTCS as compared with a pseudoseizure positive LR = 8.92 (95% CI (1.31 to 60.91)), SN = 0.62 (95% CI (0.40 to 0.83)), SP = 0.89 (95% CI (0.60 to 0.98))
2. five times more likely to have suffered a GTCS as compared with non-convulsive syncope positive LR 4.60 (95% CI (1.25 to 16.90)), SN = 0.71 (95% CI (0.49 to 0.87)), SP = 0.85 (95% CI (0.55 to 0.98)). "

The second meta-analysis found:[22]

1. "Elevated serum prolactin assay, when measured in the appropriate clinical setting at 10 to 20 minutes after a suspected event, is a useful adjunct for the differentiation of generalized tonic-clonic or complex partial seizure from psychogenic nonepileptic seizure among adults and older children (Level B)."
2. "Serum prolactin assay does not distinguish epileptic seizures from syncope (Level B).
3. "The use of serum PRL assay has not been established in the evaluation of status" epilepticus, repetitive seizures, and neonatal seizures (Level U)."

The serum prolactin level is less sensitive for detecting partial seizures.[23]

EEG

An isolated abnormal electrical activity recorded by an electroencephalography examination without a clinical presentation is called subclinical seizure. They can identify background epileptogenic activity, as well as help identify causes of seizures.

Determining the underlying cause

Additional diagnostic methods include CT Scanning and MRI imaging or angiography. These may show structural lesions within the brain and heart, but the majority of those with epilepsy show nothing unusual.

As seizures have a broad differential diagnosis, it is common for patients to be simultaneously investigated for cardiac and endocrine causes. Checking glucose levels, for example, is a mandatory action in the management of seizures as hypoglycemia may cause seizures, and failure to administer glucose would be harmful to the patient. Other causes typically considered are syncope and cardiac arrhythmias, and occasionally panic attacks and cataplexy. In addition, 5% of patients with a positive tilt table test may have seizure-like activity that seems to be due to cerebral hypoxia.[24] For more information, see non-epileptic seizures.

Differential

Differentiating an epileptic seizure from other conditions such as syncope can be difficult.[14] Other possible conditions that can mimic a seizure include: decerebrate posturing, psychogenic seizures, dystonia, migraine headaches, and strychnine poisoning.[14]

Management

Potentially sharp or dangerous objects should also be moved from the vicinity, so that the individual is not hurt. After the seizure if the person is not fully conscious and alert, they should be placed in the recovery position.

A seizure longer than five minutes is a medical emergency. Caregivers may carry medicine.

Medication

The treatment of choice for someone who is actively seizing is lorazepam.[19] This may be repeated if there is no effect after 10 minutes.[19] If there is no effect after two doses, barbiturates or propofol may be used.[19] Ongoing medication is not typically needed after a first seizure and is generally only recommended after a second has occurred or those with structural lesions in the brain.[19] However, in severe cases where patients experience frequent unpredictable seizures everyday, the use of anti-epileptic drugs (AEDs) are recommended to reduce the likelihood and duration of seizures. Approximately 70% of the patients could have the seizures fully under control with continuous use of medication. The National Institute of Health and Clinical Excellence (NICE) suggests patients taking only one type of AEDs at a time.[25] Monodrug therapy is recommended in the treatment of epileptic seizures because it helps in the lowering of adverse effects from the medication, prevents the patient from experiencing multiple drug interactions and makes it easier for a patient to maintain a proper dosing schedule. [26]

Other

A seizure response dog can be trained to summon help or ensure personal safety when a seizure occurs. These are not suitable for everybody. Rarely, a dog may develop the ability to sense a seizure before it occurs.[27]
Helmets may be used to provide protection of the head during a seizure.

Prognosis

In adults, after 6 months seizure free, after a first seizure the risk of a subsequent seizure in the next year is less than 20% regardless of treatment.[28] Up to 7% of seizure that present to the emergency are in status epilepticus.[19] In those with a status epilepticus mortality is between 10 and 40%.[14]

Epidemiology

About 7 per 1000 people in the United States have a seizure in a given year.[14] Rates are highest in those less than 1 year of age and greater than 55.[14]

History

The word epilepsy is derived from the Greek word for "attack."[29] Seizures were long viewed as an otherworldly condition being referred to by Hippocrates in 400B.C. as "the sacred disease".[14]

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External links

- Seizure First Aid (http://www.epilepsyfoundation.org/about/firstaid/index.cfm)
- MEDLINEplus: Seizures (http://www.nlm.nih.gov/medlineplus/seizures.html)
- Epilepsy and seizure information, news and support for people with epilepsy and their friends, families, doctors and caregivers: Epilepsy Foundation (http://epilepsyfoundation.org/)
- Epilepsy and seizure information for patients and health professionals : Epilepsy.com (http://www.epilepsy.com)

Parasomnia

Parasomnia	
Classification and external resources	
ICD-10	F51.3 [1]-F51.4 [2]
ICD-9	307.47 [3], 327.4 [4], 780.59 [5]
eMedicine	med/3131 [6]
MeSH	D020447 [7]

Parasomnias are a category of sleep disorders that involve abnormal and unnatural movements, behaviors, emotions, perceptions, and dreams that occur while falling asleep, sleeping, between sleep stages, or during arousal from sleep. Most parasomnias are dissociated sleep states which are partial arousals during the transitions between wakefulness and NREM sleep, or wakefulness and REM sleep.

Non-rapid eye movement (NREM) parasomnias

NREM parasomnias are arousal disorders that occur during stage 3 (or 4 by the R&K standardization) of NREM sleep—also known as slow wave sleep (SWS). They are caused by a physiological activation in which the patient's brain exits from SWS and is caught in between a sleeping and waking state. In particular, these disorders involve activation of the autonomic nervous system, motor system, or cognitive processes during sleep or sleep-wake transitions.[8]

Some NREM parasomnias (sleep-walking, night-terrors, and confusional arousal) are common during childhood but decrease in frequency with increasing age. They can be triggered in certain individuals, by alcohol, sleep deprivation, physical activity, emotional stress, depression, medications, or a fevered illness. These disorders of arousal can range from confusional arousals, somnambulism, to night terrors. Other specific disorders include sleepeating, sleep sex, teeth grinding, rhythmic movement disorder, restless legs syndrome, and somniloquy.

Confusional arousals

With a prevalence of 4%, confusional arousals are not observed very often in adults; however, they are common in children.[9] Infants and toddlers usually experience confusional arousals beginning with large amounts of movement and moaning, which can later progress to occasional thrashings or inconsolable crying. Confusional arousal is a condition when an individual awakens from sleep and remains in a confused state. It is characterized by the individual's partial awakening and sitting up to look around. They usually remain in bed and then return back to sleep. These episodes last anywhere from seconds to minutes and may not be reactive to stimuli.[10] Confusional arousals are not considered dangerous. Another sleeping disorder may be present triggering these incomplete arousals.[11]

Sleepwalking (somnambulism)

Sleepwalking has a prevalence of 1-17% in childhood, with the most frequent occurrences around the age of eleven to twelve. About 4% of adults experience somnambulism.[12] Normal sleep cycles include states varying from drowsiness all the way to deep sleep. Every time an individual sleeps, he or she goes through various sequences of non-REM and REM sleep. Anxiety and fatigue are usually connected with sleepwalking. For adults, alcohol, sedatives, medications, medical conditions and mental disorders are all associated with sleepwalking. Sleep walking may involve sitting up and looking awake when the individual is actually asleep, and getting up and walking around,

moving items or undressing themselves. They will also be confused when waking up or opening their eyes during sleep. Some individuals also talk while in their sleep, saying meaningless words and even having arguments with people who are not there. [13]

Sleep terrors (night terrors)

Sleep terror is the most disruptive arousal disorder since it may involve loud screams and panic; in extreme cases, it may result in bodily harm or property damage by running about or hitting walls. Unfortunately, all attempts to console the individual are futile and may prolong or intensify the victim's confused state. Usually the victim experiences amnesia after the event but it may not be complete amnesia. Up to 3% of adults suffer from sleep terrors, and exhibited behavior of this parasomnia can range from mild to extremely violent.[14] They typically occur in stage 3 sleep.[15]

Teeth grinding (bruxism)

Bruxism is a common sleep disorder where the individual grinds their teeth during sleep. This can cause sleep disruption for the individual and also the bed partner. Grinding can wear and fracture the teeth, and also cause severe jaw pain. This can lead to migraines, teeth impairment, and other complications. A lot of people are not aware of their teeth grinding. Teeth grinding may be caused by stress and anxiety; it could also be caused by a non typical bite, or missing teeth.

Restless legs syndrome & periodic limb movements

Both of these conditions (RLS and PLM) are classified as dyssomnias according to the DSM-IV. They are considered parasomnias.

Sleep sex

Sleep sex, or sexsomnia, is a condition in which a person will engage in sexual acts while still asleep. A condition usually occupied by another sleep disorder it can include such acts as masturbation, fondling themselves or others, having sex with another person and in more extreme cases sexual assault and rape.

Sleep related eating disorder (SRED)

Sleep Related Eating Disorder is a condition in which individuals eat during sleep. They usually head for the kitchen and indulge in uncooked food, snacks and sometimes even toxic substances. This can be very dangerous and even deadly, especially if the individual has food allergies, or even is diabetic. This disorder usually starts with individuals who sleep walk and develop into SRED.

REM parasomnias

REM sleep behavior disorder

REM sleep behavior disorder is the most common REM sleep parasomnia in which muscle atonia is absent. This allows the individual to act out their dreams and may result in repeated injury—bruises, lacerations, and fractures—to themselves or others. Patients may take self-protection measures by tethering themselves to bed, using pillow barricades, or sleeping in an empty room on a mattress.[16] Demographically, 90% of RBD patients are males, and most are older than 50 years of age.[17]

Typical clinical features of REM sleep behavior disorder are:

- Male gender predilection
- Mean age of onset 50–65 years (range 20–80 years)

- Vocalisation, screaming, swearing that may be associated with dreams
- Motor activity, simple or complex, that may result in injury to patient or bed-partner
- Occurrence usually in latter half of sleep period (REM sleep)
- May be associated with neurodegenerative disease [18]

Acute RBD, occurs mostly as a result of a side-effect in prescribed medication—usually antidepressants. But if not then 55% of the time the cause is unknown the other 45% the cause is associated with alcohol.[19]

Chronic RBD is idiopathic, meaning of unknown origin, or associated with neurological disorders. There is a growing association of chronic RBD with neurodegenerative disorders—Parkinson's disease, multiple system atrophy (MSA), or dementia—as an early indicator of these conditions by as much as 10 years.

Patients with narcolepsy also are more likely to develop RBD.

Recurrent isolated sleep paralysis

Recurrent Isolated Sleep Paralysis is an inability to perform voluntary movements at sleep onset, or upon waking from sleep.[20]

Catathrenia

Catathrenia, a rapid-eye-movement sleep parasomnia consisting of breath holding and expiratory groaning during sleep, is distinct from both somniloquy and obstructive sleep apnea. The sound is produced during exhalation as opposed to snoring which occurs during inhalation. It is usually not noticed by the person producing the sound but can be extremely disturbing to sleep partners, although once aware of it, sufferers tend to be woken up by their own groaning as well. Bed partners generally report hearing the person take a deep breath, hold it, then slowly exhale; often with a high-pitched squeak or groaning sound.

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Notes

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Further reading

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External links

- Stanford: Parasomnias - Arousal Disorders Information (http://www.stanford.edu/~dement/para.html)
- Primary Sleep Disorders: Parasomnias (http://sleep.health.am/sleep/parasomnias/)
- Psychnet UK (http://www.psychnet-uk.com/dsm_iv/parasomnias.htm)
- Insomnia/Parasomnia (http://www.sleepwebmd.com/insomnia___parasomnia.htm)
- (http://www.mayoclinic.com/health/bruxism/DS00337)
- (http://www.webmd.com/oral-health/guide/teeth-grinding-bruxism)

Hypnagogia

Hypnagogia is the study of the transitional states to and from sleep: the *hypnagogic* and the *hypnopompic* states of consciousness. Note the spelling difference: "-agogic" but "-opompic". The related words from the Greek are *agōgos* "leading, inducing", *pompe* "act of sending", and *hypnos* "sleep".

"Hypnagogia" was coined by Dr Andreas Mavromatis in his 1983 thesis,[1] while "hypnagogic" and "hypnopompic" were coined by others in the 1800s. The term "hypnagogic" was originally coined by Alfred Maury[2][3] to name the state of consciousness during the onset of sleep. "Hypnopompic" was coined by Fred Meyers soon afterwards to denote the onset of wakefulness. The term "hypnagogia" is used by Dr Mavromatis to identify the study of the sleep-transitional consciousness states in general, and he employs *hypnogogic* (toward sleep) or *hypnapompic* (from sleep) for the purpose of identifying the specific experiences under study.[4]

Mental phenomena that occur during this "threshold consciousness" phase include lucid dreaming, hallucinations, and sleep paralysis.

Definitions and synonyms

Sometimes the word *hypnagogia* is used in a restricted sense to refer to the onset of sleep, and contrasted with *hypnopompia*, Frederic Myers's term for waking up.[5] However, *hypnagogia* is also regularly employed in a more general sense that covers both falling asleep and waking up, and Havelock Ellis questioned the need for separate terms.[6] Indeed, it is not always possible in practice to assign a particular episode of any given phenomenon to one or the other, given that the same kinds of experience occur in both, and that people may drift in and out of sleep. In this article *hypnagogia* will be used in the broader sense, unless otherwise stated or implied.

Other terms for hypnagogia, in one or both senses, that have been proposed include "presomnal" or "anthypnic sensations", "visions of half-sleep", "oneirogogic images" and "phantasmata",[6] "the borderland of sleep", "praedormitium",Mavromatis, Andreas (1987). *Hypnagogia: the Unique State of Consciousness Between Wakefulness and Sleep*. London: Routledge and Kegan Paul. p. 4. ISBN 0-7102-0282-2.</ref> the "borderland state", "half-dream state", "pre-dream condition",[7] "sleep onset dreams",[8] dreamlets,[9] and "wakefulness-sleep transition" state (WST).[10]

Threshold consciousness (commonly called "half-asleep" or "half-awake", or "Mind Awake Body Asleep") describes the same mental state of someone who is moving towards sleep or wakefulness but has not yet completed the transition. Such transitions are usually brief, but can be extended by sleep disturbance or deliberate induction, for example during meditation.

History

Early references to hypnagogia are to be found in the writings of Aristotle, Iamblichus, Cardano, Simon Forman and Swedenborg.[11] Romanticism brought a renewed interest in the subjective experience of the edges of sleep.[12] In more recent centuries, many authors have referred to the state; Edgar Allan Poe, for example, wrote of the "fancies" he experienced "only when I am on the brink of sleep, with the consciousness that I am so."[13]

Serious scientific enquiry began in the 19th century with Johannes Peter Müller, Jules Baillarger and Alfred Maury, and continued into the twentieth century with Leroy.[14]

The advent of electroencephalography (EEG) has supplemented the introspective methods of these early researchers with physiological data. The search for neural correlates for hypnagogic imagery began with Davis et al. in the 1930s,[15] and continues with increasing sophistication to this day. While the dominance of the behaviorist paradigm led to a decline in research, especially in the English speaking world, the later 20th century has seen a revival, with investigations of hypnagogia and related ASCs playing an important role in the emerging multidisciplinary study of consciousness.[16][17] Nevertheless, much remains to be understood about the experience and its corresponding neurology, and the topic has been somewhat neglected in comparison with sleep and dreams; hypnagogia has been described as a "well-trodden and yet unmapped territory."[18]

Important reviews of the scientific literature have been made by Leaning,[19] Schacter,[20] Richardson and Mavromatis.[6]

Sensory phenomena

Transition to and from sleep may be attended by a wide variety of sensory experiences. These can occur in any modality, individually or combined, and range from the vague and barely perceptible to vivid hallucinations.[21]

Sights

Among the more commonly reported,[22][23] and more thoroughly researched, sensory features of hypnagogia are phosphenes which can manifest as seemingly random speckles, lines or geometrical patterns, including form constants, or as figurative (representational) images. They may be monochromatic or richly colored, still or moving, flat or three-dimensional (offering an impression of perspective). Imagery representing movement through tunnels of light is also reported. Individual images are typically fleeting and given to very rapid changes. They are said to differ from dreams proper in that hypnagogic imagery is usually static and lacking in narrative content,[16] although others understand the state rather as a gradual transition from hypnagogia to fragmentary dreams,[24] i.e., from simple "eigenlicht" to whole imagined scenes. It has been claimed that hypnagogia can be induced with a Dreamachine,[25] which uses light pulsing at a frequency close to that of alpha waves to create this effect, but this claim has not been tested in scientific experiments. Descriptions of exceptionally vivid and elaborate hypnagogic visuals can be found in the work of Marie-Jean-Léon, Marquis d'Hervey de Saint Denys.

Tetris effect

People who have spent a long time at some repetitive activity before sleep, in particular one that is new to them, may find that it dominates their imagery as they grow drowsy, a tendency dubbed the Tetris effect. This effect has even been observed in amnesiacs who otherwise have no memory of the original activity.[26] When the activity involves moving objects, as in the video game Tetris, the corresponding hypnagogic images tend to be perceived as moving. The Tetris effect is not confined to visual imagery, but can manifest in other modalities also. For example, Robert Stickgold recounts having experienced the touch of rocks while falling asleep after mountain climbing.[8] This can also occur to people who have traveled on a small boat in rough seas, or have been swimming through waves, shortly before going to bed, and they feel the waves as they drift to sleep, or people who have spent the day skiing who continue to "feel snow" under their feet, also people who have spent considerable time jumping on a trampoline will find that they can feel the up-and-down motion before they go to sleep. Many chess players report the phenomenon of seeing the chess board and pieces during this state. New employees working stressful and demanding jobs often report doing work-related tasks in this period before sleep. This is very common amongst new waiters or waitresses in busy restaurants where they report having "Server Dreams" and restlessly wait tables in this state of mind, sometimes jolting them fully awake or preventing them from transitioning into actual sleep.

Sounds

Hypnagogic imagery is often auditory or has an auditory component. Like the visuals, hypnagogic sounds vary in intensity from faint impressions to loud noises, such as crashes and bangs (exploding head syndrome). People may imagine their own name called or a doorbell ringing. Snatches of imagined speech are common. While typically nonsensical and fragmented, these speech events can occasionally strike the individual as apt comments on—or summations of—their thoughts at the time. They often contain word play, neologisms and made-up names. Hypnagogic speech may manifest as the subject's own "inner voice", or as the voices of others: familiar people or strangers. More rarely, poetry or music is heard.[27]

Sleep paralysis

Humming, roaring, hissing, rushing, zapping, and buzzing noises are frequent in conjunction with sleep paralysis (SP). This happens when the REM atonia sets in sooner than usual, before the person is fully asleep, or persists longer than usual, after the person has (in other respects) fully awoken.[17] Sleep paralysis is reportedly very frequent among narcoleptics. It occurs frequently in about 6% of the rest of the population, and occurs occasionally in 60%.[28] In surveys from Canada, China, England, Japan and Nigeria, 20 to 60% of individuals reported having experienced SP at least once in their lifetime.[29][30] The paralysis itself is frequently accompanied by additional phenomena. Typical examples include a feeling of being crushed or suffocated, electric "tingles" or "vibrations", imagined speech and other noises, the imagined presence of a visible or invisible entity, and sometimes intense emotion: fear or euphoria and orgasmic feelings.[29][31] SP has been proposed as an explanation for at least some alien abduction experiences and shadow people hauntings.[32]

Other sensations

Gustatory, olfactory and thermal sensations in hypnagogia have all been reported, as well as tactile sensations (including those kinds classed as paresthesia or formication). Sometimes there is synesthesia; many people report seeing a flash of light or some other visual image in response to a real sound. Proprioceptive effects may be noticed, with numbness and changes in perceived body size and proportions,[27] feelings of floating or bobbing, and out-of-body experiences.[33] Perhaps the most common experience of this kind is the falling sensation, and associated hypnic jerk, encountered by many people, at least occasionally, while drifting off to sleep.[34]

Cognitive and affective phenomena

Thought processes on the edge of sleep tend to differ radically from those of ordinary wakefulness. Hypnagogia may involve a "loosening of ego boundaries ... openness, sensitivity, internalization-subjectification of the physical and mental environment (empathy) and diffuse-absorbed attention."[35] Hypnagogic cognition, in comparison with that of normal, alert wakefulness, is characterized by heightened suggestibility,[36] illogic and a fluid association of ideas. Subjects are more receptive in the hypnagogic state to suggestion from an experimenter than at other times, and readily incorporate external stimuli into hypnagogic trains of thought and subsequent dreams. This receptivity has a physiological parallel; EEG readings show elevated responsiveness to sound around the onset of sleep.[37]

Herbert Silberer described a process he called *autosymbolism*, whereby hypnagogic hallucinations seem to represent, without repression or censorship, whatever one is thinking at the time, turning abstract ideas into a concrete image, which may be perceived as an apt and succinct representation thereof.[38]

The hypnagogic state can provide insight into a problem, the best-known example being August Kekulé's realization that the structure of benzene was a closed ring while half-asleep in front of a fire and seeing molecules forming into snakes, one of which grabbed its tail in its mouth.[39] Many other artists, writers, scientists and inventors—including Beethoven, Richard Wagner, Walter Scott, Salvador Dalí, Thomas Edison, Nikola Tesla and Isaac Newton—have credited hypnagogia and related states with enhancing their creativity.[40] A 2001 study by Harvard psychologist Deirdre Barrett found that, while problems can also be solved in full-blown dreams from later stages of sleep, hypnagogia was especially likely to solve problems which benefit from hallucinatory images being critically examined while still before the eyes.[41]

A feature that hypnagogia shares with other stages of sleep is amnesia. But this is a selective forgetfulness, affecting the hippocampal memory system, which is responsible for episodic or autobiographical memory, rather than the neocortical memory system, responsible for semantic memory.[8] It has been suggested that hypnagogia and REM sleep help in the consolidation of semantic memory,[42] but the evidence for this has been disputed.[43] For example, suppression of REM sleep due to antidepressants and lesions to the brainstem has not been found to produce detrimental effects on cognition.[44]

Hypnagogic phenomena may be interpreted as visions, prophecies, premonitions, apparitions and inspiration (artistic or divine), depending on the experiencers' beliefs and those of their culture.

Physiology

Physiological studies have tended to concentrate on hypnagogia in the strict sense of spontaneous sleep onset experiences. Such experiences are associated especially with stage 1 of NREM sleep,[45] but may also occur with pre-sleep alpha waves.[46][47] Davis et al. found short flashes of dreamlike imagery at the onset of sleep to correlate with drop-offs in alpha EEG activity.[15] Hori et al. regard sleep onset hypnagogia as a state distinct from both wakefulness and sleep with unique electrophysiological, behavioral and subjective characteristics,[48][49] while Germaine et al. have demonstrated a resemblance between the EEG power spectra of spontaneously occurring hypnagogic images, on the one hand, and those of both REM sleep and relaxed wakefulness, on the other.[50]

To identify more precisely the nature of the EEG state which accompanies imagery in the transition from wakefulness to sleep, Hori et al. proposed a scheme of 9 EEG stages defined by varying proportions of alpha (stages 1–3), suppressed waves of less than 20μV (stage 4), theta ripples (stage 5), proportions of sawtooth waves (stages 6–7), and presence of spindles (stages 8–9).[51] Germaine and Nielsen found spontaneous hypnagogic imagery to occur mainly during Hori sleep onset stages 4 (EEG flattening) and 5 (theta ripples).[23]

The "covert-rapid-eye-movement" hypothesis proposes that hidden elements of REM sleep emerge during the wakefulness-sleep transition stage.[52] Support for this comes from Bódicz et al., who note a greater similarity between WST (wakefulness-sleep transition) EEG and REM sleep EEG than between the former and stage 2 sleep.[10]

Respiratory pattern changes have also been noted in the hypnagogic state, in addition to a lowered rate of frontalis muscle activity.[53]

Daydreaming and waking reveries

Microsleep (short episodes of immediate sleep onset) may intrude into wakefulness at any time in the wakefulness-sleep cycle, due to sleep deprivation and other conditions,[54] resulting in impaired cognition, amnesia.[16]

Gurstelle and Oliveira distinguish a state which they call *daytime parahypnagogia* (DPH), the spontaneous intrusion of a flash image or dreamlike thought or insight into one's waking consciousness. DPH is typically encountered when one is "tired, bored, suffering from attention fatigue, and/or engaged in a passive activity." The exact nature of the episode may be forgotten even though the individual remembers having had such an experience.[55] Gustelle and Oliveira define DPH as "dissociative, trance-like, [...] but, unlike a daydream, [...] not self-directed"—however, daydreams and waking reveries are often characterised as "passive", "effortless",[56] and "spontaneous",[16] while hypnagogia itself can sometimes be influenced by a form of autosuggestion, or "passive concentration",[57] so these sorts of episode may in fact constitute a continuum between directed fantasy and the more spontaneous varieties of hypnagogia. Others have emphasized the connections between fantasy, daydreaming, dreams and hypnosis.[58]

In his book, *Zen and the Brain*, James H. Austin cites speculation that regular meditation develops a specialized skill of "freezing the hypnagogic process at later and later stages" of the onset of sleep, initially in the alpha wave stage and later in theta.[59]

Investigative methodology

Self-observation (spontaneous or systematic) was the primary tool of the early researchers. Since the late twentieth century, this has been joined by questionnaire surveys and experimental studies. All three methods have their disadvantages as well as points to recommend them.[60]

Naturally, amnesia contributes to the difficulty of studying hypnagogia, as does the typically fleeting nature of hypnagogic experiences. These problems have been tackled by experimenters in a number of ways, including voluntary or induced interruptions,[23] sleep manipulation,[61] the use of techniques to "hover on the edge of sleep" thereby extending the duration of the hypnagogic state,[62] and training in the art of introspection to heighten the subject's powers of observation and attention.[62]

Techniques for extending hypnagogia range from informal ones (e.g., the subject holds up one of their arms as they go to sleep, so as to be awakened when it falls),[62] to the use of biofeedback devices to induce a "theta" state, characterized by relaxation and theta EEG activity.[63] The theta state is produced naturally the most when we are dreaming. It has also been linked to paranormal activities, and Rick Strassman has argued that it triggers the release of DMT from the pineal gland, causing a dreaming state.[64]

Another method is to induce a state said to be subjectively similar to sleep onset in a Ganzfeld setting, a form of sensory deprivation. But the assumption of identity between the two states may be unfounded. The average EEG spectrum in Ganzfeld is more similar to that of the relaxed waking state than to that of sleep onset.[65] Wackerman et al. conclude that "the Ganzfeld imagery, although subjectively very similar to that at sleep onset, should not be labeled as 'hypnagogic'. Perhaps a broader category of 'hypnagoid experience' should be considered, covering true hypnagogic imagery as well as subjectively similar imagery produced in other states."[66]

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External links

- " Hypnagogic and hypnopompic hallucinations: pathological phenomena? (http://bjp.rcpsych.org/cgi/content/abstract/169/4/459)" article in the *British Journal of Psychiatry*
- Gary Lachman on Hypnagogia (http://www.forteantimes.com/features/articles/227/hypnagogia.html)

Night terror

Night terror	
Classification and external resources	
ICD-10	F51.4 [2]
ICD-9	307.46 [1]
MedlinePlus	000809 [2]
MeSH	D020184 [3]

A **night terror**, also known as a **sleep terror** or ***pavor nocturnus***, is a parasomnia disorder, causing feelings of terror or dread, and typically occurring in the first few hours of sleep during stage 3 or 4 non-rapid eye movement NREM sleep.[4] Night terrors tend to happen during periods of arousal from delta sleep, also known as slow wave sleep.[5][6][7] During the first half of a sleep cycle, delta sleep occurs most often which indicates that people with more delta sleep activity are more prone to night terrors.[6] However, they can also occur during daytime naps.[8]

Night terrors have been known since the biblical times, although it was impossible to differentiate from nightmares until rapid eye movement was discovered.[7] Night terrors should not be confused with nightmares, which are bad dreams that cause feelings of horror or fear. While nightmares are relatively common during childhood, night terrors occur less frequently according to the *American Academy of Child and Adolescent Psychiatry*.[9] An estimated 1%-6% of children and fewer than 1% of adults will experience a night terror episode within their lifetime.[10] Night terrors can often be mistaken for the disorder of confusional arousal.[5] Sleep terrors begin between ages 3 and 12 years and then usually dissipate during adolescence. In adults they most commonly occur between the ages of 20 to 30. Though the frequency and severity varies between individuals, the episodes can occur in intervals of days or weeks, but can also occur over consecutive nights or multiple times in one night.[8] Clinical studies have indicated that adults who suffer from this disorder may actually be showing signs of an underlying mental disorder, and may have serious consequences.[6] Night terrors are largely unknown to most people, creating the notion that any type of nocturnal attack or nightmare can be confused with and reported as a night terror.[7]

Associated features of night terrors

The universal feature of night terrors is inconsolability.[11] During night terror bouts, patients are usually described as 'bolting upright' with their eyes wide open and a look of fear and panic on their face. They will often scream. Further, they will usually sweat, exhibit rapid respiration, and have a rapid heart rate (*autonomic signs*). In some cases, individuals are likely to have even more elaborate motor activity, such as a thrashing of limbs -- which may include punching, swinging, or fleeing motions. There is a sense that the individual is trying to protect themselves and/or escape from a possible threat which can lead to physical injury of the individual.[8] Although it seems like children are awake during a night terror, they will appear confused, be inconsolable and/or unresponsive to attempts to communicate with them, and may not recognize others familiar to them. Occasionally, when a person with a night terror is awoken, they will lash out at the person which can be dangerous for that individual.[6] Most people who experience this disorder are amnesic, or partially amnesic from the incident the next day.[5] Sleepwalking is another predisposition for the disorder.[12][7] Sleepwalking and night terrors are different manifestations of the same parasomnia disorder.[7]

During lab tests, subjects are known to have very high voltages of electroencephalography (EEG) delta activity, an increase in muscle tone, and a doubled increase in heart rate, if not more. Brain activities during a typical episode show theta and alpha activity when using an EEG. It is also common to see abrupt arousal from NREM sleep that

does not progress into a full episode of a night terror. These episodes can include tachycardia. Night terrors are also associated with intense autonomic discharge of tachypnea, flushing, diaphoresis, and mydriasis.[11]

There is a close association with psychopathology or mental disorders in adults that suffer from sleep terror disorder. There may be an increased occurrence of sleep terror disorder particularly with those that have suffered from post-traumatic stress disorder or PTSD and generalized anxiety disorder. Night terrors are closely linked to sleepwalking and frontal lobe epilepsy.[13] It is also likely that some personality disorders may occur in individuals with sleep terror disorder, such as dependent, schizoid, and borderline personality disorders. There have been some symptoms of depression and anxiety that have increased in individuals that have suffered from frequent night terrors. Low blood sugar is associated with both pediatric and adult night terrors.[8][14] Night terrors may cause children 12 and older to see paranormal substance, feel mentally and physically ill, and or commit suicide . A study of adults with thalamic lesions of the brain and brainstem have been occasionally associated with night terrors.[15]

Genetic and cultural features of night terrors

There is some evidence that a predisposition to night terrors and other parasomnia disorders can be congenital. Individuals frequently report that past family members have had either episodes of sleep terrors or sleepwalking. In some studies, a 10-fold increase in the prevalence of the disorder in first-degree biological relatives has been observed -- however, the exact link to inheritance is not known.[8] Familial aggregation has been found suggesting that there is an autosomal mode of inheritance.[16] In addition, some laboratory findings suggest that sleep deprivation and having a fever can increase the likelihood of a night terror episode occurring.[17] Other contributing factors include nocturnal asthma, gastroesophageal reflux, and central nervous system medications.[18] Special consideration must be used when the subject suffers from narcolepsy, as there may be a link between the disorders. There have been no findings that show a cultural difference between manifestations of Sleep Terror Disorder, though it is thought that the significance and cause of sleep terrors differ within cultures. Also, older children and adults provide highly detailed and descriptive images associated with their sleep terrors than younger children, who either cannot recall or only vaguely remember. Sleep terrors in children are also more likely to occur in males than females; in adults, the ratio between sexes are the same.[8] A longitudinal study of twins, both monozygotic and dizygotic were examined and found to show that a high concordance rate of night terror was found much more in monozygotic twins than in dizygotic.[19] [20]

Though the symptoms of night terrors in adolescents and adults are similar, the *etiology*, *prognosis*, and *treatment* are qualitatively different. There is some evidence that suggests that night terrors can occur if the sufferer does not eat a proper diet, does not get the appropriate amount or quality of sleep (e.g. sleep apnea), or is enduring stressful events in his or her life. Adult night terrors are much less common, and often respond best to treatments that rectify causes of poor quality or quantity of sleep. There is no scientific evidence of a link between night terrors and mental illness. There is some evidence of a link between adult night terrors and hypoglycemia.[21] According to Carranza and Dill (2004), some adult night terror sufferers share some characteristics with depressed individuals, e.g., "inhibition of aggression, self-directed anger, passivity, anxiety, impaired memory, and the ability to ignore pain."[22]

DSM-IV-TR diagnosis

The **DSM-IV-TR** diagnostic criteria for **sleep terror disorder** requires recurrent periods where the individual abruptly wakes from sleeping with a scream (*criterion A*), the individual experiences intense fear and symptoms of *autonomic arousal* (see autonomic nervous system) such as increased heart rate, heavy breathing, and increased perspiration, (*criterion B*), the individual cannot be soothed or comforted during the episode (*criterion C*), the individual is unable to remember details of the dream or details of the episode (*criterion D*), the occurrence of the sleep terror episode causes *clinically significant* distress or impairment in the individual's functioning (*criterion E*), and the disturbance is not due to the effects of a substance or general medical condition (*criterion F*).[10]

Adults

Night terrors in adults have been reported in all age ranges.[23] Though the symptoms of night terrors in adolescents and adults are similar, the etiology, prognosis and treatment are qualitatively different. These night terrors can occur each night if the sufferer does not eat a proper diet, get the appropriate amount or quality of sleep (e.g. sleep apnea), is enduring stressful events in their life or if they remain untreated. Adult night terrors are much less common, and often respond to treatments to rectify causes of poor quality or quantity of sleep. There is no scientific evidence of a link between night terrors and mental illness. A study done about night terrors in adults showed that psychiatric symptoms were prevalent in most patients experiencing night terrors hinting at the comorbidity of the two.[24] There is some evidence of a link between adult night terrors and hypoglycemia.[25] In addition to night terrors, some adult night terror sufferers have many of the characteristics of depressed individuals including inhibition of aggression, self-directed anger, passivity, anxiety, impaired memory, and the ability to ignore pain.[26][26][27][28][29] When a night terror happens it is typical that person can wake themself up screaming, kicking, and often can not make out what they are saying. Often the person can even run out of the house (more common among adults) which can then lead to violent actions.[30] It has been found that some adults who have been on a long-term intrathecal clonidine therapy show side effects of night terrors, such as feelings of terror early in the sleep cycle.[31] This is due to the possible alteration of cervical/brain clonidine concentration.[32] In adults, night terrors can be symptomatic of neurological disease and can further investigate through a MRI procedure.[33]

Children

The sleep disorder of night terrors typically occurs in children between the ages of three to twelve years, with a peak onset in children aged three and a half years old.[34] An estimated one to six percent of children experience night terrors. Boys and girls of all backgrounds are affected equally. The disorder usually resolves during adolescence.[34] Sleep disruption is parents' most frequent concern during the first years of a child's life. Half of all children develop a disrupted sleep pattern serious enough to warrant physician assistance. In children younger than three and a half years old, peak frequency of night terrors is at least one episode per week. Among older children, peak frequency of night terrors is one or two episodes per month. Children experiencing night terrors may be helped by a pediatric evaluation. During such evaluation, the pediatrician may also be able to exclude other possible disorders that might cause night terrors.[34]

Treatment

Reassuring a parent that this disorder will almost always outgrow the child is very important to treatment.[35] There is some indication that night terrors can result from being overtired, in which case interventions such as creating a bedtime schedule can increase the chances of restful sleep. If the night terrors are more chronic, however, some evidence suggests that the sufferer should be awakened from sleep just before the time when the terrors typically occur to interrupt the sleep cycle. In some cases, a child who has night terrors will require additional comfort and reassurance during the day and before bedtime. Psychotherapy or counseling can be helpful in many cases. Benzodiazepine medications (such as diazepam) used at bedtime will often reduce the occurrence of night terrors; however, medication is rarely recommended to treat this disorder.[36]

Prevalence of night terrors is unknown or unclear because there have been very few epidemiological studies over time.[37] [38] [39]

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External links

- Night Terror Resource Center (http://www.nightterrors.org/)
- National Institutes of Health, Medline Plus: Night Terrors (http://www.nlm.nih.gov/medlineplus/ency/article/000809.htm)
- National Library of Medicine - Medical Subject Headings: Night Terrors (http://www.nlm.nih.gov/cgi/mesh/2005/MB_cgi?index=18870)

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