Possible Influence on Subject(s)

Application of the microwave hearing technology could facilitate a private message transmission. It may be useful to provide a disruptive condition to a person not aware of the technology. Not only might it be disruptive to the sense of hearing, it could be psychologically devastating if one suddenly heard "voices within one's head."

Technological Status of Generator/Aiming Device

This technology requires no extrapolation to estimate its usefulness. Microwave energy can be applied at a distance, and the appropriate technology can be adapted from existing radar units. Aiming devices likewise are available but for special circumstances which require extreme specificity, there may be a need for additional development. Extreme directional specificity would be required to transmit a message to a single hostage surrounded by his captors. Signals can be transmitted long distances (hundreds of meters) using current technology. Longer distances and more sophisticated signal types will require more bulky equipment, but it seems possible to transmit some type of signals at closer ranges using man-portable equipment.

Range

The effective range could be hundreds of meters.

Incapacitating Effect: Disruption of Neural Control

The nature of the incapacitation is a rhythmic-activity synchronization of brain neurons that disrupts normal cortical control of the corticospinal and corticobulbar pathways; this disrupts normal functioning of the spinal motor neurons which control muscle contraction and body movements. Persons suffering from this condition lose voluntary control of their body. This synchronization may be accompanied by a sudden loss of consciousness and intense muscle spasms.

Biological Target/Normal Functions/Disease State

The normal function of the brain is to control all forms of behavior, voluntary control of body, and the homeostatic parameters of the organism. In normal conditions, all the brain structures, neuron populations, networks, and single units function with specific rhythmic activity depending on the incoming sensory information, information from mnemonic structures, and signals from visceral organs. Each single neuron provides specific processing of information it receives and forms a specific pattern of impulse firing as outgoing information. Synchronization of neuron activity is a natural mechanism of the brain function that uses such controlling processes as motivation, attention and memory (experience) in order to organize behavior. For example, motivational processes are considered as activating ascending signals that synchronize the neuron activity of specific brain structures and neuron networks; this activation/synchronization in turn activates specific forms of behavior such as sexual, aggressive, ingestive activities.

In normal functioning the degree of neuronal synchronization is highly controlled. From experiments that record the neuronal activity in different brain areas simultaneously in animals, it is known that correlation of spike activity between neurons (measured by the correlation level of synchronization) changes depending on the stage of behavior, motivation, attention, or activation of the memory processes. However, under some conditions, such as physical stress, heat shock, or strong emotional stress, the level of synchronization may become higher, involving nonspecific large populations of brain neurons and the synchronization may become uncontrollable.

Depending on at which frequency the synchronization rhythm occurs and how many neurons are involved, it may produce different physical effects; muscle weakness, involuntary muscle contractions, loss of consciousness, or intense (tonic) muscle spasms. The higher level of synchronization takes place in persons affected with epilepsy when they experience periodic seizures since they have a pathologic source (e.g., from injury to the brain) of rhythmic synchronization. Because the neurophysiological mechanisms of epileptiform synchronization are better documented, this incapacitating technology is described in terms of epileptogenesis.

The neurophysiological mechanisms active in epileptogenesis involve changes in membrane conductances and neurotransmitter alterations as they affect neuronal interaction. In the process of epileptogenesis, either some neurons are discharging too easily because of alterations in membrane conductances or there is a failure of inhibitory neurotransmission. The actual discharges have been recognized to result from a neuronal depolarization shift with electrical synchrony in cell populations related in part to changes in membrane conductances. The ionic basis and biochemical substrate of this activation have been areas of considerable study but still leave many questions unanswered. What are the basic cellular properties, present in normal cells and tissue, that could contribute to the generation of abnormal activity? What parts of the systems are low threshold and function as trigger elements?

One of the current hypotheses is involved with microcircuitry, particularly local synaptic interactions in neocortical and limbic system structures. In the hippocampus, the role of the trigger element has been long attributed to the CA3 pyramidal cells--a hypothesis based on the fact that spontaneous synchronous burst discharge can be established in CA3 neurons Some studies describe an intrinsically bursting cell type in the neocortex that plays a role similar to that of CA3 cells in the hippocampus and that of deep cells in the pyriform cortex. The intrinsic nature of these cells appears to be an important contributor to the establishment of synchronized bursting in these regions. Another apparent requirement in such a population is for a certain degree of synaptic interaction among neurons, such that discharge of even one cell enlists the activity of its neighbors. Given the presence of these bursting cells and the occurrence of excitatory interactions among them in normal tissue, it may actually be the morphologic substrate for epileptiform discharges.

Another hyptothesis has focused particularly on the role of N-methyl-D-aspartate (NMDA) receptors. Various factors regulate the efficacy of NMDA receptors: their voltage-dependent blockade by magnesium and modulation by glycine and polyamines. For example, in the low magnesium model, spontaneous synchronous burst discharge in hippocampal pyramidal cell populations is sensitive to NMDA antagonists. That finding suggests that it is the opening of NMDA channels, by relieving the magnesium blockade, that facilitates epileptiform activity.

Significant attention in the literature is also being given to gamma-amino butyric acid (GABA) receptors for the potential role in control of excitability. Changes in GABA inhibitory efficacy can lead to important effects on the excitability of the system. GABAergic inhibitory post-synaptic potentials (IPSPs) have been shown to be quite labile in response to repetitive activation of cortical cell populations, as may occur during epileptiform discharge. Scientists have shown that even a small percentage change in GABA inhibition can have profound effects on neocortical epileptogenesis. These changes in GABAergic inhibition may be the key to an explanation of how repetitive discharge patterns give rise to ictal discharge. Further, there appears to be a significant increase in excitatory postsynaptic potential (EPSP) frequency prior to seizure initiation an observation that is consistent with loss of IPSP efficacy prior to ictal onset.

The above hypotheses describe different mechanisms of epileptogenesis, but it is quite possible that all of these mechanisms take place, and they reflect large variety of types of epileptic seizures. The common principle of the mechanisms proposed is the change of membrane properties (i.e., conductance, permeability etc.) of certain neurons which results in depolarization and burst discharging. Some factors (e.g., trauma) can affect these specific neurons and initiate synchrony for neurons that control internal communication and communication with various muscle systems not associated with vital functions (i.e., heart beating, breathing). High strength pulsed electric fields could also be such a factor.

Mechanism to Reproduce the Desired Effects

Application of electromagnetic pulses is also a conceptual nonlethal technology that uses electromagnetic energy to induce neural synchrony and disruption of voluntary muscle control. The effectiveness of this concept has not been demonstrated. However, from past work in evaluating the potential for electromagnetic pulse generators to affect humans, it is estimated that sufficiently strong internal fields can be generated within the brain to trigger neurons. Estimates are that 50 to 100 kV/m free field of very sharp pulses (~ 1 nS) are required to produce a cell membranic potential of approximately 2 V; this would probably be sufficient to trigger neurons or make them more susceptible to firing.

The electromagnetic pulse concept is one in which a very fast (nanosecond timeframe) high voltage (approximately 100 kV/m or greater) electromagnetic pulse is repeated at the alpha brain wave frequency (about 15 Hz). It is known that a similar frequency of pulsing light can trigger sensitive individuals (those with some degree of light-sensitivity epilepsy) into a seizure and it is thought that by using a method that could actually trigger nerve synapses directly with an electrical field, essentially 100% of individuals would be susceptible to seizure induction. The photic-induced seizure phenomenon was borne out demonstrably on December 16, 1997 on Japanese television when hundreds of viewers of a popular cartoon show were treated, inadvertently, to photic seizure induction (figure 31). The photic-induced seizure is indirect in that the eye must receive and transmit the impulses which initially activate a portion of the brain associated with the optic nerve. From that point the excitability spreads to other portions of the brain. With the electromagnetic concept, excitation is directly on the brain, and all regions are excited concurrently. The onset of synchony and disruption of muscular control is anticipated to be nearly instantaneous. Recovery times are expected to be consistent with, or more rapid than, that which is observed in epileptic seizures.

Time to Onset

No experimental evidence is available for this concept. However, light-induced seizures latency onset in photosensitive epileptics varies from 0.1 to about 10 seconds. Because of the fact that the electrical impulses triggered by light must spread to other parts of the brain, photic-induced seizures are expected to have a generally slower onset than neural synchrony induced by high-strength pulsed electric fields.

Duration of Effect

For epileptic individuals, the typical duration of a petit mal event or a psychomotor event is 1 minute or 2, possibly longer, while the duration of a grand mal seizure is 1 to 5 minutes. In a non-epileptic individual who is induced by electromagnetic means, the durations of the different events are expected to be roughly the same as the epileptic individual's events after the external excitation is removed.

Tunability

There are many degrees of epileptic seizure in diseased persons, and it seems reasonable that electromagnetic stimulation of neural synchrony might be tunable with regard to type and degree of bodily influence, depending on the parameters associated with the chosen stimulus. Because there are no actual data to build on, these statements must be considered tentative. It is known that in the study of photic-induced seizures, parameters can be varied so that the individual under study does not actually undergo a grand mal seizure. This knowledge gives confidence that the proposed technology would be tunable.

Distribution of Human Sensitivities to Desired Effects

It is anticipated that 100% of the population would be susceptible. The mechanism is one that could act on many individual neuronal cells concurrently and hence does not depend on spreading regions of electrical activity as in the disease state.

Possible Influence on Subjects(s)

If the technology functions approximately as envisioned, the targeted individual could be incapacitated very quickly. Because there have been no reported studies using the conditions specified, experimental work is required to characterize onset time. Different types of technologies could be employed to influence wide areas or single individuals. Because this technology is considered to be tunable, the influence on subjects could vary from mild disruption of concentration to muscle spasms and loss of consciousness. The subject(s) would have varying degrees of voluntary control depending on the chosen degree of incapacitation.

Technological Status of Generator/Aiming Device

An electric field strength of roughly 100 Kv/m over a time period of 1 nanosecond is approximately the condition thought to be necessary to produce the desired effect when provided to an overall repetition rate of 15 Hz. Such a field may be developed using a radar-like, high-peak-power, pulsed source or an electromagnetic pulse generator operated at 15 Hz. These technologies exist today sufficient to evaluate the disabling concept. Power requirements are not high because the duty factor is so low. Aiming devices are currently available, but a high degree of directionality at long distances will require development. It may be necessary to provide bursts of these nanosecond pulses in order to stimulate the desired effect. As the duty time increases so does the average power requirement for power source. Because there were no open literature reports from which to make inferences, there is some uncertainty about the power levels required.

Range

The effective range could be hundreds of meters.

Defeat Capabilities/Limitations

Shielding can be provided by conductive barriers like metal or metal screen. There are a number of drugs that are capable of inducing convulsive seizures and others, like phenobarbital, diphenyllhydantoin, trimethadione, 2-4 dinitrophenol, and acetazolamide, which are anticonvulsive. Anticonvulsive drugs are known to be helpful in reducing the effect of seizures in epileptic patients, but their ability to reduce the effect of the proposed technology is unknown (possibly no effect) but expected to be less than for photic-induced seizures.

Incapacitating Effect; Acoustic Energy

The nature of the incapacitation consists of severe pressure sensations, nystagmus (a spasmodic, involuntary motion of the eyes), and nausea caused by high intensities of 9140-155 dB). Nystagmus occurs when convection currents are produced (cupula movement) in the lateral ear canal. This cupula movement causes the eyes to move involuntarily; hence, the external world is interpreted as moving. The subject "sees" his surroundings turning round him and at the same time experiences a sensation of turning. Persons exposed to these levels of sound experience nausea.

Biological Target/Normal Functions/Disease State

The two lateral semicircular canals, one located in each inner ear, alert a person to the fact that his upright head is experiencing angular acceleration. Within the ampulla of the canal are several so called hair cells. The cilia of these cells protrude into the lumen of the ampulla where they are encased in a mass of jelly-like material (the cupula) which is attached to the opposite wall of the canal. As the head accelerates, the cilia are bent by an inertial force of the cupula and the viscous liquid in the canal lumen. The bending of the cilia excites hair cells which in turn excite afferent neurons; these then alert the brain that a change of position of the head has occurred. Similar events occur when the head stops moving. The result of a strong hair cell stimulus to the brain is a rapid eye movement, call nystagmus, a feeling of dizziness and disorientation, and a possibility of nausea and vomiting.

Normal hearing is in the range between the frequencies of 20,000 to 16,000 Hz with the optimal sensitivity for most people between the frequencies of 500 to 6000 Hz.

Mechanism to Produce the Desired Effects

Because the end organs for acoustic and vestibular perception are so closely related, intense acoustic stimulation can result in vestibular effects. The hypothesis is that the sound of normal intensity produces oscillations of the endolymph and perilymph, compensated for by oscillations of the round window. High intensity sound produces eddy currents, which are localized rotational fluid displacements. High intensity sound can also produce nonlinear displacement of the stapes, causing a volume displacement, the result of which can be a fluid void in the labyrinth. To fill the void, fluid may be displaced along the endolymphatic duct and/or block capillary pathways, which, in turn, could stimulate vestibular receptors. Stimulation of the vestibular receptors may lead to nausea and vomiting if

the sound pressure level is high enough. Conclude that both eddy currents and volume displacement serve to stimulate vestibular receptors in humans, when exposed to high levels of noise.

One study found nystagmum in guinea pigs exposed to high levels of infrasound via stimulation of the vestibular receptors. However, the same lab was unable to produce nystagmus in human subjects at 5- and 10-second exposures to a pure tone at 135 dB, broadband engine noise, or a 100 Hz tone at 120 dB, pulsed three times/s or 2 minutes. The same research was unable to elicit nystagmus at levels up to 155 dB, and also equally unable to produce nystagmus using infrasound levels of 112-150 dB in guinea pigs, monkeys, and humans. However, research with audible components in the sound spectrum with guinea pigs and monkeys produced nystagmus. Other researchers report other vestibular effects in addition to nystagmus at the following thresholds: 125 dB from 200-500 Hz, 140 dB at 1000 Hz, and 155 dB at 200 Hz. Decrements in vestibular function occur consistently for broadband noise levels of 140 dB (with hearing protection).

Human subjects listened to very high levels of low-frequency noise and infrasound in the protected or unprotected modes. Two-minute duration as high as 140 to 155 dB produced a range of effects from mild discomfort to severe pressure sensations, nausea, gagging, and giddiness. Effects also included blurred vision and visual field distortions in some exposure conditions. The nature and degree of all effects was dependent on both sound level and frequency with the most severe effects occurring in the audible frequency range (as opposed to infrasound), at levels above about 145 dB. The investigators found no temporary threshold shift (TTS) among their subjects, and the use of hearing protectors greatly alleviated the adverse effects.

Since the early days of jet-engine testing and maintenance, anecdotal evidence has appeared linking exposure to intense noise, with such complaints as dizziness, vertigo, nausea, and vomiting. As a result of siren noise at 140 dB, subjects consistently reported a feeling of being pushed sideways, usually away from the exposed ear, and one subject reported difficulty standing on one foot.

These effects were not as dramatic as from the jet-engine (broadband) noise at 140 dB. This research concludes that the threshold of labyrinthine dysfunction is about 135 to 140 dB and that these effects occur during, but not after, exposure.

Time to Onset

No times to onset of nausea or nystagmus were identified in the literature but is presumed to be relatively immediate based on effects to the labyrinth system occurring during, but not after, exposure to sound pressure levels of 135 to 140 dB.

Duration of Effect

The incapacitation lasts only as long as the incapacitating sound is present.

Tunability

Based on the data presented above, it is unclear whether the degree of nausea or nystagmus is tunable, but similar symptoms caused by other stimuli are variable in degree.

Distribution of Human Sensitivities to Desired Effects

It is most probable that all individuals will be susceptible to this stimulus with the exception of those with a disease or defect (i.e., deaf mutes) of some part or parts of the vestibular system. Data showed no consistent decrease in vestibulo-ocular reflects with increased age.

Recovery/Safety

Normal subjects are likely to recover immediately and experience no or unmeasurable changes in hearing unless well known frequency-intensity-time factors are exceeded. This is based on studies which found no temporary threshold shift in hearing of subjects tested at low frequency. Occupational safety personnel generally recognize that 115 dB(A) is to be avoided and that 70 dB(A) is assumed safe. Is believed that the noise energy with predominating frequencies above 500 Hz have a greater potential for hearing loss than noise energy at lower frequencies. Occupational standards for noise state that a person may be exposed continuously for 8 hours to 90 dB(A) or 15 minutes to 115 dB(A).

Possible Influence on Subject(s)

Induction of nystagmus and nausea will have variable effects on individuals. Effects may be sufficiently incapacitation to allow offensive advantage; the perception of sickness may make a subject susceptible to persuasion. It would be difficult to target single individuals at the present level of sound directing technology. This technology may be better suited for groups of people.

Technological Status of Generator/Aiming Device

Sound generating technology is well developed but not highly portable. Aiming devices are poorly developed.

Range

Under normal circumstances the sound pressure level decreases 6 dB(A) when the distance from the source is doubled. For example if the sound is 100 dB(A) at 100 ft, at 200 ft the sound would be 94 dB(A). At very high sound levels, certain conditions may lead to nonlinear effects in propagation and greatly increase range accuracy.

Defeat Capabilities/Limitations

Negative effects of audible sound are greatly decreased if hearing protection is worn. High frequency sound is more easily blocked than low frequency sound due to wavelength effects.

Laser-Induced Biological Effects

Their are three basic damage mechanisms associated with exposure to laser radiation: chemical, thermal, and mechanical or acoustic-mechanical.

The laser-induced, chemical alterations in irradiated tissue are referred to as photochemical damage. The likelihood of laser radiation in the blue-light portion of the electromagnetic spectrum (.380 to .550 microns) inducing photochemical reactions progressively decreases with increasing wavelength. Photochemical effects are not observed upon exposure to radiation with wavelengths exceeding .550 to .650 microns because the kinetic energy associated with these photons is insufficient to initiate a photochemical change.

On the other hand, the thermal effect is a primary mechanism for laser-induced injury. The extent of the injuries induced depends upon the wavelength and energy of the incident radiation, duration of exposure, and the nature of the exposed tissue and its absorption characteristics. Generally, this mechanism predominates in the visible and the near-infrared (.760 to 1.4 microns) portions of the electromagnetic spectrum and for almost all CW and pulsed exposures between 0.1 milliseconds and 1 to 5 seconds.

The third injury mechanism associated with exposure to laser radiation is the mechanical or acoustical-mechanical effect. The radiant energy is absorbed into the tissue and, as a result of rapid thermal expansion following a short (1 nanosecond to 0.1 millisecond) laser radiation pulse, a pressure wave is generated that may result in explosive tissue injury.

Generally, all three mechanisms operate concurrently in an irradiated animal. Thermal effects currently predominate

for continuous wave (CW) lasers, while mechanical effects are of increased significance for pulsed-mode lasers. With even higher power, one must also consider nonlinear phenomena such as multiphoton absorption and electromagnetic field effects.

The organs most susceptible to external laser radiation are the skin and eyes. The severity of injury is affected by the nature of the target, the energy density delivered to the target, the frequency and power of the laser, atmospheric attenuation of the beam, and the use of filtering or amplifying optics by the target, etc.

The primary effect on the skin is thermal damage (burns). The severity varies from slight erythema or reddening to severe blistering or charring, depending on such factors as total energy deposition, skin pigmentation, and the tissue's ability to dissipate heat.

The eye is particularly susceptible to intense pulse of laser radiation because of its unique sensitivity to light. The focusing effect is similar to that of a magnifying lens, which focuses the energy on a particular spot. Since the cornea and lens of the eye amplify the intensity of the light incident upon the retina, the retina is extremely sensitive to visible and near-infrared light, and damage to the retina may result in temporary or permanent loss of visual acuity. Laser eye injuries vary according to incident power, spot size, beam angle, temporal mode (CW or pulsed), and pulse repetition frequency. Reported effects include corneal lesions, burns, cataracts, and retinal lesions.

Some high-power lasers can cause antipersonnel effects by the deposition of thermal energy. These lasers must operate at a wavelength that is readily absorbed by the skin or the cornea. These generally include the far- and mid-IR regions (10 to 12 microns and 3 to 5 microns) as well as the ultraviolet region (<0.4 microns). However, ultraviolet wavelengths generally do not propagate well in the atmosphere, so the primary threat wavelengths to be considered are between 3 and 12 microns. Although relatively modest amounts of far-IR laser power are required to produce superficial burns on the skin at short ranges, and efforts to design rheostatically lethal laser weapons are on going.

Nonlethal blinding laser weapons generally use collimated beams with very low beam divergence, and the energy contained in the beam diminishes relatively slowly over great distances. Imaging systems such as eyes and EO vision systems have focusing optics that bring the incident plane wave of light to focus at the sensor plane. This results in a high optical gain (greater than 100,000 for eyes), which makes the associated sensor vulnerable to relatively low fluences of laser energy.

The effects of lasers on eyes are threefold:

- Dazzling or induced glare.
- Flashblinding or loss of night adaptation.
- Permanent or semipermanent blinding.

The severity of laser eye injuries varies according to the incident power, spot size, beam angle, pupil diameter (ambient light conditions), temporal mode (CW or pulsed), and PRF of the laser. Reported effects include corneal burns, cataracts (a permanent cloudiness of the lens), and retinal burns and perforations. Low-energy laser weapons are capable of causing the latter.

Exposure to relatively low laser energies can produce temporary changes in the ability to see without producing permanent injury. Exposure to laser light can produce an effect called glare or dazzle, which is similar to the temporary loss of vision experience when viewing the headlights of an oncoming car. The visual effects last only as long as the light is present in the field of view (FOV). At slightly higher energy exposures, the same laser radiation can saturate or flashblind the photoreceptor cells, resulting in after images that fade with time after exposure. Only visible radiation will induce veiling glare or after images; near-IR radiation will not produce these effects even though the radiant energy reaches the photoreceptor cells. Flashblindness and dazzle, while not permanent injuries, can cause discomfort and temporary loss of vision. Some studies have shown that dazzle and flashblindness can