into a house through an open window, a chimney, or a crack under a closed door.

Some of our experiments on acoustic excitation of sensory resonances which provide a basis for the present invention will be discussed presently. Of all the responses to the excitation of the ½ Hz resonance, ptosis of the eyelids stands out for distinctness, ease of detection, and sensitivity. When voluntary control of the eyelids is relinquished, the eyelid position is determined by the relative activities of the sympathetic and parasympathetic nervous systems. There are two ways in which ptosis can be used as an indicator that the autonomic system is being affected. In the first, the subject simply relaxes the control over the eyelids, and makes no effort to correct for any drooping. The more sensitive second method requires the subject to first close the eyes about half way. While holding this eyelid position, the eye are rolled upward, while giving up voluntary control of the eyelids. With the eyeballs turned up, ptosis will decrease the amount of light admitted to the eyes, and with full ptosis the light is completely cut off. The second method is very sensitive because the pressure excerted on the eyeballs by partially closed eyelids increases parasympathetic activity. As a result, the eyelid position becomes somewhat labile, exhibiting a slight flutter. The labile state is sensitive to small shifts in the activities of the sympathetic and parasympathetic systems. The method works best when the subject is lying flat on the back and is facing a moderately lit blank wall of light color.

The frequency at which ptosis is at a maximum is called the ptosis frequency. This frequency depends somewhat on the state of the nervous and endocrine systems, and it initially undergoes a downward drift, rapid at first and slowing over time. The ptosis frequency can be followed in its downward drift by manual frequency tracking aimed at keeping ptosis at a maximum. At a fixed frequency, the early ptosis can be maintained in approximately steady state by turning the acoustic stimulation off as soon as the ptosis starts to decrease, after which the ptosis goes through an increase followed by a decline. The acoustic stimulation is turned back on as soon as the decline is perceived, and the cycle is repeated.

At fixed frequencies near ½ Hz, the ptosis cycles slowly up and down with a period ranging upward from about 3 minutes, depending on the precise acoustic frequency used. The temporal behavior of the ptosis frequency is found to depend on the acoustic pulse intensity; the drift and cycle amplitude are smaller near the low end of the effective intensity window. This suggests that the drift and the cycling of the ptosis frequency is due to chemical modulation, wherein the chemical milieu of the neural circuits involved affects the resonance frequency of these circuits, while the milieu itself is influenced by the resonance in delayed fashion. Pertinent concentrations are affected by production, diffusion, and reuptake of the substances involved. Because of the rather long characteristic time of the ptosis frequency shift, as shown for instance by the cycle period lasting **3** minutes or longer, it is suspected that diffusion plays a rate-controlling role in the process.

The resonance frequencies for the different components of the ½ Hz sensory resonance have been measured, using acoustic sine waves with a sound pressure of 2×10^{-9} N/m^2 at the subject's left ear. Ptosis reached a steady state at a frequency of 0.545 Hz. Sexual excitement occurred at two frequencies, 0.530 Hz and 0.597 Hz, respectively below and above the steady-state ptosis frequency. For frequencies of 0.480 Hz and 0.527 Hz the subject fell asleep, whereas tenseness was experienced in the range from 0.600 to 0.617 Hz.

The resonance near 2.5 Hz may be detected as a pronounced increase in the time needed to silently count backward from 100 to 70, with the eyes closed. The counting is done with the "silent voice" which involves motor activation of the larynx appropriate to the numbers to be uttered, but without passage of air or movement of mouth muscles. The motor activation causes a feedback in the form of a visceral stress sensation in the larynx. Counting with the silent voice is different from merely thinking of the numbers, which does not produce a stress sensation, and is not a sensitive detector of the resonant state. The larynx stress feedback constitutes a visceral input into the brain and may thus influence the amplitude of the resonance. This unwanted influence is kept to a minimum by using the count sparingly in experiment runs. Since counting is a cortical process, the 2.5 Hz resonance is called a cortical sensory resonance, in distinction with the autonomic resonance that occurs near ½ Hz. In addition to affecting the silent counting, the 2.5 Hz resonance is expected to influence other cortical processes as well. It has also been found to have a sleep inducing effect. Very long exposures cause dizziness and disorientation. The frequency of 2.5 Hz raises concerns about kindling of epileptic seizures; therefore, the general public should not use the 2.5 Hz resonance unless this concern has been laid to rest through further experiments.

The sensitivity and numerical nature of the silent count makes it a very suitable detector of the 2.5 Hz sensory resonance. It therefore has been used for experiments of frequency response and effective intensity window. In these experiments, rounded square wave acoustic pulses were produced with a frequency that was slowly diminished by computer, and the subject's 100–70 counting time was recorded for certain frequencies. The acoustic transducer was a small loudspeaker mounted in a sealed cabinet such as to provide acoustic monopole radiation. At fixed frequency, the acoustic monopole strenght in m^3/s varies linearly with the voice coil current, with a constant of proportionality that can be calculated from measured speaker dome excursions for given currents. The sound pressure level at the entrance of the subject's nearest external ear canal can be expressed in terms of the acoustic monopole strength and the distance from the loudspeaker. For each experiment run, the sound pressure level at the entrance of the subject's external ear canal can thus be calculated from the measured amplitude of the voice coil current and the pulse frequency. Since for the subaudio frequencies the distance from the acoustic radiator to the subject's ear is much smaller than the wavelength of the sound, the near-field approximation was used in this calculation. The sound pressure level was expressed in dB relative to the reference sound pressure of 2×10^{-5} N/m^2. This reference pressure is traditionally used in the context of human hearing, and it represents about the normal minimum human hearing threshold at 1.8 KHz.

FIG. **9** shows the result of experiment runs at sound pressure levels of −67, −61, −55, and −49 dB. Plotted are the subject's 100–70 counting time versus pulse frequency in a narrow range near 2.5 Hz. Resonance is evident from the sharp peak **57** in the graph for the sound pressure level of −61 dB. The graphs also reveal the effective intensity window for the stimulation, as can be seen by comparing the magnitude of the peaks for the different sound pressure levels. For increasing intensity, the magnitude of the peak first increases but then decreases, and no significant peak shows up in the graph for the largest sound pressure of −49 dB; this can be seen better from the insert **58**, which shows the graphs for −67 and −49 dB in a magnified scale. It follows that the effective intensity window extends approxi-