Testicular weight is normally maintained only by photoperiods of 12.5 hours or more (*6*), yet the hamsters subjected to the 36-hour or 60-hour cycles received only one 6-hour light period every 1½ or 2½ days, respectively. Thus, whatever the concrete physiological system that measures photoperiodic time in the hamster, it does not measure the absolute duration of light, the absolute duration of darkness, or the ratio of light to darkness. In our judgment, these data can be reasonably interpreted only with reference to Bünning's hypothesis. The effect of light on the hamster's reproductive system depends primarily on the relation of the light period to the phase of the circadian rhythm of sensitivity. In the 24- and 48-hour cycles, light is present only during the insensitive phase of this rhythm and yields a "short-day" response; in the 36- and 60-hour cycles it is present during the sensitive phase of the rhythm and so maintains testicular weight (Fig. 1).

This interpretation gains support from the locomotor activity data (Fig. 2). We can arbitrarily divide the hamster's day into an active phase (beginning with the onset of activity and ending 12 hours later) and an inactive phase (the time remaining until activity onset the next day). In those light cycles allowing testicular regression (24- and 48-hour cycles), only the inactive phase receives light. In those light cycles (36- and 60-hour cycles) that maintain testis weight, light is present in both the early and late portions of the active phase. We cannot determine from these data the duration of the photosensitive portion of the cycle; the early portion of the active phase, the late portion, or both may contribute to the photoperiodic response. To put it more generally, we do not know the form or amplitude of the circadian rhythm of photosensitivity which underlies photoperiodic time measurement. We do know that such a rhythm exists.

In view of the role of the pineal gland in mediating the effects of light on the reproductive response of hamsters (*5*), the data presented herein suggest that pineal function is regulated by a circadian rhythm of photosensitivity. Unfortunately, this suggestion will be difficult to test experimentally.

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16. Hamsters (6 weeks old) were obtained from Lakeview Hamster Colony, Newfield, New Jersey. The animals were initially housed six to eight per cage in a room and were maintained on LD 14 : 10 at room temperature until well after sexual maturity was attained. Before the experiment began, the animals were placed two per cage, two or three cages per light-controlled box. Each box was illuminated with a clock-controlled fluorescent bulb [Ken-Rad, 4 watts (F4T5/cw)] that produced an intensity of 50 to 100 lux at the floor of each cage. On day 47 of the experiment, seven animals of each group were killed; the remainder were housed one per cage for the rest of the experiment. Food (Purina laboratory chow) and water were continually available.
17. This experimental design, one form of the "resonance" experiment, was first employed by K. C. Hamner (*10*).
18. The 36-, 48-, and 60-hour cycles were controlled by Flexopulse timers (Eagle Signal Co., Davenport, Iowa), the periods of which could be set with only limited accuracy. As a result these light cycles had period lengths approximately 1 to 3 minutes shorter than intended (for instance, the 60-hour cycle was in actuality 59 hours 57 minutes). Thus the gradual shift to the left in the phase of activity onset reflects the gradual drift to the left in the phase of the light cycle.
19. We thank S. Siddiqui and L. Mostafavi for technical assistance. Supported by NIH grants HD-03803 and GM-00836 and NIH career development award HD-9327 to M.M.

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Spontaneous Middle Ear Muscle Activity in Man: A Rapid Eye Movement Sleep Phenomenon

Abstract. *Changes in compliance of the tympanic membrane have been detected in normal human sleep, presumably due to spontaneous contraction of the stapedius and tensor tympani muscles of the middle ear. In the waking state, these muscles generally respond to loud sound (middle ear reflex). Middle ear muscle activity typically erupts before or at the onset of rapid eye movement (REM) sleep and persists throughout the REM period in a discontinuous pattern resembling that exhibited by rapid eye movements. Approximately 80 percent of all nocturnal middle ear muscle activity is contained in REM sleep. Half of the remaining 20 percent occurs in the 10-minute intervals just prior to the onset of REM sleep. Middle ear muscle activity is often associated with other phasic events such as momentary enhancement of electromyogram inhibition, apnea, and K complexes. Rapid eye movements and middle ear muscle activity, though significantly correlated in REM sleep, are not always simultaneous.*

The commencement of rapid eye movement (REM) sleep in mammals is associated with a number of distinct alterations in physiological processes. These may be categorized into two major classes, tonic and phasic (*1*). Tonic phenomena, such as desynchronization of the electroencephalogram (EEG) and active inhibition of muscle tone, are continuous throughout the course of the REM period and define its temporal limits (*2*). Phasic phenomena, which generally commence at the onset of REM sleep, are short-lived and occur intermittently until termination of the REM stage.

Phenomena that erupt episodically in REM sleep have been described (*3*).

In view of the vivid dreaming that characterizes REM sleep, several studies have attempted to correlate certain phasic events (for example, REM's, limb movements, respiratory rate irregularities) with specific hallucinated sensations and activities of the dreamer (*4*). In search of a physiological indicator of auditory imagery during dreaming, preliminary investigations of tympanic membrane motility in sleep, which employed a different technique than that used in this study, were undertaken in our laboratory (*5*). These initial efforts were not successful, but with utilization of acoustic impedance recordings we report here a phasic discharge, spontaneous middle ear muscle activity (MEMA), hitherto undemonstrated in human sleep.

In the awake individual, reflex contractions of the middle ear muscles, the stapedius and tensor tympani, may be elicited by loud sound (70 to 90 db, hearing threshold level) as well as by tactile stimulation in the area of trigeminal innervation (*6, 7*). The middle ear muscles are among the shortest striated muscles in the body. Only they and the extraocular muscles have slow and twitch muscle elements (*8*) and share in possessing the highest known proportion of nerve to muscle fibers. The tensor tympani originates in the cartilaginous portion of the eustachian tube and, after crossing the middle ear cavity, inserts at the manubrium of the malleus; the stapedius emerges from the pyramidal eminence in the posterior aspect of the cavity and connects to the stapes near the footplate. The motor arm of the middle ear reflex is innervated by cranial nerves V and VII, the former supplying the tensor tympani and a ramification of the latter, the stapedius. In man and other animals, activity in one or both muscles influences the compliance of the mechanical system comprising the tympanic membrane and ossicular chain of the middle ear. This anatomical arrangement constitutes a mechanism for modulation of sound energy conducted from the external environment to the cochlea.

Middle ear muscular activity has been studied by means of direct muscle implantation in cats. In the sleeping animal, the middle ear muscles are active during 60 percent of total REM sleep and 14 percent of nonrapid eye movement (NREM) sleep (*9*). The phasic contractions in one or both middle ear muscles, which appear during REM sleep (*9, 10*) as well as during the orienting reflex (*10*), cause a reduction in the cochlear neural response to sound input, measured at the round window. These intermittent reductions disappear after the muscles are sectioned (*10*). In humans, Djupesland (*6*) has shown that a highly reliable correlate of implanted electrode registration of MEMA is the compliance variation of the tympanic membrane, measured with acoustic impedance techniques (Madsen electroacoustic bridge).

In this study of spontaneous MEMA in human sleep, we employed an advanced model (ZO-70) of the Madsen instrument. The probe of this instrument was mounted in a soft plastic individually cast ear mold fitting snugly into the external auditory canal, close to but not in contact with the eardrum (*11*). A baseline level of sound pressure at 220 hertz was introduced into the space between mold and tympanic membrane. It measured 95-db sound pressure level (SPL), reflecting the resting acoustic impedance of the system. The SPL changes when, as a result of MEMA and its effect on the ossicles of the middle ear, the tympanic membrane moves or even varies in compliance. This change in SPL is recorded by means of the acoustic bridge and may be directed to a paper writeout, in our case a Beckman type R polygraph.

The acoustic impedance recording in the absence of MEMA shows a rhythmically pulsating baseline, presumably of vascular origin, coincident with heart rate. For purposes of scoring, we accepted as MEMA deflections in excess of 2 mm (20 mv) from this baseline. With the Madsen bridge (position 3), which was sensitive at 0.03 cm^3/volt in a 1-cm^3 space, connected to a 9806A Beckman coupler set at a time constant of 0.3 second and with a calibrated 100 mv/cm amplification factor, a 2-mm pen deflection represented the volumetric change resulting from an eardrum displacement of 0.6 $\times 10^{-3}$ cm^3.

Because certain physical maneuvers can create shifts in acoustic impedance similar to those caused by MEMA, we instituted controls to discriminate actual MEMA from MEMA-like artifacts. For example, swallowing, murmuring, snoring, talking, and the valsalva may alter air pressure in the middle ear through the eustachian tube and thus change the compliance of the eardrum. In addition, facial movements larger than small twitches, associated with action of the temporomandibular joint, may cause variation in the compliance of the lateral walls of the external auditory canal.

Accordingly, for purposes of differentiating changes in acoustic imped-

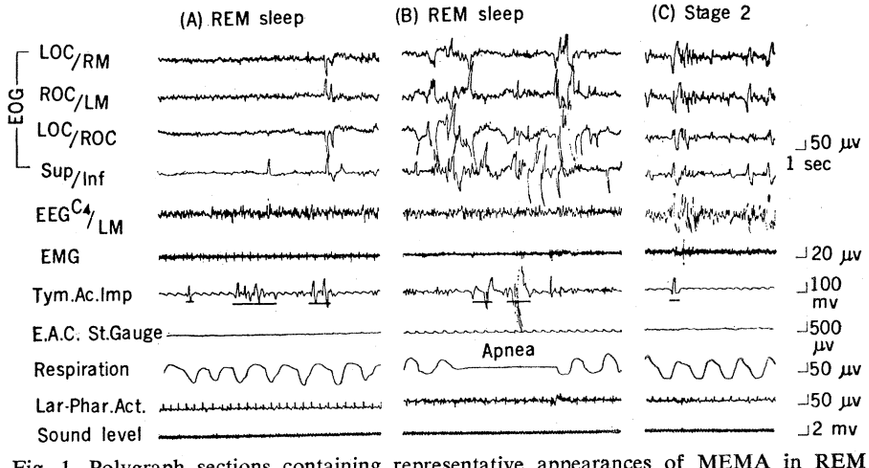

Fig. 1. Polygraph sections containing representative appearances of MEMA in REM and NREM sleep. (A) REM sleep, MEMA with and without concurrent rapid eye movements. (B) REM sleep, MEMA associated with a period of apnea. (C) Stage 2 sleep, MEMA simultaneous with a K complex and slight laryngeal activation. Electrooculogram (EOG) horizontal leads: LOC/RM, left outer canthus referred to right mastoid; ROC/LM, right outer canthus to left mastoid; LOC/ROC, left outer canthus to right outer canthus; EOG vertical leads: Sup/Inf, supraorbital ridge referred to infraorbital ridge. Electroencephalogram (EEG) leads: C_4/LM right central to left mastoid. Electromyogram (EMG) leads: masseter muscle referred to submentalis placement. Tympanic acoustic impedance (Tym.Ac.Imp.) deflections underlined, 100 mv = 12.5 acoustic ohms. External auricular canal strain gauge (E.A.C. St.Gauge) mounted on ear mold. Laryngopharyngeal activity (Lar.Phar-Act.) recorded from laryngeal prominence of thyroid cartilage. Sound level monitored from sleep room (2 mv = 7.7 db). Activity from possible sources of MEMA-like artifact is absent during acoustic impedance deflections, indicating that they represent true MEMA.

ance generated in sleep by spontaneous MEMA from artifactual impedance changes, electrodes and sensors were placed at several critical points. A strain guage was mounted longitudinally along the axis of each ear mold to indicate movement within the external auditory canal not of eardrum origin; the chin-to-cheek electromyogram (EMG) electrodes were positioned on the same half of the head as the ear under study in order to record jaw and neck muscle activity from that side; electrodes over the larynx indicated swallow, vocalization, and the other laryngopharyngeally mediated functions; a thermocouple placed over the nares registered breathing rate; and the sleep room, though sound-deadened to the level of a clinical audiological testing suite (single-wall type), was constantly monitored for adventitial sound. All changes in acoustic impedance during sleep were deleted from consideration if accompanied by simultaneous deflections on channels indicating more than slight laryngopharyngeal activity, movement of the somatic musculature or of the walls of the external auditory canal, vocalization, or external noise (*12*).

We considered the possibility that additional unknown sources of impedance variation might be responsible for a substantial proportion of the activity that we accepted as true MEMA. However, we obtained results that rendered this possibility unlikely when examining for a conceivable generator of such artifact. We studied a patient whose middle ear muscles had been removed by bilateral tympanoplasty. In this individual the only impedance deflections recorded in sleep were those associated with discharges from other recording sites, signaling snoring, swallowing, or jaw movement. In other words, all sources of MEMA-like artifact that could potentially add to our tallies of MEMA were already being routinely monitored.

In this study, five subjects (two female and three male, ages 20 to 38) were audiologically examined for normal hearing as well as for a functioning middle ear response. Wearing the standard electrode array used for physiological sleep recording, the subjects were monitored in the laboratory during sleep with the ear molds, containing the impedance probe, rendered airtight by surgical adhesive. After several acclimatization nights, two additional nights of data were collected from each subject and analyzed.

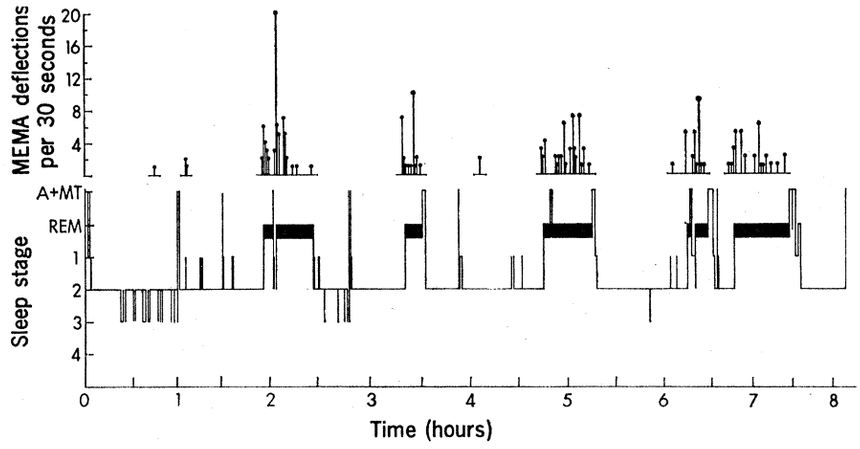

Fig. 2. The typical distribution of MEMA (top) in relation to sleep stage pattern in a normal night of sleep (bottom) in man; Awake plus movement time, A + MT. In the top section the height of each vertical bar corresponds to the number of acoustic impedance deflections per 30-second epoch. Note the prevalence of spontaneous MEMA during REM periods.

We found that MEMA was present in every subject studied and in every REM period (Table 1). Frequently MEMA appeared in advance of the conventionally scored REM periods or with the start of REM sleep before commencement of REM's. The acoustic impedance deflections in sleep closely resembled those seen in the awake state but were generally larger. They occurred singly and in clusters in a discontinuous pattern similar to the eruptions of REM's during REM sleep (Fig. 1). The duration of a single deflection was 0.3 to 0.7 second and ranged in amplitude from 2.5 to 43.5 acoustic ohms. Clusters usually lasted less than 10 seconds but occasionally endured 20 seconds or longer. Bilateral recordings from both ears in two additional subjects demonstrated temporal concordance with rare exception.

Often, MEMA was accompanied by small phasic twitches in the facial musculature or by phasic enhancement of muscle inhibition. In REM sleep, acoustic impedance variations were commonly associated with sawtooth waves and occasionally with apnea, sometimes without concurrent rapid eye activity (Fig. 1, A and B). In stage 2, concordances of MEMA with K-complex EEG slow waves, or phasic EMG inhibition, or both, though striking when observed, were not consistent.

Approximately 80 percent of total nocturnal MEMA was located within REM periods. The remaining 20 percent appeared mainly in stage 2 sleep. Average MEMA per minute was 15 times higher in REM sleep than in NREM sleep. Considerable between-subject variability was apparent in terms of total amount and frequency of

Table 1. Middle ear muscle activity (MEMA) and stage of sleep. Calculations based on a total sleep time of 400 minutes (M, male; F, female).

Subject	Night	REM sleep (%)	Total MEMA			MEMA per minute	
			Deflections (No.)	In REM sleep (%)	In NREM sleep (%)	REM sleep (No.)	NREM sleep (No.)
S.L. (M)	1	24	200	89.8	10.2	1.87	0.07
	2	23	226	95.1	4.9	2.38	0.04
L.C. (M)	1	17	266	86.1	13.9	3.44	0.11
	2	22	265	88.3	11.7	2.79	0.10
H.P.R. (M)	1	18	181	81.2	18.8	2.06	0.10
	2	23	323	88.2	11.8	3.13	0.12
L.P. (F)	1	22	748	71.8	28.2	6.14	0.68
	2	20	818	72.7	27.3	6.76	0.71
C.K. (F)	1	14	62	67.7	32.3	0.75	0.06
	2	13	51	63.5	36.5	0.65	0.05
Mean (± S.E.)		19.6 (±1.2)	314.5 (±82.9)	80.4 (±3.4)	19.6 (±3.4)	3.0 (±0.6)	0.2 (±0.1)

MEMA; however, all within-subject measures (nights 1 and 2) were remarkably stable in four of the five individuals studied (Table 1) (*13*).

Almost half of all MEMA during NREM sleep appeared in the 10-minute intervals preceding the onset of each REM period. The frequency of MEMA in these 10-minute segments rose almost linearly with the approach of REM sleep. Within the REM period, the first half contained almost two-thirds of the MEMA of the period. A typical distribution of MEMA in relation to the REM-NREM cycle is portrayed in Fig. 2. That MEMA in man heralds the REM period, builds to an early crescendo, and diminishes in frequency before the end of the REM period is reminiscent of the temporal distribution in the cat of the primary brainstem phasic event, the pontogeniculooccipital spike.

In order to determine the distribution of ear activity relative to REM's, the first four REM periods of each subject were divided into 1.25-second epochs and scored for the presence or absence of initial deflections of MEMA and REM's. In the five subjects, 3.0 ± 0.8 percent of total REM sleep epochs contained MEMA (*14*) and 19.5 ± 3.3 percent contained REM's. When MEMA and REM densities were examined in sequential REM periods for possible time-of-night influences, an almost parallel pattern emerged. The densities of the two episodic activities in the second and third REM periods were almost twice the values in the first REM period. The REM densities in the fourth REM period remained high but MEMA densities diminished to a level intermediary between the first and succeeding two REM periods (*15*).

Although the number of epochs positive for REM's was more than six times greater than the number positive for MEMA, REM deflections were not simultaneous with MEMA in 44.0 ± 0.8 percent of the epochs containing middle ear discharge (simultaneity defined as occurrence within the same 1.25-second epoch) (*16*). However, correlation coefficients for the incidence in REM sleep of the two phasic phenomena were between .11 and .23 ($P < .01$ in every subject). These findings, taken together, indicate that though motor ear and eye activities are not randomly associated in REM sleep, neither are they inexorably linked. It follows that when MEMA recording is not undertaken, episodes of REM sleep free of eye movement and twitch activity may be mistakenly thought to represent tonic intervals (*17*).

The existence in REM sleep of a spontaneous phasic motor activity related in the waking state to auditory and tactile sensation affords once again the opportunity of studying the possible connection of "inner" perceptual events with physiological events during dreaming sleep. It is of interest to us, therefore, to study the nature of the subjective phenomena experienced in sleep in association with MEMA. We have some evidence that indicates that more auditory imagery is reported when MEMA is present, although more data are necessary to establish a definitive relationship.

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12. This procedure operated to omit from our counts instances of actual MEMA that may have occurred in conjunction with movements of throat structures, neck, or head. Of the total acoustic impedance episodes in REM sleep, no more than 10 percent were of this combined discharge type.
13. Since the electro-acoustic method of registering MEMA necessitates the input of a constant sound (though at a level below that required to provoke the middle ear reflex), we felt it possible that the totals and stage-related proportions of MEMA during sleep were influenced by the sound. Therefore, an additional subject was recorded twice, once by acoustic impedance and once by a tympanometric technique that required no sound input but utilized the air pressure changes due to movement of the tympanic membrane, see K. Terkildsen, *Arch. Otolaryngol.* **66**, 484 (1957). The MEMA values obtained by the two recording methods were very similar.
14. This proportion of total REM sleep in which MEMA occurred, as well as the per minute frequency of MEMA (see Table 1), was scored from a-c recording of acoustic impedance. The a-c deflections mark the incidence of initial and secondary changes in MEMA but do not provide a measure of sustained activity. This type of data is not comparable to the percents of sleep stage time containing MEMA, which were reported in cats (*9*). The latter were calculated from direct muscle recordings or from the cochlear neural response and reflect the total time that any MEMA, varying or sustained, was monitored. In the absence of direct middle ear muscle recording in man, d-c recording of acoustic impedance, though less sensitive than direct electromyography, may provide a parallel measure.
15. Densities in percent of REM sleep time in REM periods 1, 2, 3, and 4 for MEMA were 2.5, 4.4, 4.0, and 2.9 and for REM's were 9.3, 18.5, 22.4, and 18.1.
16. Eye movements were recorded by electro-oculography (gain, 10 mm = 50 μv) from vertical and horizontal electrode pairs. Strain gauges placed on the upper eyelids, were used additionally and confirmed that REM's were not present in over 40 percent of the instances of MEMA.
17. The finding of a partial asynchrony in phasic motor ear and eye activities is consistent with the possibility of more than one primary source of phasic activation operating in the brainstem as part of the REM sleep mechanism. However, these data do not rule out the possibility that in REM sleep a single brainstem generator source may always deliver simultaneous phasic activation to both motor systems. For the partial asynchrony of REM's and MEMA does not necessarily indicate that the primary brainstem discharges, which originate activation in the different systems, are not synchronous. It indicates only that the end motor responses recordable with our transducers (for example, eyeball deflections and changes in eardrum tension) are not simultaneous. It is conceivable that impulses transmitted in parallel from a unitary generator into several neuronal channels may eventuate as *apparently* asynchronous peripheral phasic events. For example, selective inhibition may come into play along only one of several neuronal pathways transmitting impulses from a primary activating source to different motor systems. This could result in complete inactivity in one end structure, because of failure to reach its threshold for movement, concomitant with generous activity in another. A mixture of partial and complete inhibitions variously affecting different motor pathways could thus account for the entire range of relative amplitudes observed in the two sets of end phenomena. Even given equal transmission of impulses in the brainstem pathways leading from the source generator, a further contribution to end organ asynchrony may be the unique peripheral mechanics of the different motor systems (that is, different efficiencies in the translation of brainstem discharges into structural displacements).
18. We thank Dr. R. Ruben (Department of Otorhinolaryngology, Albert Einstein College of Medicine) for providing critical advice concerning electro-acoustic impedance recording and sound conduction in the middle ear; Dr. F. Michel (University of Lyon) for suggesting an important control; D. Vertes and S. Lamstein for technical assistance; Irwin Penzack (Bronx Hearing Aid Center) and Scientific Plastics for fabricating the specialized earmolds used in this study, and I. Klar (Madsen Electronmedics) for supplying us with the Madsen ZO-70 electro-acoustic impedance bridges.
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