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Middle and long latency auditory evoked potentials in cat. II. Component distributions and dependence on stimulus factors

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The middle (10–50 ms) and long (50–600 ms) latency periods of the auditory evoked potential (AEP) were investigated in muscle-paralyzed, artificially respired cats with respect to two issues: (1) the distribution of components across the skull, and (2) the effects of changing stimulus intensity on component latencies and amplitudes. The distributional data were gathered during a behavioral study in which four behavioral tasks related to classical pupillary conditioning were used to vary attentional and arousal processes. The distributions across the skull surface (averaged across tasks) of 12 peaks and troughs (P10, N13, P17, N22, P31, N41, P55, N70, N100, N140, P260 and N520) and seven principal components derived from the set of waveforms collected during this experiment are reported. Both peak amplitudes and principal component scores were distributed differentially across the skull surface. In the second experiment, acoustic stimulus intensity was varied, and AEPs collected from a vertex and temporal electrode site. In general, increasing stimulus intensity had a stronger influence on the earlier portions of the AEP, where increased amplitude and decreased latency was the rule, than on later ones. The relationships between cat and human AEP components were discussed based on both the data presented in this paper and in previous papers.

Key words: auditory evoked potential; cat; middle latency; long latency; distribution.

Introduction

There are three criteria for making comparisons of auditory evoked potentials (AEPs) that can be obtained with relative ease from both humans and non-human species. First, the amplitudes, latencies and polarities of various components of the AEP can be examined and compared. Second, the 'functional relationships' of the AEP can be defined, including the effects of stimulus and behavioral manipulations on component amplitude and latency. Finally, the topographic distribution of components can be compared between species.

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In previous papers [6,36] we examined the latency, amplitude and polarity of the cat AEP, along with the effects of behavioral manipulations on these measures. In this paper we report on the distribution of the cat AEP components across the skull surface, and examine the effects of stimulus intensity on components of the cat AEP. The results are considered in formulating the correspondences between cat and human AEP components of middle and long latency.

Methods

Surface distribution of AEP components

The methods used to derive the data on skull surface distributions of cat AEP components have been completely described in a companion paper [6].

Stimulus intensity series

Signal intensity effects on AEPs were studied in unanesthetized cats that were paralyzed and artificially respired. Seven electrode sites (V1, V2, V4, I3, C3, IL1, CL1) were selected for recording since these seemed particularly representative of the data collected for the entire electrode array. Connections for recording data on the tape recorder and for monitoring animal state were made as in the previous study [6].

After preparation, the animals were allowed about 15 min to adapt to the situation; for the most part, they went to sleep, and remained asleep, during the remainder of the procedure. An intensity series for clicks was run starting at 70 dB attenuation and increasing intensity by 10 dB steps until 0 dB attenuation (113 dB peak SPL) was reached. The increasing order of intensities was chosen to minimize carry over of adaptation and fatigue effects from one intensity to another. For each intensity, 200 clicks were delivered, one per 999 ms. After changing intensities, the animals were allowed about 30 s to adjust to the new stimulus level before recording was again initiated.

When the 0 dB attenuation clicks had been recorded, approximately 1 min of silence was instituted to allow the animals to readapt to low levels of stimulation. Then, a noise intensity series was started. Sets of 150 noise bursts (500 ms duration, one per 2.997 s) were delivered at a given intensity. The longer period between stimuli was to minimize adaptation and fatigue. Intensities were changed in 10 dB steps from 70 to 0 dB attenuation (83 dB SPL), with 30 s adjustment periods given when intensities were changed.

Data were retrieved from tape and digitized in a manner similar to that described for the behavioral study [6], except that a sample of 200 clicks and 150 noise bursts were used to form the AEPs. Further analysis, including score calculation and statistical analyses, will be described in appropriate portions of the results sections.

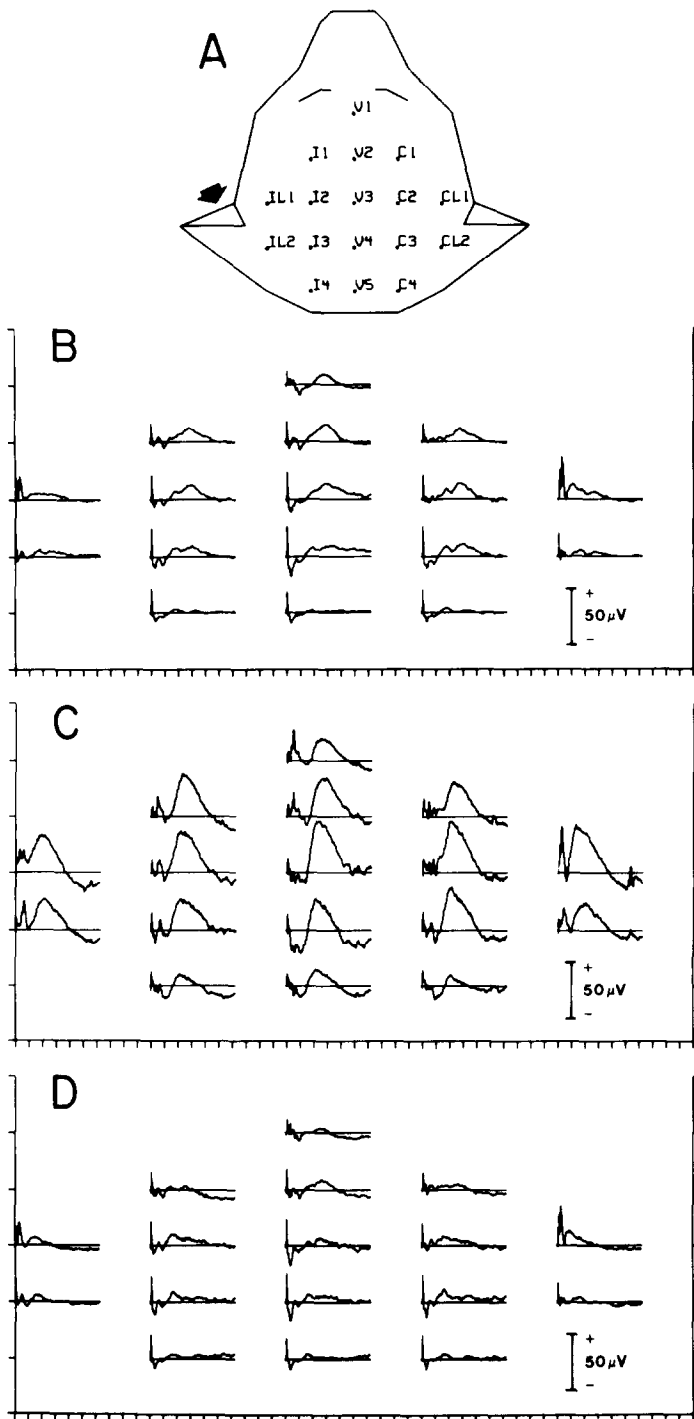


Fig. 1. Distribution of the AEP raw waveforms. AEP waveforms have been averaged across animals and behavioral conditions for each electrode location. These are plotted spatially along a linear time base, starting at stimulus onset, with abscissa ticks separated by 100 ms and ordinate ticks representing 50 μ V. A diagram of electrode locations and names is provided in plate (A). The arrow indicates the stimulated ear. AEP waveform distributions are plotted for (B) pre-noise clicks, (C) noise stimuli, and (D) post-noise clicks.

Results

Qualitative skull surface distribution (raw waveforms)

In Fig. 1, AEPs averaged across animals and behavioral conditions are displayed on a linear time base for the 17 electrodes. Pre-noise click AEPs are shown in Fig. 1B, noise AEPs in Fig. 1C, and post-noise AEPs in Fig. 1D. The different stimuli evoked waveforms that were quantified as to both peak amplitude and PCA component scores over the scalp.

Distribution of components (peak measures)

Fig. 2 shows isopotential distributions ($2.5 \mu\text{V}$ spacings) of typical peaks and troughs, averaged across subjects, conditions and stimuli. Positivity is indicated by shading. N13, P17 and N22 are quite similar in topographical distribution, although with different absolute voltages. Because of this similarity, only the N22 distribution

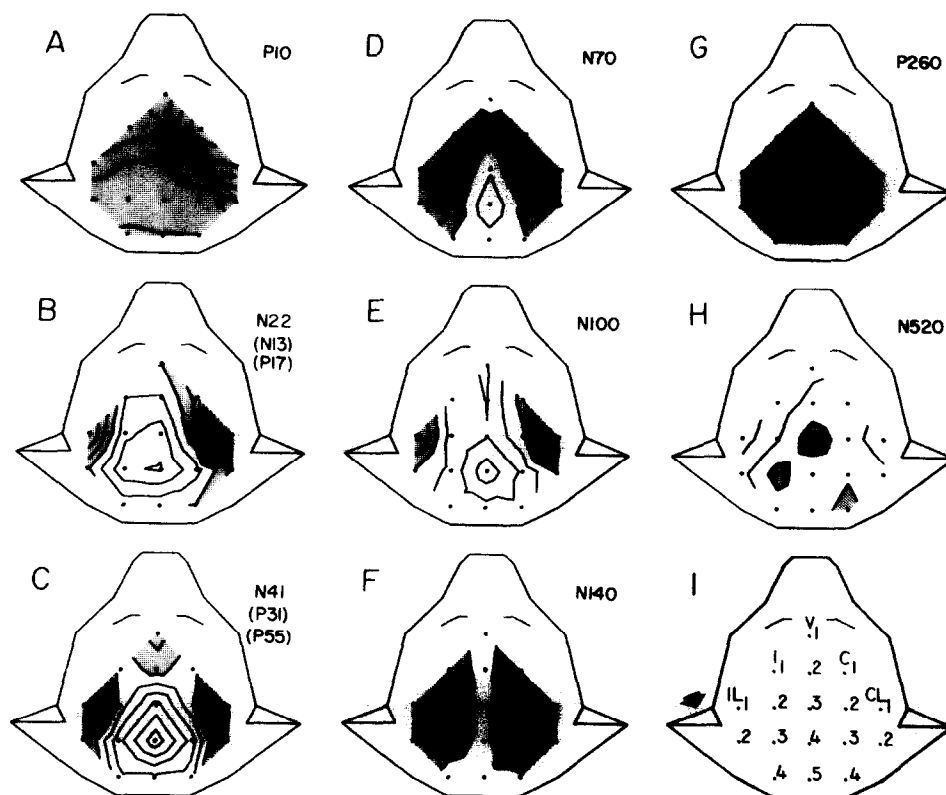


Fig. 2. Topographic distribution of voltages of the AEP at latencies of selected peaks. Iso-potential contours are plotted for representative AEP waveform peaks and troughs in plates A-H. Regions of positive voltage are shaded; negative voltages are unshaded. Contours are separated by $2.5 \mu\text{V}$. Plate I provides electrode location names. The acoustic stimuli are delivered monaurally to the left ear, as indicated by the arrow.

is plotted. Likewise, P31, N41 and P55 showed distribution patterns that were qualitatively similar, and thus only the N41 pattern is shown.

To summarize, P10 (Fig. 2A) has a broadly distributed positivity having an anterior to posterior distribution, and a maximum over the C1 electrode. The next three peaks and troughs (Fig. 2B) have high amplitude positivities located over the IL1 and CL1 electrodes, with the contralateral site being larger, and an asymmetrical region of negativity, centered over the midline (V4). N41 (and associated P31 and P55) are symmetrically distributed (Fig. 2C), with three regions of positivity centered over IL1, CL1 and V1, and a region of negativity centered over V4. N70 (Fig. 2D) shows two relatively symmetrical regions of negativity centered over V1 and V4, but positive regions are centered over different electrodes, IL2 and CL1, giving a somewhat diagonal distribution. N100 (Fig. 2E) has a broadly distributed negativity with maxima on the midline (V1 and V4), and regions of positivity centered over IL1 and CL1, with the latter being of larger amplitude. N140 (Fig. 2F) shows a relatively symmetrical distribution, with regions of strong positivity over IL1 and CL1, and a weak band of negativity running down the midline. As can be seen in Fig. 2G, P260 is a broadly distributed, symmetrical positivity centered over the V3 electrode; no electrode showed a negative voltage for this peak. Finally, N520 (Fig. 2H) has a broadly distributed negativity centered over the IL1 and CL1 electrodes (the former being largest), with islands of positivity at V3, I3 and C4.

Distribution of components (PCA)

A complementary view of the distribution of these waveforms is provided from PCA. The distribution of mean component scores (averaged across cats and conditions) across the electrode array is shown in Fig. 3A–G. Iso-score contours have been interpolated between them. Regions of positive scores are shaded.

The first three components can be characterized by their asymmetrical distribution. Component 1 (Fig. 3A) displays a strong region of positive scores lying bilaterally over the auditory areas in temporal cortex, but stronger over the hemisphere contralateral to the stimulated ear. Component 2 (Fig. 3B) also shows an asymmetrical bilateral distribution with strongest positive scores over the contralateral hemisphere. However, this contralateral positivity has moved posteriorly, and a negative score area has developed over CL1, giving a strongly asymmetrical distribution. Component 3 (Fig. 3C) again shows an asymmetrical distribution, in this case with positive scores over the ipsilateral posterior electrodes (especially IL2) and negative scores over the contralateral and frontal electrodes (especially V1 and CL1).

The next three components have relatively symmetrical distributions about the midline. Component 4 (Fig. 3D) displays a negative score region on the midline centered over V2, with bordering regions of positive scores maximum over the IL1 and CL1 electrodes, slightly larger on the contralateral side. By contrast, component 5 (Fig. 3E) has a symmetrical, broadly distributed positive score region, again centered over V2, which falls off gradually in the posterior, as well as lateral, directions. Component 6 (Fig. 3F) displays a broadly distributed, posteriorly centered region of positive scores surrounded anteriorly and laterally by regions of negative component scores.

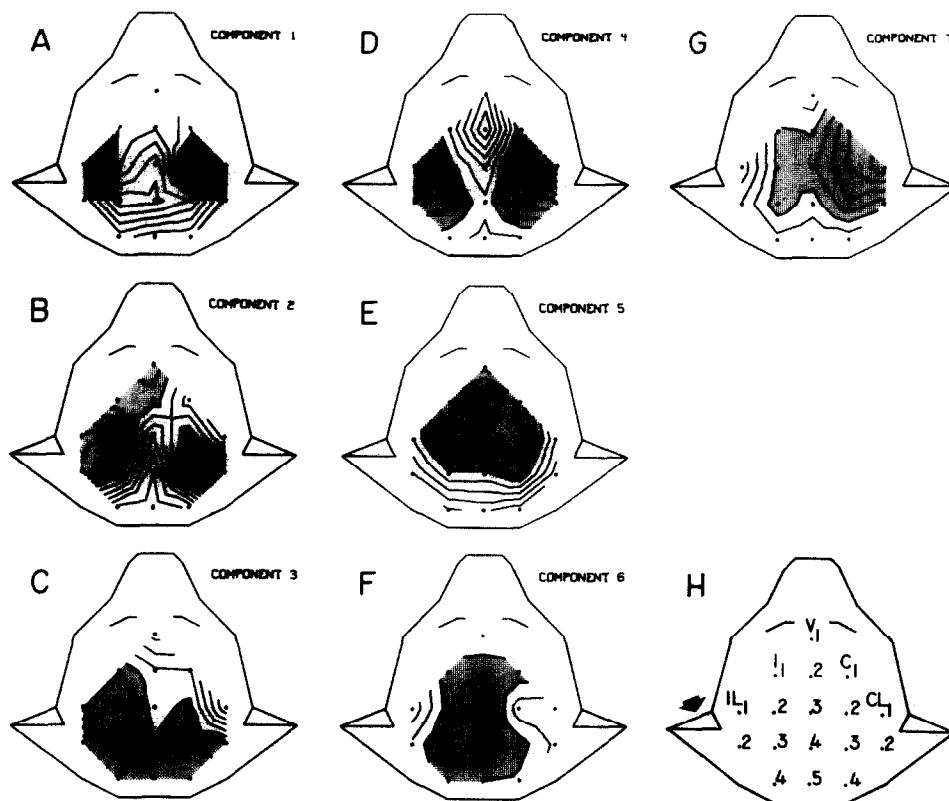


Fig. 3. Topographic distribution of principal component scores. Iso-component score contours are plotted for each of the seven principal AEP components in plates A–G. Regions of positive scores are shaded; negative scores are unshaded. Contour spacing corresponds to a change in component score of 0.01. Plate H provides electrode location names. The acoustic stimuli are delivered monaurally to the left ear, as indicated by an arrow.

Finally, Fig. 3G shows that component 7 presents a strongly asymmetrical, lateralized distribution, with positive scores centered over CL1 and negative scores over IL1. This suggests that this component is either relatively unique to the contralateral temporal regions or inverted between the two hemispheres. Since this component has maximum loading at a latency earlier than component 4, one might suggest an overall pattern of change from an asymmetrical to a symmetrical distribution between shorter and longer latencies.

Results from the intensity series

Fig. 4 shows the effects of increasing stimulus intensity on the AEP. Data from different cats have been averaged together and are shown for two different electrodes, V1 (Fig. 4A, B) and CL1 (Fig. 4C, D), in response to clicks (Fig. 4A, C) and noise stimuli (Fig. 4B, D). In general, increasing stimulus intensity was associated with increased response amplitudes, and decreased peak latencies.

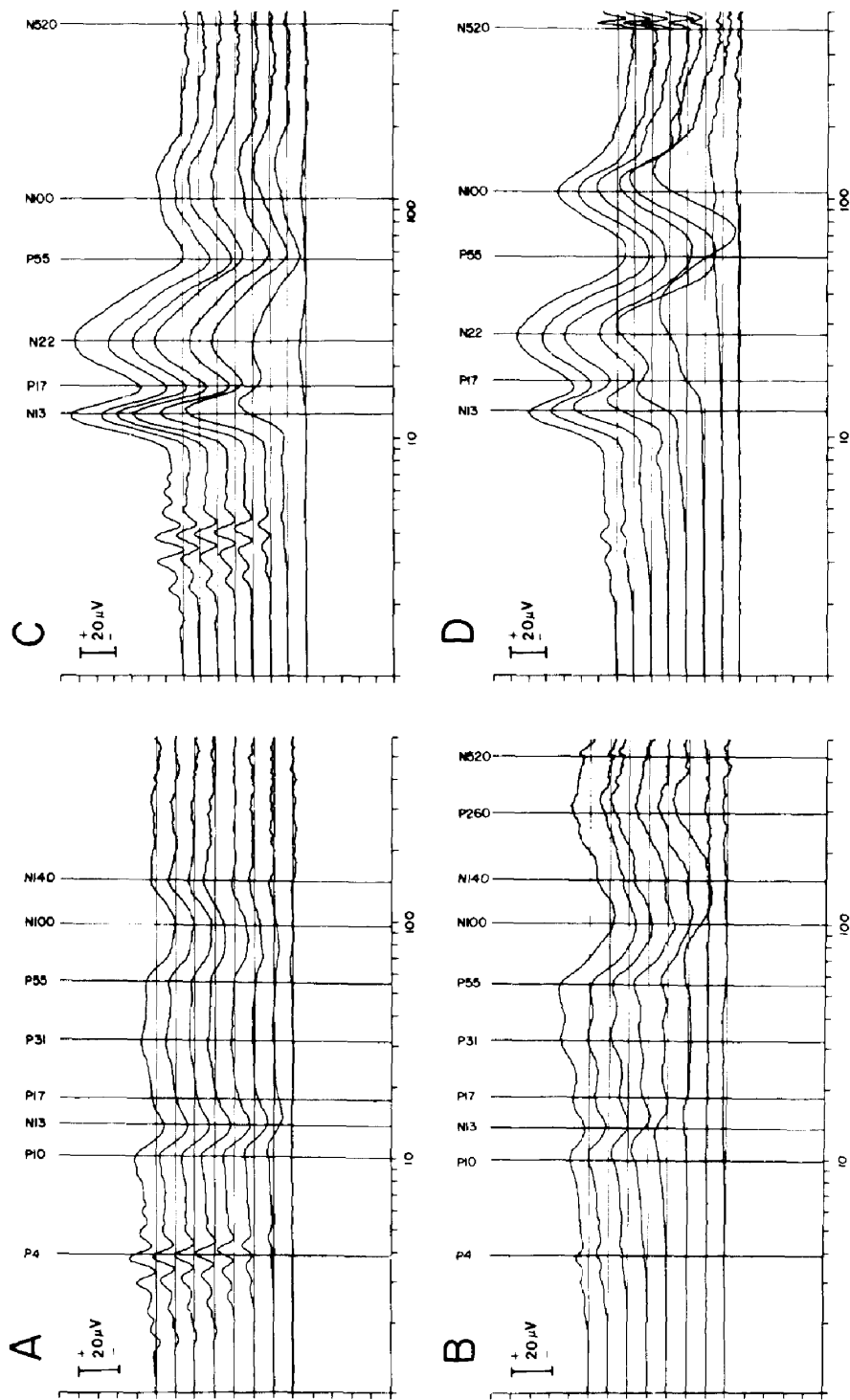


Fig. 4. Stimulus intensity series for the AEP at two electrode locations. AEPs have been averaged across animals to give one waveform for each intensity (0-70 dB attenuation range for clicks, 0-60 dB attenuation for noise, relative to the stimuli used in the behavioral study). These are plotted in order of stimulus intensity, with the uppermost trace being 0 dB attenuation. Lines are drawn in the figure to show which peaks were chosen for measurement and subsequent statistical analysis, along with the labels used to identify the peaks so measured. Abscissa units are ms, ticks on the ordinate represent 10 μ V. (A) Click AEPs, V1 electrode. (B) Noise AEPs, V1 electrode. (C) Click AEPs, CL1 electrode. (D) Noise AEPs, CL1 electrode.

TABLE I
ANOVA OF AEP PEAK MEASURES AS A FUNCTION OF STIMULUS INTENSITY

lat. = latency; ampl. = amplitude.

Peak	V1 electrode			CL1 electrode		
	Click (3,18 df)			Click (3,18 df)		
	lat.	ampl.	Noise (3,15 df)	lat.	ampl.	Noise (3,15 df)
P4	211.94	23.41	48.91	-	-	-
	< 0.001	< 0.001	< 0.001			
P10	14.66	15.63	34.73	-	-	-
	< 0.001	< 0.001	< 0.001			
N13	9.59	2.47	7.92	24.98	5.81	6.51
	< 0.001		< 0.01	< 0.001	< 0.05	< 0.01
P17	12.07	0.54	7.19	10.64	1.55	0.65
	< 0.001		< 0.01	< 0.001		
N22	-	-	-	0.97	12.70	9.77
					< 0.001	< 0.001
P31	3.05	9.99	8.33	-	-	-
		< 0.001	< 0.01			
P55	3.09	4.15	12.81	1.35	0.93	2.31
		< 0.025	< 0.001			
N100	0.70	6.12	5.26	8.93	10.07	0.70
		< 0.01	< 0.025	< 0.001	< 0.001	
N140	0.24	0.94	4.92	-	-	-
			< 0.025			
P260	-	-	0.69	-	-	-
N520	-	-	1.21	0.50	0.47	0.62

Peak latencies and amplitudes were determined in each waveform of each cat, and subjected to a one-way repeated measures ANOVA, the results of which are shown in Table I. Earlier latency peaks (less than 100 ms) typically showed clearer latency and amplitude changes than did later ones. Because of the obvious parametric latency shifts in the data, a complementary PCA analysis is not presented.

Discussion

In this paper we have described the surface distribution of a series of AEP components recorded from cats during behavioral testing, and effects of stimulus intensity on these components. The components of the cat AEP will now be compared to human AEP components on the basis of both the current data and data presented previously [6,36] concerning their waveshape and behavioral correlates.

Correspondences between cat and human AEPs

Early components (ABR). Cat ABR components showed little variation in their distribution across the skull, but were strongly determined, in both amplitude and latency, by stimulus variables including type (noise versus click) and intensity, as previously reported for humans [11,27]. In previous work, we found little variation attributable to behavioral variables [6], similar to humans, whose ABRs are immutable even during drug induced coma [33].

P10 through N45 (principal components 1 and 2). We previously noted the similar waveshape of cat and human AEPs at latencies between 10 and 45 ms [6], and the fact that these cat components were little affected by behavioral task [6], similar to human AEPs [19], although some reduction in amplitude had been reported for deeper sleep in humans [19–21]. Stimulus intensity changes strongly influenced these components' amplitudes, both in this study, and in comparable human studies [16–18,35]. Much the same was true of the effects of long- versus short-duration acoustic stimuli [15,23,29,37]. Buchwald et al. [3] investigated the waveform morphology, stimulus rate-dependence and barbiturate sensitivity of a similar set of cat AEP components, with results also suggesting strong functional similarity between cat and human AEPs at these 'middle' latencies.

Both principal component 1 and its encompassed peaks were localized over auditory cortical areas, and data from Kaga et al. [12] indicate that, for cat, at least some middle latency activity depends on the structural integrity of auditory cortex. In contrast, middle latency components of the human AEP have a frontal and central distribution [27]. However, in the cat, the waveforms at the lateral electrode sites were sufficiently different from those recorded at V1 to raise a question as to their identity. Different middle latency waveforms between midline and temporal sites have also been observed in humans and lower primates [1,4,5,8].

Currently, only tentative peak for peak assignments between cat and human middle latency AEPs might be postulated: cat P10 to human P12, cat P31 to human

P30, and cat N41 to human N45. Occasionally, human N20 is further differentiated into an N22–P25–N28 complex [19], which could correspond to the N13–P17–N22 complex in the cat. However, these peaks and troughs appear regularly in cats [3,6], whereas they are not a constant feature of the human middle latency response.

P55 (principal component 2). Cat P55 resembles human P50 in latency and polarity [6]. It also shows parameter amplitude changes with stimulus intensity, and is more strongly evoked by longer duration stimuli, in similarity to human P50s [13,27]. Buchwald et al. [3] report that this peak (their ‘wave C’) is strongly affected by stimulus rate and by pentobarbital anesthesia, as is human P50. Further, the current study shows that cat P55 is largest over the frontal and auditory cortices, and thus has a similar distribution to that described for human P50 by Peronnet et al. [24] and Goff et al. [8]. Of the criteria available, the only major discrepancy between cat P55 and human P50 is that cat P55 does not precede a large N90 wave, where human P50 does.

N140 (principal component 4). On the basis of behavioral criteria, N140 and its related principal component 4 were thought related to human N300, although their absolute latencies are quite different [6]. The symmetry of N140’s distribution, with an anterior midline negativity, is similar to human N300 [27]. However, cat N140’s large temporal positivities are quite unlike human N300 [27]. In cat, N140 is weakly influenced by stimulus intensity, but is larger for noise than for click stimuli.

P260 (principal component 5). Cat P260 is markedly similar in its latency and polarity [6], in its behavior during stimulus probability [6,36] and modality changes [36], and in its relationship to behavioral variables (attention and arousal) [6], to P300 recorded in humans [9,25,30,32]. Furthermore, a component of similar latency can be obtained in cat to a missing stimulus, as can human P300 [31]. Cat P260’s broadly distributed positivity (centered on the midline in regions posterior to somatosensory-motor but anterior to visual areas) is quite similar to human P300’s distribution [8]. Most stimulus parameter manipulations had little effect on cat P260, although noise bursts, in the intensity series, evoked a more detectable peak than did clicks. This peak was approximately the size of that seen during habituation in the behavioral study, and thus was much smaller than that seen when noise was task relevant.

N520 (principal component 5). We previously noted [6] that this component was difficult to relate to any single human component of similar latency. The relatively broadly distributed region of negativity seen in this paper, together with its stimulus independence, does little to clarify these uncertainties.

N70 and N100 (principal components 3 and 7). Major components of the human vertex response, N90 and P170, do not have clear counterparts in the cat [6]. N100 in cats was distributed over temporal areas, while N70 had a posterior midline negativity as well as temporal positivities. Thus, neither component showed the

frontal and central distribution reported for human N90 and P170. The strong, but saturating effect of increasing stimulus intensity on N100 amplitude resembles that described for human N90 [23,27].

We offer three possible explanations for the absence of human N90–P170 analogs in this study in cats. First, these components might be obscured by our recording techniques, especially the stimulus delivery rate and recording bandpass, which differ from those used to record optimally the human vertex response [2]. However, human N90 and P170 have been recorded under conditions similar to this study, so this explanation seems unlikely.

Second, N90 and P170 analogs might have been present, but unrecognized. Temporal overlap with other components might be responsible, as when human P170 is obscured by the ‘N200 mismatch negativity’ [22,28]. Alternatively, species brain geometry differences, especially in auditory cortical areas, which face dorsally in man but laterally in cat, might be to blame. If N90 and P170 are generated in the temporal areas [8,14,24], then one might expect the frontal and central distribution in humans to become a lateral distribution in cats. Of interest in this light is the slow potential shift (SPS), which is seen over the vertex in humans, while its most obvious cat analog is recorded over temporal cortex [13]. It is thus possible that component 7, a multiphasic wave peaking around 100 ms, corresponds to N90, P170, or both.

Finally, the cat may simply lack N90 or P170. These components’ frontal distribution in humans has suggested to some researchers that they are generated in frontal cortex [26,27], although recent studies [14] indicate that humans with extensive frontal lobe ablations show normal N90–P170 amplitudes. The cat certainly has little frontal cortex, and if it is responsible for generating N90–P170, these peaks might be lacking.

Relation to other animal experimental studies

Numerous animal studies relating AEP features to such processes as habituation, attention, arousal and conditioning have been previously reported, and several reviews are available [10,13,26,34]. These efforts studied mainly short latency AEP components recorded from particular structures, and thus cannot be easily related to the more recently defined human AEP recorded from scalp.

Several studies have dealt with the issue of correspondence more directly. Keidel [13] suggested, mainly on waveform morphology criteria, that the major peaks of the cat auditory cortical AEP correspond with P50, N90 and P170 of the human vertex response, while the peri-stimulatory d.c. shift in this region corresponds to the human SPS. Goff et al. [7] found an N70–P125 complex which was largest on the midline and grew in amplitude when cats entered slow wave sleep, suggesting a relationship with the human K-complex (and thus, perhaps N300). Arrezo et al. [1] intracranially mapped AEPs in rhesus monkeys, and found seven distinct components, most of which inverted near the superior temporal plane, with some additional activity just anterior to the central sulcus. They cautiously equated P12 with human P12, P22 with P30, N38 with N45, P52 with P50, N70 with N90, P110 with P170, and N140 with N300, mostly on the basis of peak latency and distributional similarities between species. Working with anesthetized, paralyzed cats, Kaga et al.

[12] reported a component of about 15 ms latency that was strongly dependent on stimulus parameters, and appeared to be generated in auditory cortex. Buchwald et al. [3], on the basis of stimulus rate, barbiturate sensitivity and brain lesion effects, suggested the correspondence of cat vertex recorded 'wave 7' (10–12 ms latency) to human P12, 'wave A' (17–27 ms latency) to human P30, and 'wave C' (50–75 ms latency) to human P50. Finally, Squires and Buchwald [31] reported four components enhanced during an eyeblink conditioning paradigm. A 300 ms latency component, which was controlled by stimulus probability and task relevance and could be elicited by a missing stimulus, was thought related to human event-related endogenous components.

The present and former studies [1,3,7,12,13,31] indicate that a long-lasting, complex series of AEP components can be recorded from the scalps of experimental animals, and that these components might have certain correspondences in AEPs recorded from the human scalp. Our initial efforts in cat to define these correspondences in terms of signal parameters (intensity), surface distribution, and behavioral variables [6,36] suggest that certain components may indeed be comparable. The use of animal models for AEP analysis by lesion and depth recording techniques can provide knowledge of the anatomical and physiological substrates of scalp recorded events that may well have applications in humans.

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