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RESEARCH ARTICLE

G. Loquet · K. Meyer · E. M. Rouiller

Effects of intensity of repetitive acoustic stimuli on neural adaptation in the ventral cochlear nucleus of the rat

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Abstract To study neural adaptation as a function of stimulus intensity, auditory near-field evoked potentials were recorded from the ventral cochlear nucleus in awake Long Evans rats. Responses to 250-ms trains of repetitive clicks (pulse rates ranging from 100 to 1000 pulses per second) were collected at stimulus intensities of 5, 10, 30, 50 and 70 dB SPL. The amplitude of the first negative (N_1) component of the average evoked potentials to individual pulses in the train was measured by using a subtraction method. The N_1 responses were normalized with respect to the highest cochlear nucleus potential observed in the train, and then plotted as a function of click position in the train. As expected, the general trend of the curves was an exponential decay reaching a plateau more or less rapidly as a function of both intensity and rate of stimulation. Fitting these curves with exponential decay equations revealed that the rapid time constant decreased for increasing stimulus intensities whereas the short-term time constant is relatively independent of intensity. The amount of adaptation (expressed as the ratio of the plateau to the first peak amplitude) was substantially less prominent at low intensities (5–10 dB SPL) and low rates (100–200 pulses per second) than at higher intensities and high rates. These results indicate that adaptation patterns obtained in the ventral cochlear nucleus by using near-field evoked potentials exhibit properties comparable to those already present at the level of the auditory nerve.

Keywords Unanesthetized · Brainstem · Auditory evoked potentials · Click

G. Loquet · K. Meyer · E. M. Rouiller (✉)
Division of Physiology, Department of Medicine, University of Fribourg,
Rue du Musée 5,
1700 Fribourg, Switzerland
e-mail: eric.rouiller@unifr.ch
Tel.: +41-26-3008609
Fax: +41-26-3009675

Introduction

From a psychophysical point of view, the subjective intensity of a pure-tone that lasts more than a few seconds and does not exceed 30 dB SL (sensation level) decreases during stimulation. This loudness decrease, referred to as adaptation, is due to the reduction of neural response to continuous stimulation over time (Gelfand 1997). In profoundly deaf patients, however, the current generation of cochlear implants does not reproduce adaptation properties, so imparting a loss of information which may contribute, at least in part, to the commonly reported low level of speech intelligibility in noisy conditions.

Experimental studies on auditory adaptation at the neural level have generally been conducted in animal models (cat and rodent) by recording compound action potentials (Eggermont and Spoor 1973; Abbas 1984; Chimento and Schreiner 1990, 1992) or action potentials from single auditory nerve fibers (Smith and Zwislocki 1975; Smith 1977, 1979; Harris and Dallos 1979; Westerman and Smith 1984; Rhode and Smith 1985; Yates et al. 1985; Javel 1996). In the auditory nerve (AN), the compound action potential (CAP) in response to a transient sound is characterized by a negative deflection, reflecting the synchronized discharges of several individual fibers. In response to a variety of different stimulus types such as high frequency tones (Gorga and Abbas 1981; Abbas 1984), low frequency tones (Chimento and Schreiner 1990, 1991, 1992), short repetitive tone-bursts (Peake et al. 1962a, 1962b; Eggermont and Spoor 1973; Müller and Robertson 1991), or click trains (Kiang et al. 1965; Wickesberg and Stevens 1998), both techniques (CAP and single unit recordings) yielded similar results, namely an initial rapid decrease of CAP amplitude or firing rate during the first few milliseconds (*rapid adaptation*), followed by a slower decay over tens of milliseconds (*short-term adaptation*), and then an even slower decrease over several seconds (*long-term adaptation*) or minutes (*very long-term adaptation*). The similarity of adaptation patterns to pure tones and to series of clicks could possibly be attributed to the fact that

at high repetition rates, the latter stimulus is close to a cosine-phase harmonic complex (Hafter and Richards 1988). Nevertheless, small differences were reported at the single unit level, especially between high and low spontaneous rate AN fiber populations (Rhode and Smith 1985; Müller and Robertson 1991), suggesting that different mechanisms may underlie adaptation. Moreover, it is important to note that the measures of adaptation were essentially performed in two ways: either over the duration of the stimulus (Peake et al. 1962a, 1962b; Eggermont and Spoor 1973; Westerman and Smith 1984; Müller and Robertson 1991; Javel 1996) or during the recovery period (Gorga and Abbas 1981; Abbas 1984; Chimento and Schreiner 1990, 1991, 1992). However, both approaches are closely related, and thus lead to comparable results (rapid, short-term, and long-term adaptation). Similar to the AN, the near-field evoked potential recorded from the cochlear nucleus (CN) in response to transient sounds is characterized by a negative deflection (with a longer latency), believed to reflect the synchronized discharges of secondary auditory neurons (Møller 1983). With this approach, the measurements of adaptation in the ventral division of the CN (VCN), carried out with short repetitive tone-bursts (Møller 1969; Huang and Buchwald 1980; Huang 1981) or click trains (Møller 1969; Loquet and Rouiller 2002), showed an adaptive pattern similar to that of the AN, with at least three distinct decay components. At the single unit level, only two of the three major response types described in the VCN (primary-like and chopper) exhibited adaptation patterns similar to those reported in the auditory nerve fibers in response to tones (Evans 1975). The same conclusion was reached in studies using a forward-masking paradigm (Boettcher et al. 1990; Shore 1995).

As to the mechanisms involved, adaptation phenomena are still not fully understood. On the one hand, for the auditory nerve, authors agree to consider adaptation to be the result of neural refractory properties and depletion of available transmitter at the hair cell–nerve fiber synapse (for review see Eggermont 1985; Javel 1996). Therefore, the multi-component adaptation may be attributed to a multi-stage transmitter depletion (Smith and Brachman 1982), with transmitter release depending to some extent on the nature of the stimulus. For example, there would be an increase of adaptation in AN fibers when the intensity of a tone stimulation increased (Peake et al. 1962b; Westerman and Smith 1984; Yates et al. 1985; Rhode and Smith 1985; Müller and Robertson 1991). On the other hand, in CN, adaptation was expected to be more complex, mainly because of (1) the variety of afferent inputs, (2) the intrinsic membrane characteristics of each cell type, and (3) inhibitory inputs. Nevertheless, at the single unit level, it was reported that adaptation patterns in firing rate of primary-like and chopper units when the intensity of a tone was increased were similar to those observed in single AN fibers (Boettcher et al. 1990; Shore 1995; Burkard and Palmer 1997). In contrast, there are no studies addressing this issue at the whole CN level based on near-field evoked potentials.

In a recent report (Loquet and Rouiller 2002), we demonstrated that the dynamic properties of adaptation to trains of repetitive clicks in VCN were comparable to those of the AN at a fixed intensity. Whether this similarity persists at various intensity levels needed further investigation. To address this question, VCN near-field evoked potentials were chronically recorded in an animal model (unanesthetized adult rats) in response to pulsatile acoustical stimuli of varying intensities (ranging from 5 to 70 dB SPL) and pulse rates (ranging from 100 to 1000 pulses per second, pps). The results are compared to adaptive properties previously established for the auditory nerve.

Materials and methods

Animals

Experiments were conducted on male adult Long-Evans rats (Janvier Laboratories, Le Genest-Saint-Isle, France) weighing approximately 300 g and aimed at recording auditory evoked near-field potentials from a chronic electrode implanted in the left VCN. The experimental procedure was approved by the Swiss veterinarian authorities and was performed in accordance with the *Principles of laboratory animal care* (US NIH Publication No. 86-23, revised 1985) and the 1964 Declaration of Helsinki for animal care. The procedure was described in detail in a recent report (Loquet and Rouiller 2002) and will only be summarized here. Briefly, before surgery, the animals (n=6) were treated with atropine sulfate (0.05 mg/kg s.c.) to minimize respiratory distress, and with a nonsteroidal anti-inflammatory drug (Carprofen, 4 mg/kg i.m.) to reduce inflammation and pain. Then, they were deeply anesthetized with pentobarbital (Vetanarcol, 40 mg/kg i.p.) and placed in a stereotaxic apparatus (Model 1404; Muromachi Instruments, Japan) in order to implant the chronic recording tungsten electrode (2–4 MΩ impedance) in the left VCN (coordinates: AP=−9.80 mm, ML=4.30 mm, DV=7.99±0.35 mm from bregma). A ground electrode was placed in the rostral cranium on the dura mater, and the two electrodes were soldered to a socket and fixed to the skull with dental cement. The animals were allowed to recover for 1 week before beginning chronic recording. The location of the recording electrode in the VCN was histologically verified in the brains of all rats at the end of the experiment (see Loquet and Rouiller 2002).

Acoustic stimulation

Testing was performed with Tucker-Davis Technologies System II (TDT, Alachua, FL, USA) equipment in a sealed sound proof booth (IAC, Niederkrüchten, Germany) on awake rats placed in a restraining device (Loquet and Rouiller 2002). Acoustic signals were synthesized digitally using TDT SigGen32 software and, after digital-to-analog conversion, fed into a programmable attenuator before delivery to a speaker positioned 10 cm away from the left pinna of the rat. The calibration of the system was carried out with a sound level meter (B&K, model 2231) by measuring the sound pressure level (SPL root mean square re 20 μPa) emitted by the speaker when it was driven by a pure tone signal at 9 V peak level. The sound field was calibrated by positioning the microphone (B&K, model 4155, prepolarized free-field 1/2 inch) at the point normally occupied by the center of the animal's head. Within the audiometric room, the ambient noise level did not exceed 69 dB SPL with regard to the overall spectra linear level, and 38 dB SPL with regard to frequencies above 1 kHz. Cochlear nucleus near-field potentials (CNP) were amplified (2×10^3), bandpass filtered between 30 Hz and 5 kHz and then fed into an analog-to-digital converter.

The TDT data acquisition software BioSig32 was used to automate CNP averaging over 50 presentations and for offline analysis.

Acoustic stimulation consisted of repetitive short condensation-rectangular-pulse clicks (100 μs) delivered in a train of 250 ms duration followed by a pause (silent period) of 250 ms before the next train. The intra-train pulse rates varied from 100 to 1000 pps, and five intensities were tested: 70, 50, 30, 10 and 5 dB SPL. The analysis window stretched over 250 ms, and the amplitude of the N_1 component of the CNP was measured as the voltage difference between the first negative (N_1) and the first positive (P_1) peak. At repetition rates greater than 400 pps, responses to individual clicks started to overlap so that the amplitude of a certain response was influenced by the preceding one. To circumvent this contamination, the 250-ms trains were presented in an order so as to always increment click number in the train by one. The average CNP obtained in response to a certain train (*n* clicks) was then subtracted from the CNP to the following train (*n*+1 clicks), the resulting curve exposing solely the *n*+1 click (see Loquet and Rouiller 2002). Thus, individual clicks could be studied free of contamination. Finally, N_1–P_1 amplitudes were normalized with respect to the highest CNP observed in the train (usually the CNP to the first click), and were plotted as a function of the position of the corresponding click in the train.

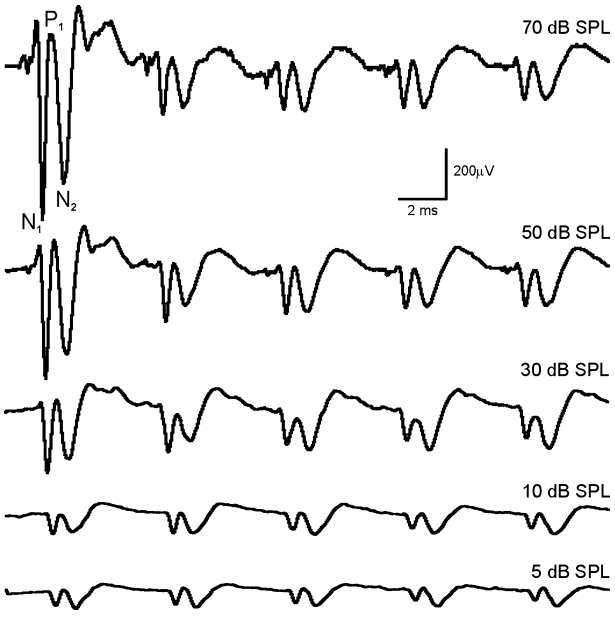

Fig. 1 Typical ventral cochlear nucleus near-field evoked potentials (CNP) elicited by 250-ms trains of repetitive clicks presented at a rate of 200 pps (Rat #6). Only the first 25 ms of the train are shown in order to easily identify the N_1, P_1 and N_2 deflections (negative polarity is downward). Note that the decrease of N_1–P_1 amplitude as a function of time became more obvious at high levels of stimulation. Stimulus intensity is given to the *upper right* of each trace

Results

As shown in Fig. 1, CNP amplitude measured as the voltage difference between the negative peak N_1 and the subsequent positive peak P_1 varied as a function of both intensity of stimulation and position of the stimulating click in the train. In Fig. 1, one of the most obvious effects of an augmentation in stimulus intensity is the increase of the first through-to-peak N_1–P_1 amplitude and the decrease of its latency. Then, it can also be noticed that increasing the intensity of stimulation induced an accentuation of amplitude differences between responses to the first click and the subsequent ones. In other words, these qualitative data show that adaptation was more pronounced when stimulus intensity increased.

Amplitudes of individual CNPs to consecutive clicks in the train were plotted for each rat; one representative animal is depicted in Fig. 2. In general, curves tend to display an exponential decrease of the normalized CNP amplitudes as a function of time (position of the corresponding stimulating click in the train). Most curves exhibit an initial rapid adaptation followed by a slower adaptation and a plateau, except for those obtained at low repetition rates (100 and 200 pps) and low intensities (5–10 dB SPL) where one phase was sometimes missing. In addition, for each rate tested (100–1000 pps), adaptation became more pronounced when intensities were increased from 5 to 70 dB SPL, as represented by a decrease of the plateau level and a shortening of the rapid and short-term adaptive components. This latter assumption was verified by determining the decay time constants of the two

Table 1 Time constants of cochlear nucleus auditory-evoked potentials as a function of stimulus rate and intensity. K_1, rapid, and K_2, short-term adaptation time constants were determined with non-linear regression fitting curves (see Results section). The few curves that were better described by a one-component time-constant equation are not included in this table. Mean values and standard deviations (SD) were calculated from data obtained from five rats

Stimulus rate	Value	Time constant at stimulus intensity									
		5 dB		10 dB		30 dB		50 dB		70 dB	
		K_1	K_2	K_1	K_2	K_1	K_2	K_1	K_2	K_1	K_2
100 pps	Mean	15.5[a]	46.6[a]	11.4	61.2	12.2	49.9	5.3	50.6	2.4	48.1
	SD			3.8	17.1	5.5	12.1	3.4	8.1	3.2	12.6
200 pps	Mean	6.5	74.4	9.7	64.0	4.6	54.8	2.8	41.4	2.4	44.0
	SD	1.5	25.7	4.3	37.1	1.4	16.0	1.2	21.1	1.6	34.1
400 pps	Mean	4.4	64.5	4.6	66.4	2.2	54.6	1.4	36.4	0.5	6.3
	SD	2.3	25.5	1.4	21.2	0.7	15.5	0.5	36.9	0.5	2.7
600 pps	Mean	3.8	52.2	3.1	45.9	1.2	30.2	0.8	46.4	0.2	6.8
	SD	1.2	15.1	1.6	25.4	0.6	23.4	0.2	49.4	0.2	0.9
800 pps	Mean	2.6	100.0	1.6	38.5	0.7	28.6	0.2	28.1	0.1	4.9
	SD	1.4	100.8	1.0	24.9	0.2	21.8	0.2	18.8	0.1	2.7
1000 pps	Mean	1.5	54.5	1.1	49.7	0.3	15.0	0.3	8.8	0.2	5.1
	SD	1.6	62.5	0.8	55.3	0.3	11.8	0.3	5.4	0.1	0.9

[a]Adaptation curve of only one animal fitted by a two-component time-constant equation

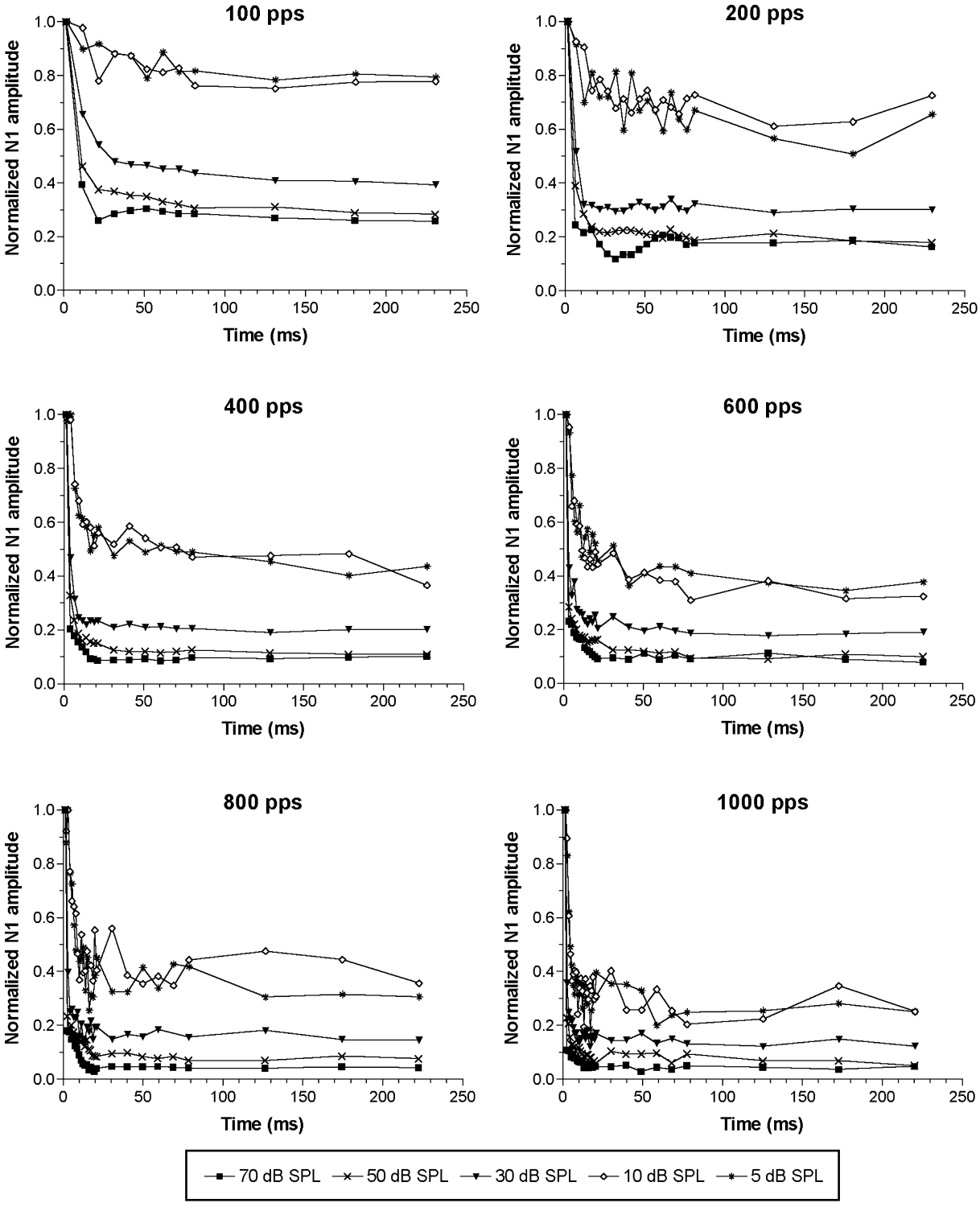

Fig. 2 Normalized amplitudes of ventral cochlear nucleus near-field evoked potentials in Rat #6 displayed as a function of the position of the stimulating clicks in the 250 ms train. Five intensities ranging from 70 to 5 dB SPL are presented for each of the click rates ranging from 100 to 1000 pps

adaptive components using exponential decrease equation fittings that we previously demonstrated to be consistent with adaptation properties of VCN near-field potentials (Loquet and Rouiller 2002). The equation used was:

$$NP(t) = NP_1 e^{-t/\kappa_1} + NP_2 e^{-t/\kappa_2} + Plateau$$

where NP_1, NP_2 are the y-intercepts of the rapid and short-term components respectively; *Plateau* is equal to the

N_1–P_1 amplitudes during the steady state response, and K_1, K_2 are the decay time constants of the two postulated adaptation components. In some instances, in particular at the lowest intensities of stimulation (5–10 dB SPL), data were best fitted with a one-component time constant adaptation equation (NP_2=0). The curve-fitting was run using the Levenberg-Marquardt method with GraphPad Prism v3.02 software and the deviation from the model was assessed by considering the correlation coefficient ($R^2 \geq 0.70$) and by testing the Gaussian distribution of the residuals around the curve ($P>0.1$). Mean values of K_1 and K_2 were calculated from data obtained in five animals; they are summarized in Table 1 and depicted in Fig. 3 (results of fittings using only one-component time constant have not been included in the data presented). The rapid time constants (K_1) exhibited a progressive decrease as intensity of the stimulus increased. A maximum reduction of 13.1 ms (from 15.5 to 2.4 ms) was obtained at 100 pps, and was less pronounced at higher pulse rate (1.3 ms at 1000 pps). The short-term time constants (K_2) appeared to be roughly independent of intensity over an intensity range of 45 dB (from 5 to 50 dB SPL). In contrast, at 70 dB SPL, a marked decrease of K_2 was observed at 400 pps and above. Large inter-individual variabilities were observed at rates of 800 and 1000 pps.

For each rat, the total extent of adaptation was estimated by the ratio of the plateau amplitude to the highest CNP amplitude in the train. The magnitude of the plateau was determined by averaging the CNP amplitude values obtained during the last 150 ms of the train. These data were then averaged across the six rats and plotted in Fig. 4. On the right of the figure, an adaptation indicator has been introduced to underline the fact that the smaller the ratio, the more pronounced the adaptation. The curves, progressively declining from left to right, thus indicate an increase in adaptation for higher levels of stimulation. Indeed, increasing the intensity from 5 to 70 dB SPL led to an augmentation of adaptation of about 50% at 100 pps, and 40% at 200 pps. Interestingly, one can notice that at all higher repetition rates (400 to 1000 pps) this augmentation of adaptation with intensity remained around 30%. This suggests a separation of the data into two groups: at high repetition rates (400, 600, 800, 1000 pps) the change of adaptation with intensity is comparable, whereas at low pulse rates (100, 200 pps), the progressive increase of adaptation with intensity is rate-dependent and, in addition to that, more marked than at high repetition rates.

Discussion

CNP waveforms obtained in the present study in response to click stimuli (Fig. 1) are similar to those elicited by tone bursts that were reported recently (Loquet and Rouiller 2002). The 2.11 ms latency of the N_1 response at 5 dB SPL is consistent with previously observed values for the CN ranging between 2.0 and 3.0 ms (Huang and Buchwald 1980; Møller 1983), as opposed to shorter latency values obtained from the AN ranging between 0.5 and 1.8 ms (Møller 1983). However, as to the exact origin of the N_1 deflection within the CN, latency data do not allow for further conclusions since a large overlap exists between anteroventral, posteroventral and dorsal CN latencies (Godfrey et al. 1975a, 1975b). Nevertheless, under our experimental conditions, the N_1 response most likely reflects synchronized discharges in the VCN for the following reasons. Firstly, the histological verification in the brainstems of all rats (data not shown) demonstrated that the tip of the chronic recording electrode was clearly located in the anteroventral part of the CN. Secondly, the contribution of units located in the dorsal CN (pauser and buildup units) is unlikely, considering their inhibitory response patterns and their low ability to follow repetitive clicks (Rhode and Smith 1986).

The exponential decrease of CNP amplitudes in response to repetitive stimuli shown in Fig. 2 is in

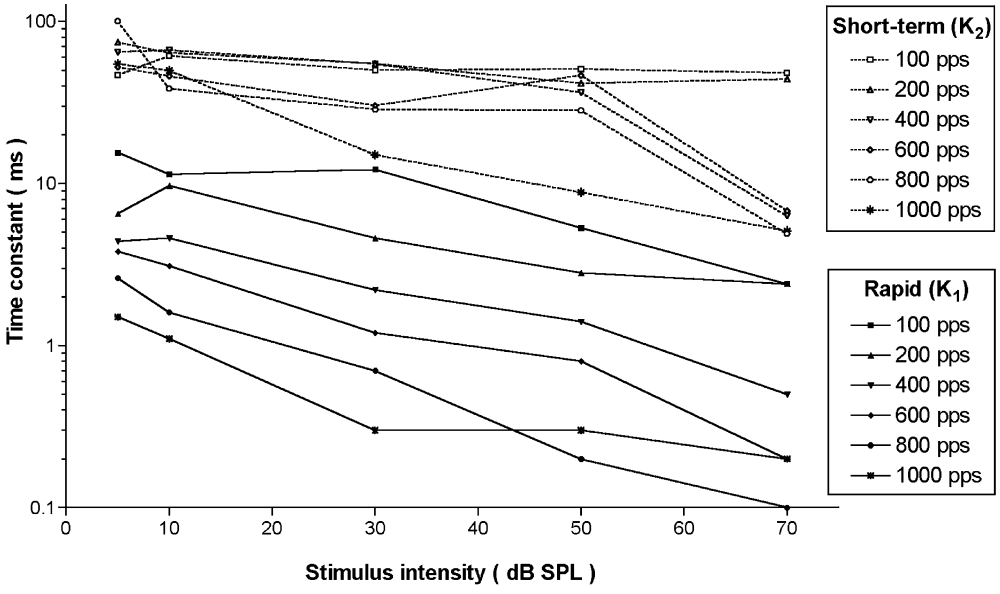

Fig. 3 Mean fitted decay time constants displayed as a function of stimulus rate and intensity. Time constants for rapid (K_1) and short-term (K_2) adaptation components were obtained from five animals by fitting normalized N_1–P_1 amplitude curves (see "Results" section). Standard deviations of data points are omitted for the sake of clarity but can be found in Table 1

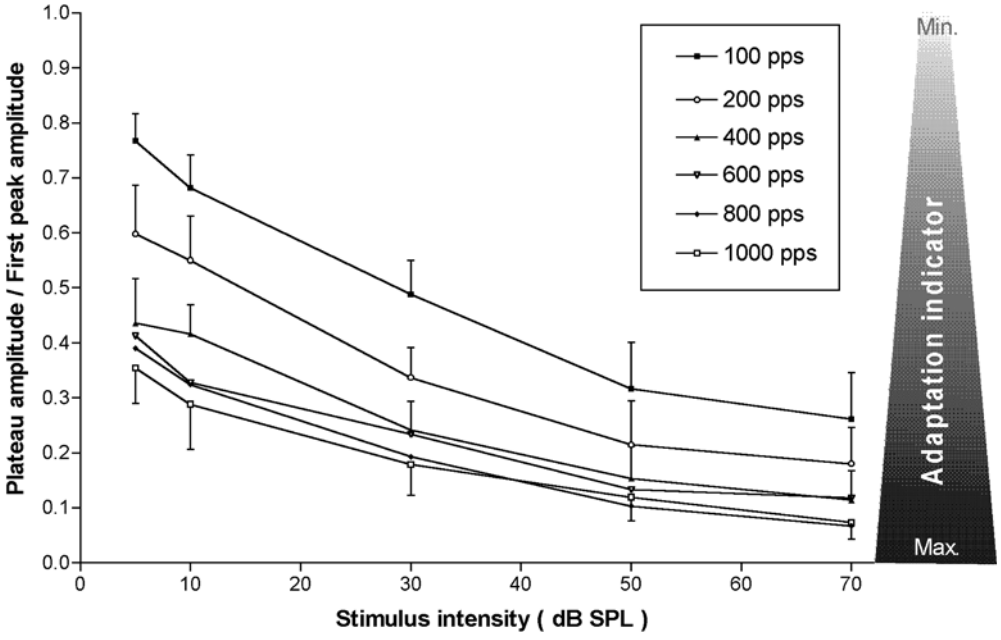

Fig. 4 Amount of adaptation expressed as the ratio of the plateau to the first peak amplitude, as a function of stimulus intensity. Mean values were derived from six animals, the *bars* representing 95% confidence intervals. At two stimulation rates (600 and 800 pps), the bars are omitted for the sake of clarity. An adaptation indicator is depicted on the *right* of the graph

agreement with the data of Huang (1981) and Loquet and Rouiller (2002). Such an adaptation generally follows a two-phase exponential decline (rapid [K_1] and short-term [K_2] adaptive components) to finally reach a plateau. This pattern varies as a function of stimulation rate with a faster (decreasing time constants) and larger (decreasing plateau level) decay as frequency increases. This repetition-rate effect on neural adaptation in the CN is in agreement with our previous report (Loquet and Rouiller 2002) and with data obtained in the AN (Peake et al. 1962a), for which the response amplitude decreased as soon as stimulus rate exceeded 10 pps. Unexpectedly, however, the present study demonstrates that the augmentation of the amount of adaptation as a function of increased stimulus intensity (Fig. 4) does not depend strongly on the repetition rate at 400 pps and above. The reason for such a segregation into two groups (high and low repetition rates) is not known but one may speculate that it is related to the refractory period. At 100, 200 and 300 pps, the unit is not affected by refractory mechanisms, in contrast to high rates (400 pps and above) for which the period between two consecutive pulses falls within the relative refractory period.

Concurrently with the rate effect, the present data indicate that adaptation in VCN depends on the level of stimulation. Indeed, increasing the stimulus intensity from 5 to 70 dB SPL led to a more prominent exponential decrease of CNP amplitudes, irrespective of the rate tested (Fig. 2). As mentioned in earlier studies (Westerman and Smith 1984; Yates et al. 1985), this effect is mainly attributable to the rapid adaptation time constant (K_1), which shows a constant decrease with increasing intensity (Table 1 and Fig. 3). In contrast, the short-term adaptation time constant (K_2) shows little intensity dependence (except for the interval between 50 and 70 dB SPL), an observation in line with data on AN fibers (Smith and Zwislocki 1975; Westerman and Smith 1984). The effects of intensity on VCN adaptation is also well characterized by the level of the plateau (steady-state component), whose decrease was at its most significant between 10 and 30 dB SPL (Figs. 2 and 4). At high stimulus intensities (50–70 dB SPL), average amounts of adaptation were similar to those found in high-spontaneous-rate nerve fibers (Brown 2001). Therefore, in our experimental conditions (awake rats), VCN near-field evoked responses appear to be essentially the same as those recorded in the AN, as far as adaptation is concerned.

Physiological mechanisms

In line with the present study, authors presenting simple short tone bursts, either as described by Blackburn and Sachs (1989) or in the context of a forward-masking paradigm (Boettcher et al. 1990), observed a more pronounced adaptation with increasing intensity in some CN neurons, namely the bushy cells (which produce primary-like and primary-like-with-notch responses) and stellate cells (usually associated with chopper responses), than in cell types generating other response patterns (pauser, buildup, onset). More recently, response decrement differences were found within VCN (Shore 1995), in particular between primary-like, primary-like-with-notch, sustained chopper, transient chopper, low-intensity chopper, onset and on-chopper responses. The author suggested that both adaptation and inhibition were involved in producing these differences. In the present study, it is likely that adaptation recorded in the VCN by using near-field evoked potentials (summation of individual neurons) mainly reflects the adaptive properties of primary-like units, which have themselves properties close to those of AN fibers, in particular comparable adaptive time constants. As far as chopper units are concerned, their contribution seems to be substantially less than that of primary-like units according to Shore (1995) and Burkard

and Palmer (1997). Moreover, it can be noticed that AN and CN adaptations are likely to mirror still more peripheral changes, primarily located at the hair cell-nerve fiber synapse (Norris et al. 1977; Furukawa et al. 1978). At this stage, adaptation was suggested to involve depletion of neurotransmitter in a cascade of reservoirs (Eggermont 1985).

Overall, despite the fact that adaptation is complex and may depend on specific properties of each relay along the auditory pathway, the present study supports the idea of a reconciliation between single unit data and near-field evoked potentials. Indeed, in the same way that adaptation of VCN primary-like units is essentially the same as that of AN fibers, CNP adaptation obtained in VCN was found to be comparable to CAP adaptation obtained in AN. Therefore, it seems reasonable to hypothesize that our CNP data mainly reflect properties of primary-like units in VCN. As a consequence, the present animal model can be used to better understand auditory neural adaptation phenomena and to transpose these features to cochlear implants that aim at restoring, as accurately as possible, normal physiological hearing in profoundly deaf patients. Such studies could lead to direct clinical applications. They could, for example, be used to improve current stimulation paradigms in order to achieve better speech recognition in ambient noise by cochlear implant patients.

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