After hepatectomy, the hepatic parenchyma of the intact lobes readily grows to restore the original hepatic mass and this constitutes an adequate stimulus for the sprouting of monoaminergic nerves [7, 8].

Results obtained in this study would seem to indicate that nervous sprouting of vagal afferent fibers does not occur in regenerated rat liver during the experimental period investigated (until 3 weeks after hepatectomy). The decreased number of intensely labeled neurons in the left nodose ganglion of hepatectomized rats is probably due to a decreased availability of the neural tracer at the terminal endings due to the increased hepatic mass. The finding that the number of intensely labeled neurons in the right nodose ganglia is not significantly different from that of controls could be due to the fact that their afferent terminal fibers innervate some hepatic areas which are not involved in the regenerative process.

The behavior of afferent fibers in the regenerated liver seems to be quite different from that of the sympathetic efferent ones. Experiments regarding regeneration of monoaminergic nerves in the rat liver after partial hepatectomy [7, 8] have shown a characteristic hyperinnervation in the central area of the hepatic lobe at 7 days after surgery, whereas the innervation in the peripheral area was about 50% compared to controls. Two weeks after partial hepatectomy the hyperinnervation was still present in the central area of the lobe and there was a 70–85% innervation of the peripheral area. Liver innervation was complete by the sixth week.

Afferent hepatic fibers seem to behave differently. In fact, if hyperinnervation had occurred, the experimental results would have been completely different from those obtained; an increased, not a decreased number of intensely labeled neurons would have been found in the vagal nodose ganglia three weeks after regeneration.

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Location of an acoustic window in dolphins

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Summary. Auditory brainstem responses (ABR) to sound clicks from sources in different positions were recorded in dolphins *Inia geoffrensis*. The position of the acoustic window was determined by measurement of acoustic delays. The acoustic window was found to lie close to the auditory meatus and the bulla rather than on the lower jaw.
Key words. Auditory brainstem response (ABR); dolphins; hearing; acoustic window.

The mechanisms of sound transmission to the ear in aquatic mammals, particularly in dolphins, remain obscure. It has been suggested that sound reaches the inner ear directly via head tissues, or sound transmission involves the middle ear and the closed auditory meatus [2–4]. This assumption was supported by calculations indicating that the closed auditory meatus can serve as an effective acoustic transformer for the sounds to be transmitted to the middle ear [5].

On the other hand, there is the popular so-called mandibular hypothesis emphasizing the key role of the lower jaw with its peculiar fat body as a specific sound-conducting pathway [6, 7].

Sounds are believed to reach the fat body through a thin bone plate on the lateral mandibular surface and are transmitted further via the fat body to the bulla. Therefore, the hypothesis postulates the presence of an acoustic window on the lower jaw which allows for the percep-

tion of sounds. The validity of the mandibular hypothesis is supported by data on lower thresholds of both mircophonics and evoked potentials to the contact acoustic stimulation of the mandible as compared with those observed after a similar stimulation of other body sites[8-10]. In other experiments, it was shown that a sound-proof hood attached to the lower jaw impaired the dolphin's ability to recognize echolocating signals[11].

These data can, however, be interpreted in several ways. The contact acoustic stimulation in the air is not adequate for dolphins, and its effect depends to a large extent on the properties of the tissue with which a hydrophone is in contact. The sound-proof hood may affect the acoustic field near the head and influence auditory perception even if the acoustic window is not located on the lower jaw. Therefore, the acoustic window problem remains to be elucidated.

In an attempt to obtain additional data, we measured acoustic delays of evoked auditory potentials at different positions of sound sources. The principle of the method is illustrated in figure 1. Two sources of sound signals, S1 and S2, are placed in known positions; the position of receiver O is to be determined. The latency of the response of the receiver to signals S1 and S2 can be measured. This latency is the sum of the acoustic delay (the time of sound transmission from the source to the receiver) and the response-time proper. We do not know the exact ratio of the acoustic delay to the response time. Let us therefore assume that the latter is a constant (for equal sound intensity) and the total response latency is a function of the acoustic delay. It is thus possible to determine the difference dT (1,2) between acoustic delays for sources S1 and S2 from measurement of the two response latencies. The velocity of sound transmission (C) being known, the difference between acoustic delays is a measure of the difference between the distances: d(1,2) = C*dT(1,2). Receiver O is therefore located on an MM line defined by the equation: 1(2) − 1(1) = (1,2). The use of one more sound source (S3) allows us to draw another line (NN) from the difference d(2,3), which is also the site of the location of the receiver. The intersectionpoint of the MM and NN lines gives the position O of the receiver, or the acoustic window through which the sound is specifically transmitted to the receiver.

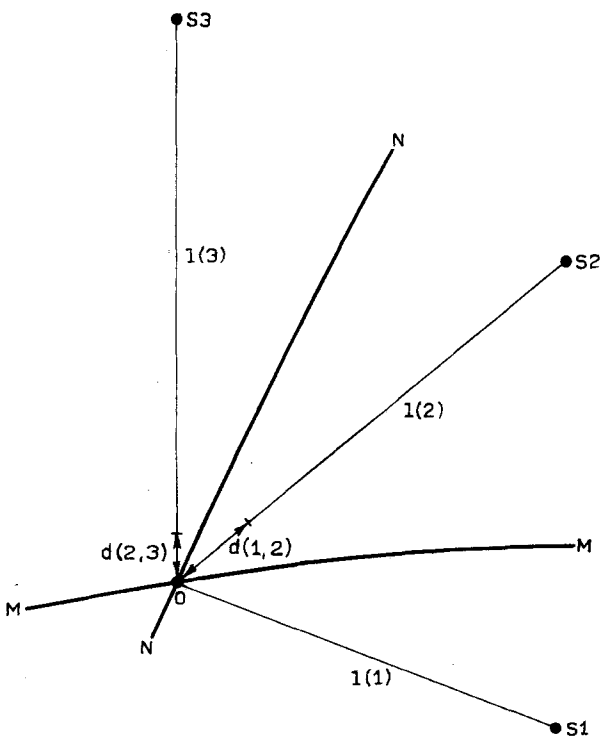

Figure 1. Calculation of the position of the receiver from the difference between acoustic delays. S1, S2, S3 – sound sources, O – receiver, d(1,2) – difference between distances 1(1) and 1(2) from S1 and S2 to O, d(2,3) – difference between distances from S2 and S3 to O.

To employ this technique for the location of the acoustic window, the difference between distances must be measured with an accuracy within 3–5 cm which corresponds to a 20–30 μs difference in acoustic delays. This level of accuracy can be ensured by recording short-latency evoked potentials, e.g. auditory brainstem response (ABR), from either the skull or the body surface of dolphins[12,13]. This ABR contains very short waves (about 0.5 ms). The peak latency of these waves can be measured with an accuracy within 20 μs.

The experiments were performed on two adult Amazon river dolphins *Inia geoffrensis* (a male and a female) weighing 55 and 60 kg. A dead animal of similar size (60 kg) was used to verify the location of the acoustic window relative to the skull.

During the experiments the dolphins were placed in a round pool 6.5 m in diameter. The water column was only 40 cm high, to minimize the echo from both the pool bottom and the water surface; that is, sound transmission occurred as if in a plane layer. Each animal was supported by a stretcher while the greater part of its body was covered with water but the back, the dorsal part of the head, and the blowhole remained above the water surface. The stretcher was made of a sound-transparent material (fine net). The head of the dolphin was positioned in the center of the pool.

Sound clicks emitted through a spherical hydrophone were used as acoustic stimuli. The spectral bandwidth of the click was 10–120 kHz at the −20 dB level, and the maximum spectral density was at 30 kHz. Position of the sound source (hydrophone) was determined relative to a reference point which coincided with the rostral tip of the melon (point O in fig. 2B). The hydrophone was situated at a distance of 1 m from the reference point, at an angle of 0 to +/−165° relative to the longitudinal axis.

ABRs induced by sound clicks were recorded from the head surface using needle electrodes inserted into the skin to the depth of 2–3 mm. This procedure required neither immobilization nor anesthesia of the dolphins. The active electrode was placed on the dorsal head surface 6 to 8 cm caudally from the blowhole and the reference one

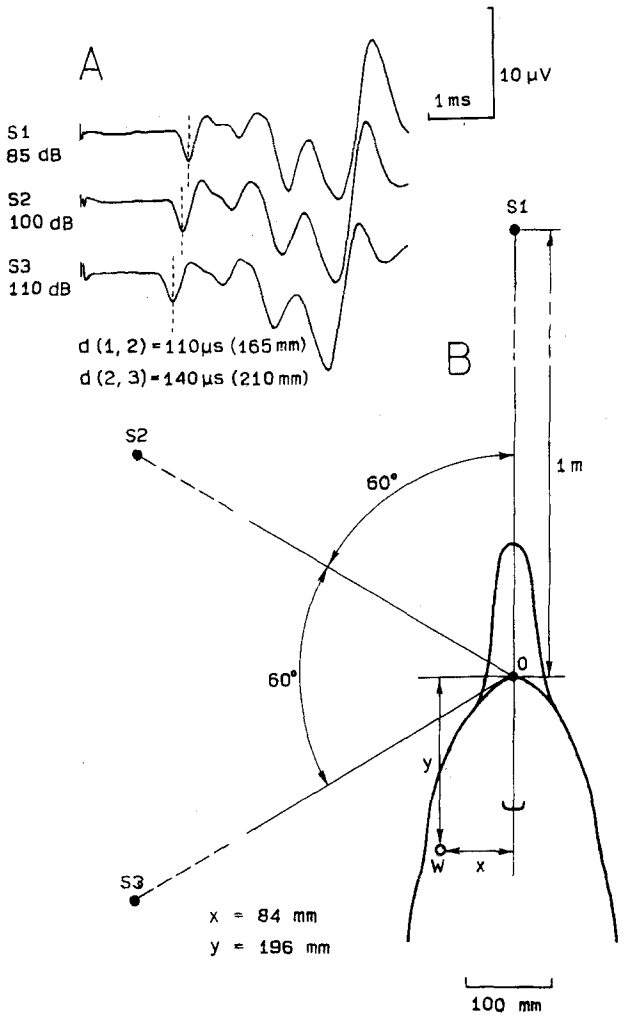

Figure 2. Locating of the acoustic window from the difference between acoustic delays. *A* ABRs elicited by stimulation from sources S1, S2, and S3. Vertical dotted lines are temporal cursors indicating the peak of the first wave; d(1,2) – the difference between latencies of responses to stimuli S1 and S2; d(2,3) – that to stimuli S2 and S3. *B* The positions of the sound source relative to the dolphin's head (dorsal view); O – reference point (the melon tip).

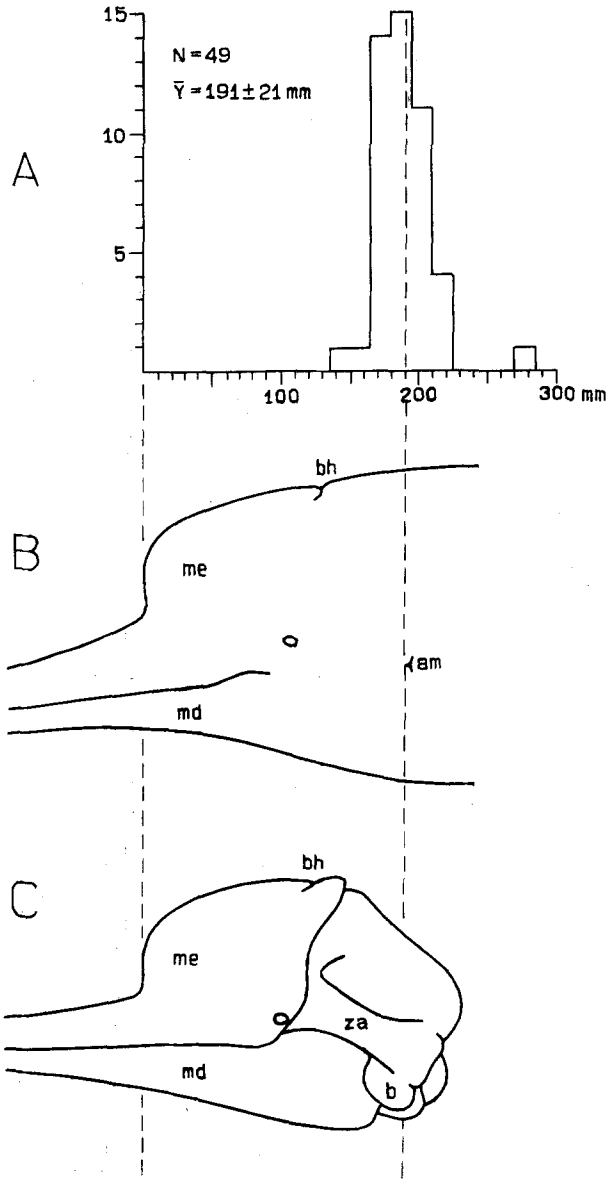

Figure 3. *A* Distribution of the observed distances between the melon tip and the acoustic window. *B* and *C* show the side-views of the intact and of the dissected dolphin's head; the scale is the same as on the Y axis of histogram A. Vertical interrupted lines indicate the reference point (the tip of the melon) and the mean distance to the acoustic window. b, bulla; za, zygomatic arc; md, mandible.

was on the back. Both electrodes were above the surface of the water. The potentials were amplified in a frequency range of 50–5000 Hz and then averaged using a routine technique. The peak latency of ABR was measured using an averaging processor at either 10 or 20 µs discretion. ABRs elicited by sound clicks are shown in figure 2A and the position of the sound sources in figure 2B. It is evident that responses induced by the stimuli from the S1, S2, and S3 sources had somewhat different latencies due to different acoustic delays. The accurate measurement of these delays was possible, based on the peak latency of the first positive wave (indicated by the cursors). The differences d(1,2) between the response latencies to the S1-S2 stimuli and d(2,3) between the latencies to S2-S3 stimuli are shown in figure 2.

When recording ABRs, we took into account that hearing sensitivity of dolphins depends on the position of a sound source. The highest ABRs were obtained when the source was situated in front of the animal's head, and they diminished when the source was moved laterally and further caudally [14]. That is, at an equal physical intensity of the stimulus the sound source in front of the animal was the loudest. On the other hand, a change of loudness evidently affected both the amplitude and the latency of ABRs [12]. It was important for our purpose that the ABR-proper response time remain unaltered. Therefore, the click intensity at each position of the sound source was chosen to elicit ABRs of equal amplitude. Figure 2 shows click intensity in dB relative to a peak sound pressure of 1 mPa.

The difference between acoustic delays observed was used to calculate the location of the acoustic window. In an example shown in figure 2, with the positions of sources S1, S2, and S3 differing by 60°, point W with coordinates x = 84 mm and y = 196 mm (relative to the reference point O) is defined by the requirement that d(1,2) = 110 μs (165 mm) and d(2,3) = 140 μs (210 mm). A total of 47 measurements was available for the calculation of the acoustic window coordinates. Of special interest is the longitudinal Y coordinate along the body axis because it is this parameter that is likely to indicate whether the acoustic window is located on the lower jaw or near the bulla and the auditory meatus. Taken together, these data are presented in figure 3 A showing the distribution of the observed distances between the reference point (melon tip) and the acoustic window. The mean distance and the standard deviation were 191 +/ −21 mm, with more than 95% of the measurements (45 out of the 47 available) exceeding 165 mm.

To compare these results with the basic head and cranial measurements, figure 3 B presents the side-view of the experimental dolphin's head and figure 3 C shows the side-view of the partially dissected head of the dead animal with the intact melon and exposed lower jaw, basal and occipital parts of the skull. A site 19 cm from the melon tip can be seen lying near the auditory meatus and the bulla, and outside the lower jaw.

Thus the data presented herein suggest that the acoustic window is located close to the auditory meatus and the bulla rather than on the lower jaw.

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Response of the starling circadian system to transitions between light and darkness

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Summary. The effects of exposure to sudden transitions from dark to light (D/L) and light to dark (L/D) were determined on the free running circadian feeding rhythm of European starlings (*Sturnus vulgaris*). D/L transitions (step-up) produced phase advances throughout the circadian cycle. In contrast L/D transitions (step-down) produced both advances and delays. The L/D transition phase-response curve has a contour and shape similar to the phase-response curves previously obtained in birds with light pulses.
Key words. Light-dark transitions; light pulse; phase-response curves; *Sturnus vulgaris*.

Recent views about how light/dark cycles synchronize circadian rhythm have originated primarily from studies of how brief pulses of light affect the timing of circadian rhythms [2]. Light pulses provided in 24-h-cycles reset the phase of the circadian clock (either by advancing or delaying) so that the rhythms under their control express a 24-h periodicity. Effects of light pulses and their phase-dependent phase shifts have been documented in detail in many species [3]. In contrast the effects of other forms of lighting information such as 'step-up' and 'step-down' (D/L and L/D transitions, respectively) have received a much lesser attention. Just as pulses of light and/or darkness do, D/L and L/D transitions also produce shifts in the phase of circadian rhythms depending upon the time of presentation within the cycle [4]. Such phase-dependent phase shifts, produced by simple transitions like D/L and L/D, have been recognized in organisms ranging from plants to mammals [5]. Despite these observations few data are available that systematically analyse the phase shifts produced by these light-on and light-off steps. The purpose of the present study is to investigate how these discrete D/L and L/D transitions phase shift circadian