Annotation

Hearing defects in preterm infants

Neurosensory hearing defects occur with a frequency of about 0.5% in children both in Europe and in North America. In those born preterm, however, hearing deficits are much more frequent. Three possible aetiological factors have hitherto been incriminated and have therefore been particularly investigated. (1) Preterm termination of pregnancy *per se*. (2) Perinatal risk factors such as hypoxia, hyperbilirubinaemia, drugs, infections, etc.¹ (3) Noise exposure in incubators.^{2,3}

For a prospective study in the neonatal period, auditory-evoked responses, auditory-evoked heart rate changes, and auditory-conditioned orientating reflexes can be regarded as hearing tests. All methods available to test neonatal hearing heavily rely on intact sensory pathways, both specific and non-specific, within the central nervous system. This is a great advantage for research into the development of sensory brain mechanisms where these methods have become a most informative tool to study the influence of nonspecific environmental factors such as oxygen, temperature, nutrition, and handling on brain development, both in animals and in human infants. For the determination of cochlear damage—for instance due to noise pollution—these methods have disadvantages, since normal cochlear function may be masked by a severely abnormal brain. However, neurosensory hearing loss can certainly be detected by these methods even in the neonatal period. Moreover, after the pioneering work of Jewett and his associates⁴ it seems as if brain stem auditory-evoked potentials occurring between 2 and 12 ms after the click are an extremely promising new tool to detect hearing deficits even in small infants.⁵

We studied the effect of preterm delivery and a 3-week period of incubator care on the development of auditory-evoked responses (AER) by comparing infants of the same conceptional age but with different extrauterine life spans. The maturation of the AER was nearly identical inside vs outside the uterus in otherwise normal, low-risk newborn infants.⁶

For a retrospective study in older children, the various audiometric methods, i.e. pure tone and speech audiograms (Fowler, Sisi and Langenbeck

test) can be applied with certain limitations for deciding whether the damage is cochlear or retro-cochlear. Although for these tests a reasonable degree of intellectual integrity is necessary, general brain dysfunction has the least effect with pure tone audiograms, making this method particularly useful in high-risk preterm infants who may well have a few behavioural abnormalities. Except for severely handicapped children, by 8-10 years repeatable thresholds can be recorded.

We applied audiometric tests to infants that had been born preterm, both high and low-risk, and found 12.4% of cases with some neurosensory hearing loss.⁷ No relationship of hearing loss with gestational age, hyperbilirubinaemia, and drugs could be discerned. Nor was there any correlation with the length of stay in an incubator, as one parameter of noise exposure. There was, however, a strong correlation between hearing loss and the 'perinatal nonoptimal score', i.e. the sum of perinatal risk factors.

Thus, both approaches, neonatal AERs and audiometric follow-up, failed to corroborate the assumption that the duration of incubator noise exposure is responsible for substantial hearing loss in preterm infants. Slight degrees of cochlear damage may have remained undetected⁸ and our data do not exclude the possibility that in high-risk preterm infants incubator noise pollution might play an additional role in producing hearing defects. While it is not our intention to stop engineers from constructing less noisy incubators, the paediatrician, using the incubator as it is, can influence only one parameter of incubator noise pollution—its duration—whereas the two other important parameters, i.e. intensity and frequency of the sound, cannot at present be altered. In so far as hearing loss was related to periods of incubator care, however, we could find no support for the anxieties that have been expressed.

References

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Erratum

In Fig. 2, p. 114 (February issue) in 'Management of nephroblastoma in childhood', the dosage regimen of actinomycin D 15 µg/kg IV should read:

Then at 12 weeks
Total 8 courses.

The dosage regimen of vincristine 1·5 mg/m² IV should read:

Then at 2 weeks to
24 months from diagnosis.