one would expect the SIDS incidence to increase in Austria shortly after its introduction in 1971. Yet in our country a marked increase cannot be discerned until after 1977, whereas in England and Wales the rise of SIDS occurred much earlier (1971).

Secondly, it can be observed in Austria, as in England and Wales, that the decline of SIDS started several years before the introduction of media campaigns against prone sleeping. The peak year for SIDS was 1988 in both countries, whereas the first nationwide media campaigns against prone sleeping were not launched until October 1991 in England and Wales,⁴ and in April 1993 in Austria.

Thirdly, the rise and fall of SIDS does not seem to have affected the continuity of the decline of postneonatal mortality in our country (see figure). Postneonatal mortality was neither increased by the occurrence and the increase of SIDS, nor was its further steady decline accelerated by the decreasing SIDS rate.

Our claim is that the dynamics of cot death need to be explained by diagnostic transfer between the SIDS and non-SIDS components of postneonatal mortality. Diagnostic transfer should be taken into account before interpreting trends of SIDS as a result of public health interventions.

FRANZ PAKY
Department of Paediatrics,
Landeskrankenhaus Moedling
Sr M Restituta-Gasse 12,
2340 Moedling, Austria
JOSEF KYTIR

Institute for Demography,
Austrian Academy of Sciences,
Hintere Zollamtsstrasse 2B,
1033 Vienna, Austria

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Uses and abuses of pulse oximetry

EDITOR,—The recent review article by Moyle correctly outlined sources of inaccuracy in the use of the pulse oximeter,¹ however an important omission was the behaviour of pulse oximetry in conditions of low arterial pressure. Significant inaccuracies² can occur at mean pressures less than 30 mm Hg and pulse pressures of only 20 mm Hg. The 'dropout' (the inability to produce a signal at low arterial pressures) at finger placement sites limits the usefulness of these devices under conditions of reduced cardiac output, hypotension, and peripheral vasoconstriction.

Moyle in his review also mentions that central placement of oximeter probes (tongue and cheek placement) would confer an advantage in terms of response times to changes in saturation of haemoglobin. It

could be inferred from this review that oximeter probes located around the head should be the preferred site of probe placement. This placement could lead to further erroneous readings.

It has been shown that the overall performance of ear, nose, and forehead probes for various devices may be worse than for finger probes when hypoxia is induced at low radial arterial pressures.³ The ability to detect a weak signal when the probes of some devices are located on the head can give erroneous values for saturated oxygen. Unless the clinician knows precisely the specific behaviour of his particular probe at low arterial pressures it can be recommended that head placement of oximeter probes should be used only if the finger site is unavailable. Signal dropout, as well as an indicator that there is a problem with the peripheral perfusion, is then also a cue for an urgent alternative measure of saturation by blood gas analysis.

DONALD G KRUCHEK
Department of Anaesthesia
The North Hampshire Hospital,
Aldermaston Road,
Basingstoke RG24 9NA

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