Paradoxical pain

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Relation of birth variables to death from cardiovascular disease

EDITOR—D J P Barker and colleagues' study puts a further nail in the coffin of those who doubt that the intrauterine environment influences later health—in this instance, death from cardiovascular disease. A theme running through the Scottish and English perinatal mortality studies on this topic is that maternal nutrition is primarily responsible for reduced prenatal growth. Though there can be no doubting the importance of maternal malnutrition as a cause of reduced fetal growth in poor countries and as an influence on birth weight in the developed world, an influence of maternal factors on the offspring in the early part of this century, where Barker and colleagues' cohorts were born and brought up, there is no strong evidence of undernutrition now being responsible for restraining intrauterine growth in developed countries.

Maternal diet is only one of the many factors that can lead to fetal growth retardation. To begin to understand mechanisms that might link the environment of fetal life and infancy with later death from cardiovascular disease in adult life, BMJ 1993;306:422-6. (13 February.)

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Paradoxical pain

EDITOR—David Bowsher defines paradoxical pain as pain in one area, which is not worse when morphine or other opioids when there is peripheral or central neural sensitisation. Sensitisation occurs in damaged tissue and the surrounding area and in areas subserved by either an injured peripheral nerve or an intact central nervous system. Pain associated with inflammation is a typical example of peripheral sensitisation, hence the need to use a non-steroidal anti-inflammatory drug in most patients with painful soft tissue injury.1,2 Paradoxical pain is sometimes inadequately, but there is nothing paradoxical about this. Central sensitisation may also occur in such cases as part of a secondary "wind up" phenomenon in the dorsal horn. Occasionally this is due to a specific contribution—for example, a high concentration of an N-methyl-D-aspartate receptor blocker such as ketamine.3 Central sensitisation in neuropathic pain is more complex and, as Bowsher points out, demands a range of alternative measures.4,5

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1 Bowsher D. Paradoxical pain. BMJ 1993;306:473-4. (20 February.)


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Editor—David Bowsher’s editorial oversimplifies a complex and contentious issue. Paradoxical pain may well exist but is neither well documented nor common; it does not account for the majority of cases of uncontrolled pain, and we are not aware of any evidence that it was an important factor in the care of the patient in the recent highly publicised court case.6

Furthermore, large interindividual variations exist not only in the metabolism of morphine but also in the distribution of opioid receptors.7 This animal data on this subject cannot, and should not, be extrapolated to humans and many questions remain.

Though recognition of this potential therapeutic problem is welcome, until the clinical importance of the morphine metabolites in humans is completely understood these rare cases of paradoxical pain will remain unexplained.

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Editor—The concept of paradoxical pain and its relation to morphine metabolites raises many questions.1 There are several conceptual errors inherent in this discussion. One of the most fundamental is that the pain syndromes as described should have at any time actually respond to opioids. This makes the assumption that so-called paradoxical pain is nociceptive pain, with the second assumption that all nociceptive pain...