into individual cases. One approach would be to bring techniques from qualitative research[6] to bear on the process of implementation, collation, and dissemination of results. Secondly, it should broaden its perspective. We need to know about all homicides committed by the mentally abnormal and not just a selected subgroup. The government white paper, The Health of the Nation, sets targets for suicide prevention in those with severe mental illness[7]; this is not synonymous with contact with the psychiatric services. Jayne Zito's remarkable response to her husband's murder by Christopher Clunis, who suffered from schizophrenia, should inspire us to make much more effort than we do to obtain systematic information and opinions from relatives—of victims of homicide, of mentally abnormal offenders, and of suicides. Thirdly, the inquiry needs to adopt more rigorous investigative methods. Risk factors for violent death can be identified only by more rigorous use of techniques such as case control studies and case ascertainment and data collection can be comprehensive and unbiased only if they are not left to the discretion of clinicians.

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Neonatal prevention of iron deficiency

Placental transfusion is a cheap and physiological solution

Iron deficiency anaemia in childhood is common even in socially advantaged populations. Low birth weight, early consumption of cows' milk, fast growth rate, and poor dietary iron intake are considered the main risk factors.[1] Iron enriched infant formula and cereals have been shown to be effective preventive measures.[2,3] In developing countries, where iron deficiency anaemia is common and iron enriched formula and cereals are often not available, preventing iron deficiency is not easy: infants who enjoy prolonged and exclusive breast feeding have been found to have good iron status,[4] but such breastfeeding is increasingly rare; and although medicinal iron is cheap, its use may be culturally unacceptable or difficult to implement. Moreover, dietary iron supplementation can be dangerous in settings where malaria and diarrhoeal infection are endemic[2,5] and for children whose iron stores are adequate.[6]

Iron stores at birth show huge individual variations, which correlate with iron stores in the same individuals at 6, 9, and 12 months of age.[7] This may explain why the iron status of some infants remains sufficient, even if they do not receive adequate daily iron. Dietary iron seems to represent only one of the factors that influence iron status in the first year of life,[8,9] probably because iron absorption from formula and cereals is modest and is inhibited by many components of the diet such as polyphenols in fruit and vegetables. If high neonatal iron stores are associated with a good iron status in late infancy,[7] how can we safely increase neonatal stores?

The merits of early or late clamping of the umbilical cord have been controversial for many years.[10] According to Usher *et al*,[11] the estimated volume of placental transfusion varies from 20% to 60% of the existing blood volume (54-160 ml) depending on the time of clamping and the position in which the infant is held before clamping.[12] Linderkamp and colleagues estimated that the amount of placental transfusion is about 35 ml/kg of birth weight when term infants are kept at the level of the vaginal opening and the cord is clamped three minutes after birth.[13] The same authors have recently investigated the effect of placing the neonate on the mother's abdomen and clamping the cord only once it stops pulsating (Leboyer delivery).[14] They found that these babies had blood volumes 32% higher than babies whose cords were clamped immediately after birth. The packed cell volume in cord blood was not affected by placental transfusion, but after 2-4 hours it rose in the group of infants whose cord was clamped late, from 0·51 (SD 0·05) to 0·62 (0·06). This difference was statistically significant when compared with infants whose cords were clamped early.

A moderate placental transfusion as achieved in the Leboyer delivery does not significantly increase neonatal jaundice, nor does it incur detrimental haemodynamic changes,[14] although occasional cases of circulatory overload from excessive placental transfusion have been reported.[10] Moreover, a moderate transfusion of about 20-30 ml/kg endows about 30-50 mg of "extra" iron and can help prevent or delay depletion of iron stores during late infancy.

Recent research from Denmark favours this hypothesis.[7] Studying 9 month old infants born in a hospital whose policy was to clamp the cord late, the researchers found serum ferritin values higher than those reported for infants from other European countries,[15-17] whose cords were assumed to have been clamped immediately after birth. Higher neonatal iron stores associated with late cord clamping could be one explanation for this observation. The positive effects of delayed cord clamping could be even more clinically and economically important among infants in developing countries. For those children a moderate placental transfusion could represent a physiological and inexpensive means of increasing iron stores. At the same time, delayed cord clamping represents a change in routine practice that favours early contact between a mother and her newborn baby. An overview of randomised controlled trials found a statistically significant association between such early contact and subsequent prolonged breast feeding[18]; this could therefore represent another measure to prevent iron deficiency.

Immediate cord clamping is currently routine practice, but its widespread acceptance was not preceded by studies evaluating the effects of depriving neonates of a significant volume of blood. A large clinical trial to compare the short and long term effects of placental transfusion is needed.

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The melanoma epidemic: reality and artefact

Warrants a reappraisal of the relation between histology and clinical behaviour

Skin watchers have their own equivalent of the Heisenberg uncertainty principle. Just as you can't know exactly both the momentum and the position of a single electron, once you excise a pigmented lesion and know its histology you forfeit the chance of knowing what would have happened if you had left it in situ; progression, metastasis, or even involution are all possible. Removal of pigmented lesions is now one of the commonest surgical procedures and has radically changed the pattern of referrals to dermatologists. Epidemiological studies show a dramatic increase in histologically confirmed melanomas[1] and raise the important questions of whether the increase is real or a diagnostic artefact and whether we should reconsider the relation between sun exposure and melanoma.

The incidence of melanoma has increased by 3-7% per year from the mid-1950s to the early 1980s.[1-3] These changes have been seen in both sexes and in a large number of different caucasian communities in both the northern and southern hemispheres.[1-5] A smaller rise has also been seen in mortality, with a reduction in case fatality from an estimated level of over 85% in 1925[3] to under 20% today. Cohort analyses show, perhaps surprisingly, that mortality from melanoma rose from the 1890s to the 1950s and then started to decline, with forecasts that the overall mortality from malignant melanoma will peak early in the next century.[1-3 5] These cohort effects are not easily explained by changes in leisure activity. However, more recently (from the early 1980s onwards) several studies have shown steep rises in the incidence of melanoma, of 15-43% per year; in parts of New South Wales a doubling of histologically confirmed melanomas has been reported over a two year period.[1 4 6]

Armstrong suggests that these recent steep increases cannot be solely attributed to earlier diagnosis, changes in histopathological criteria for melanoma, or an increase in the proportion of excised lesions being referred for histological opinion.[1 4 6 7] In the case of New South Wales, he argues that since the increase in detection and removal of thin lesions has not been followed by a reduction in incidence of thicker lesions (which one might expect if the increase was due to earlier case ascertainment), this apparent epidemic of melanoma represents in part an increasing recognition of a pre-existing non-metastasising but invasive form of melanoma. The presence of such a lesion should not be surprising. In many, if not most, cancer systems the majority of dysplastic lesions do not progress to clinically relevant malignancy. Perhaps the clearest example is squamous cell cancer of the skin, where careful epidemiological studies have shown that, despite the presence of multiple genetic changes,[8] over 99·9% of actinic keratoses fail to progress to invasive tumours in any one year and as many as 25% may regress.[2]

Understanding the relation between melanocytes, naevi, and melanoma is considerably more confusing: some argue that melanomas are nearly always derived from pre-existing naevi,[9] while others argue that this is a rare event[10]; some believe that melanomas in the radial growth phase are incapable of metastasis (although what percentage of radial growth phase proceeds to vertical growth phase is unclear)[9]; and the never ending change in terminology for atypical or dysplastic naevi betrays the difficulty in relating histology to clinical behaviour.

Does questioning the relation between histopathological description and clinical behaviour have implications for those seeking to persuade people to change their attitude to sun exposure? The answer is surely "yes." The arguments relating melanoma to sun exposure are well rehearsed,[2] but the relation is not nearly as clear cut as it is between sun exposure and squamous cell malignancy. Most melanomas occur on skin that is only intermittently exposed; individuals with higher continuous sun exposure have lower rates than those exposed intermittently; and there seems an important interaction between skin type and incidence of melanoma. There is much else that is unclear[11]: we do not understand which part of the sun's spectrum is responsible for melanoma, nor the relative importance of ultraviolet radiation to mutagenesis, tumour promotion, or impairment of cutaneous immunity in the pathogenesis of melanoma.

The need for such understanding is underlined by models that predict that changes in the pattern of leisure exposure to the sun or that the use of sunscreens may actually increase rather than decrease melanoma risk.[12] There is after all no robust empirical evidence to defend most health promotion in this area. It has been suggested that the antithesis of science is not art but politics[13]; melanoma is perhaps an example of the two having become mistakenly intertwined. An amicable separation is required. The certainties of Health of the Nation and "slip-slap-slop" already look a little shaded: molecules care little for consensus.

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