(including melanocytic naevi, lentigo, and Mongolian spots) in the more pigmented races.[1 2] In a recent study[3] of 1058 neonates only 2% had proved pigmented naevi—0·4% lentigo and over 1% melanocytic naevi—and, since these "birthmarks" are universal, most naevi must in fact develop later in life. Indeed, pigmented naevi do grow and multiply during childhood, adolescence, pregnancy, and the climacteric. The risk of malignant change is negligible except in the rare giant pigmented naevus,[4] which is always present at birth, and which merits prophylactic surgery. Nature does sometimes destroy a naevus spontaneously in a remarkable immune phenomenon known as Sutton's naevus—leukoderma acquisitum centrifugum—when a white halo appears around a naevus and engulfs it. Otherwise treatment may be prompted by cosmetic considerations precipitated by an increase in size, or the patient may become anxious about her naevus, especially if it begins to grow hairs. Curettage, cautery, or shaving flush with the skin are reasonable alternatives, but the preferred treatment is by excision—though the insult of a surgical scar must always be weighed against the cosmetic offence of nature's blemish.

The common vascular naevi are of three types. Firstly, salmon patches may be found in over 40% of neonates.[1] Fortunately those on the face are evanescent, but those on the scalp may persist into adult life.[5] Secondly, strawberry marks —capillary vessel tumours—develop in over 1% of babies; few are detectable at birth, though some may be apparent as telangiectatic areas surrounded by a pale halo.[6] The typical tumour is obvious by six weeks, and it then takes up to a year to reach its maximum size. Involution is spontaneous, sometimes starting in the first year of life, but it is slow, and the mark may take several years to resolve completely. Nevertheless, nearly all have involuted fully by puberty. When marks are exposed to maceration by the napkin ulceration may be a problem; treatment may be needed with topical antibiotics and protective dressings. Injury may cause bleeding, but pressure will control it. In large or multiple lesions sequestration of platelets may result in haemorrhage (the Kasabach-Merritt syndrome), which is best treated by systemic steroids; a daily dose of 10 to 30 mg prednisone according to infant weight is needed for three weeks. This same treatment may be necessary should a strawberry mark interfere with feeding or the development of a vital organ such as the eye.[7]

Surgery has little to offer in treating strawberry marks, except that plastic revision may be required to deal with redundant skin from the involution of large lesions. Radiotherapy does hasten involution, but it is no longer considered good practice because the resultant scar is more unsightly than that arising from natural involution, and it may prejudice the natural growth of underlying and adjacent tissues. In general, a policy of firm reassurance and non-interference is to be preferred. Thirdly, the port-wine stain (found in less than 0·3% of the population[1]) is invariably present at birth. It follows a dermatome pattern and darkens slowly during life to assume its titular colour. Since no treatment is of any avail patients may need to use the excellent cosmetic cover preparations available. Sometimes stains may be surface markers of underlying vascular anomalies, as in the Sturge-Weber syndrome, where there is an associated intracranial lesion. When found on a limb a port-wine stain may be associated with haemangiectatic hypertrophy and arteriovenous communication.

Other naevi are uncommon. The naevus sebaceus of Jadassohn is present at birth as a fawn bald patch on the scalp; it becomes warty and darker with age, but in up to 30% of cases a neoplastic tumour develops in middle life,[8] so prophylactic surgery may be wise when the patient is old enough. Epidermal naevi start as yellow streaks; they also become warty and darker, but they are benign; they may be quite extensive, and, since they follow a dermatome distribution, surgery may be difficult.

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[4] Reed, W B, *et al*, *Archives of Dermatology*, 1965, **91**, 100.
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Less usual forms of epilepsy

Kinnear Wilson[1] said that a faint, a cry, or a laugh could be a fit and that no rigid semilogical framework could embrace all epileptic phenomena. Yet 50 years later neurologists are still fascinated by a wish to classify and categorise the multifarious fragments of an epileptic attack, which may be facets of a complex seizure or provide a patient's only presenting symptom. Epilepsy may be triggered by external stimuli[2]; and, whereas there are accepted interpretations of the commoner components of temporal lobe abnormalities, the less usual manifestations of epilepsy continue to intrigue and baffle. Recently, Sethi and Surya Rao[3] have published a finely written account of a patient with epileptic laughing, crying, and running in whom they found a well-circumscribed tumour of the left temporal lobe. In a complementary report[4] Offen *et al* described a patient in whom temporal lobe atrophy was associated with paroxysmal attacks of weeping, facial contortion, head turning, and amnesia.

The location of laughing (gelastic), crying (quiritarian or dacrocystic), and running (cursive) epilepsy in the temporal lobe is supported by the common concurrence of other clinical phenomena. Amnesia for the event is usual. Head turning has been reported in four out of six cases of crying epilepsy,[4] and the presence of epigastric auras points to the temporal lobe juxtaposition of visceral, expressive, and affective functions.[5] Many studies have provided evidence of temporal and frontotemporal electroencephalographic foci, but the EEG patterns are not stereotyped: Gumpert *et al*,[6] for example, found reduced electrical activity during the periods of laughter.

The term gelastic epilepsy (gelos = mirth) includes both a subjective experience of merriment (Dostoievsky described seizures of pleasure and harmony[7]) and complex co-ordinate movements with grinning, giggling, or joyful weeping. Patients usually experience a change in mood in parallel with its involuntary external expression: so that the internal states of pleasure or happiness, fear or terror, may respectively be exteriorised as smiling or laughter, crying or sobbing, attempt at flight or fight.[8]

Although the temporal lobe is most frequently inculpated in emotional epilepsy, grey matter elsewhere may be affected,[9] and one cannot always analyse the nature of the episode. Roger *et al*,[10] reviewing accounts of epileptic attacks accompanied by laughter or smiling found that where these were accompanied by EEG foci, temporal or occasionally Sylvian, further analysis was usually possible; but in over half the cases the laughter or smiling had accompanied petit mal and it was impossible to gauge the affective content of the attack. Two

possible subcortical sites for emotional epilepsy are the limbic system and the region of the hypothalamus. Parts of the limbic system, notably Ammon's horn and the amygdala, lie within the temporal lobe and are frequently associated with epilepsy. But attacks of laughter or weeping may occur with midline lesions abutting the hypothalamus,[11] basal ganglia,[11] and third ventricle.[12] Some are associated with orgasmolepsy[11] and sexual precocity.[13] Patients may die of laughter[14 15] and Foerster and Gagel[15] recounted the story of an operation under local anaesthesia where the act of swabbing the floor of the 3rd ventricle led to outbursts of hilarity.

The pathogenesis of laughter and crying has inspired eminent neurologists[11 13] to seek to define stages in the evocation of laughter at the cortex, in the posterior hypothalamus, and in the bulbar protuberances activating faciorespiratory pathways. Ironside[13] differentiated the involuntary and explosive laughter of pseudobulbar and bulbar syndromes, fits of laughter, and disorders of laughter consequent upon mental changes—as with the Witzelsucht of frontal lobe lesions (in which the patient is intensely amused at his own poor jokes). Laughter may prove a powerful trigger for cataplexy, and if unilateral[16] the cataplexy may strikingly resemble epilepsy. When laughter or weeping are found with bulbar palsies, multiple sclerosis, hypothalamic disorders, or infarcts giving hemiplegia, and after prefrontal leucotomy,[17] they are release phenomena. These differ from epilepsy in that the patient is usually conscious of the inappropriate paroxysm—and may indeed be disgusted by the involuntary outburst it provokes.[18]

[1] Wilson, S A K, *Journal of Neurology and Psychopathology*, 1924, **4,** 299.
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[3] Sethi, P K, and Surya Rao, T, *Journal of Neurology, Neurosurgery, and Psychiatry*, 1976, **39,** 823.
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"While the balance of his mind was disturbed"

Little attention has been paid to the emotional impact of suicide inquests on the families concerned—and in particular on the husband or wife of the deceased. Barraclough and Shepherd, who recently studied the subject,[1] found that despite the courtesy and kindness of the coroner in most cases the inquest inevitably aggravated the distress resulting from bereavement by suicide. As they point out, it is now five years since the Brodrick Report[2] recommended that the coroner should have the discretion to dispense with inquests except for suspected homicides, unidentified bodies, and the deaths of those deprived of their liberty. The report is still on the shelf. Apart from the inquest itself press accounts may be a further source of distress; but here Barraclough and Shepherd disagreed with the report, which recommended no restriction on the reporting of suicide cases even if no inquest was held.

Certainly in most suicides the intention of the deceased and the circumstances of death are so clear-cut as to render any kind of publicly held inquest unnecessary—as indeed is apparent in Denmark, where a much less legalistic procedure is considered satisfactory. While coroners are mostly kind and the police usually helpful, events may sometimes take a remarkable turn. Thus, in one recent case (not one reported by Barraclough and Shepherd) an elderly man suffering from Parkinsonism and a mild degree of depression went with a party of other patients for a walk in a nearby park in the charge of a competent nurse. Having shepherded her group to the bus stop, the nurse warned them in due course that the bus was approaching. Quite suddenly and unexpectedly the patient stepped in front of the bus before it had completely stopped. Though most of the onlookers thought that the cause was probably the patient's having misunderstood what the nurse had said, because the death was due to a road accident it was necessary for the coroner to sit with a jury. Even after retiring twice the jury found it impossible to reach unanimous agreement, so that after some shifts in opinion the coroner finally accepted a majority verdict of six in favour of suicide and two against. The jury's inability to agree unanimously caused some degree of distress both to the patient's relatives and to the charge-nurse, who, although in no way to blame for what had occurred, none the less felt much concerned. Perhaps if the coroner had been able to sit without a jury he might have found himself in a position to give an open verdict, but in the circumstances this was hardly possible. There might also have been grounds for the patient's relatives to have exercised their right of appeal, but (as Barraclough and Shepherd state) relatives rarely know they have this right and are seldom told about it by the coroner.

There is yet another possible disadvantage to the public inquest system: it may distort the accuracy of the suicide statistics because some coroners tend to bring in a verdict of accidental death whenever there is any doubt about the deceased's actual intention, presumably to spare the relatives' feelings. Thus it sometimes happens that those who commit suicide but who do not die until a few days after the event may admit their intent to someone or other in the interim period. This, however, may not come to light at the inquest. Where there is no public inquiry distortion of the truth is possibly less likely. Thus the Danes, who have one of the highest suicide rates in Europe and are sensitive about it, maintain that this is because their system, which is based on having no inquest, gives a truer account of the matter.

Kessel,[3] who devised a project to measure the extent to which international comparisons of suicide rates were reliable, came to the conclusion that because more rigorous standards of legal proof were required in England this possibly could influence the verdict. But protection of the feelings of the bereaved should surely be the prime consideration. If a more humane system led to verdicts becoming more accurate that could be regarded as a statistical bonus.

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