

edited video tapes, and a group already exists—the British Cardiovascular Intervention Society—that could organise regular postgraduate programmes along these lines. As in the United States, such meetings will provide an essential forum for teaching and discussing new developments—such as lasers, stents, and atherectomy catheters—and are probably the cornerstone for training new and established practitioners alike.

The number of angioplasties in Britain has doubled yearly from 712 in 1984 to 4200 in 1987. A further increase to around 5500 occurred in 1988. Faced with this growth, consultant cardiologists will in future probably wish to practise angioplasty. Current and aspiring senior registrars should thus seriously consider fulfilling the American guidelines for acquiring this skill. On completion of the implementation of the recommended training programme most cardiologists will be competent to perform simple or single vessel coronary angioplasty procedures. More complex or multivessel lesions

may be best tackled by subspecialists.<sup>7</sup> But the message is clear: an expanded well trained workforce is needed to tackle this growing workload.

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## Hearing voices

### *May be normal but happens most commonly in schizophrenia and alcoholic hallucinosis*

The experience of hearing voices that are not there occurs most commonly in schizophrenia and alcoholic hallucinosis. Occasionally it occurs in depressive illness, mania, or after damage to one of the temporal lobes, and very rarely it is a manifestation of hysteria. Under certain conditions it may also be a transient phenomenon in normal people. In most cases the characteristics of the voices enable doctors to distinguish among these various conditions.

Just over half of patients with schizophrenia will hear voices at some time in their illness.<sup>1</sup> The voices have a particular quality: they come from one or more unknown persons; they are recognised as different in tone from the patient's own voice; the sex of the speaker is usually identified; and whereas we normally think in the first person the voice usually uses the second or third person. The voices tend to increase if sensory input is restricted<sup>2</sup> or if the patient is aroused, angry, or tense.<sup>3</sup> They usually decrease if the patient listens to meaningful and interesting speech<sup>2</sup> and, paradoxically, if he or she becomes drowsy.<sup>4</sup> The voices are poorly localised in space,<sup>5</sup> and even if they are localised there is no consistent lateralisation.<sup>6</sup> Interestingly, schizophrenic patients who have been profoundly deaf since birth hear voices; on questioning, they may say that someone is communicating with them through sign language.<sup>7</sup>

Most studies have found nothing wrong with the hearing or intensity of auditory imagery in schizophrenic patients who hear voices,<sup>8</sup> and the voices probably represent inner speech—that is, our own thoughts—which for some reason acquire an unaccustomed tone and grammatical form. Indeed, sometimes the voice echoes the patient's own thoughts as he or she thinks them—a phenomenon that is so characteristic that it is a first rank symptom of schizophrenia.<sup>9</sup>

Usually, the voices heard by schizophrenic patients will disappear along with the other symptoms once the patients have been treated with neuroleptics. Occasionally they persist despite adequate treatment, and in such cases it is worth enlisting the help of a clinical psychologist as simple procedures may sometimes diminish them. Such procedures

include wearing an earplug in one ear<sup>10</sup> or encouraging the patient to identify the conditions that are most likely to make the voices disappear—for instance, resting.<sup>11</sup>

Alcoholic hallucinosis is a variant of delirium tremens and is usually self limiting but may last for months rather than days. The chief symptom is hearing voices, and the voices have a different quality from those encountered in schizophrenic patients<sup>5</sup>: they are well localised; there is usually only one voice; the speaker of the voice is often identified; and meaningful noise and drowsiness cause them to increase. In general their sensory qualities are prominent, suggesting that unlike the voices heard by schizophrenic patients these voices are false perceptions of environmental sounds. In one study they generally coexisted with tinnitus, and the tinnitus preceded and outlasted the voices.<sup>12</sup> In another study patients with voices also experienced clicks and fluttering sounds, and the authors suggested that the voices were misinterpretations of real movements of the middle ear muscles.<sup>13</sup> In general, the voices respond poorly to neuroleptics but clear if the patient remains abstinent.

Voices may be experienced by manic or depressed patients at the height of their illness. The content usually gives a diagnostic clue, reflecting the grandiose or self deprecatory mood of the patient. In addition, the quality of the voice is no different from a customary thought, a fact that is recognised by the patient and that psychiatrists call a pseudohallucination. The voices heard as an aura of temporal lobe epilepsy are usually accompanied by non-verbal hallucinations—for example, music—and the voice is usually of someone known to the person and is experienced as part of a complex personal reminiscence that includes visual hallucinations.<sup>14</sup> Very rarely a patient with a severe personality disorder will report experiencing a stereotyped voice, usually of someone who is the focus of their anger; the patient may communicate with the voice and claim to see the person at the same time.

Finally, normal people may hear a voice when dropping off to sleep (the hypnagogic state), when tired, exposed to extreme sensory and social isolation, or at the height of a

bereavement. The voice may simply call the person's name. Normal people usually have no difficulty recognising the unreality of this experience.

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## Diet and dermatology

### *Food explains some, but by no means all, skin conditions*

"Could it be something I've eaten doctor?" is guaranteed to provoke a sigh from most doctors. Even so, although many dietary hypotheses have now been discarded,<sup>1</sup> diet does cause some skin diseases and may modify others.

Too much or too little food may affect the skin. Some obese patients with insulin resistance and hyperinsulinaemia develop acanthosis nigricans and skin tags,<sup>2,3</sup> which may be due to the binding of insulin to peptide growth factors.<sup>4</sup> The same cellular defect may predispose to both obesity and acanthosis nigricans, but diet is probably at least partly responsible because the insulin resistance responds to fasting.<sup>5</sup> Too much of a single food constituent may cause problems. Hypervitaminosis A causes diffuse alopecia and hypercarotenaemia discolours the palms and soles orange. This occurs usually in slimmers having low calorie diets who eat too many carrots and oranges.<sup>6</sup>

Chronic starvation deficiency of protein and calories causes a scaly rash, and the hair tends to become dry and sparse.<sup>7</sup> Kwashiorkor produces a scaling of the skin, which often takes on the glazed appearance of adherent enamel flakes, and a characteristic reddish discoloration of black hair. The hair may also show the "flag sign," with alternating dark and light bands in individual hairs, which is due to intermittent protein deficiency. Measurement of hair growth has been suggested as useful for assessing nutritional state, particularly for proteins.<sup>8</sup> Patients with anorexia nervosa develop dry skin, lanugolike body hair, thinning of the scalp hair, and brittle nails.<sup>9</sup> The cutaneous manifestations of specific deficiencies of various vitamins and minerals provide classic examples of dietary dermatoses.<sup>10-13</sup>

Food and drink may be either inherently toxic or toxic as a result of contaminants such as fungi or added chemicals. Alcohol is a cutaneous vasodilator, and the Chinese and Japanese are particularly susceptible to alcohol induced flushing.<sup>14</sup> Chillies and red peppers also produce flushing and sweating by a pharmacological action.<sup>15</sup> Ergotism, characterised by severe Raynaud's phenomenon leading to digital gangrene, is due to eating bread made from rye infected with a fungus. An example of a dermatosis caused by chemical contamination occurred in Turkey, when the ingestion of wheat treated with a fungicide, hexachlorobenzene, resulted in many cases of porphyria cutanea tarda.<sup>16</sup>

Urticaria is commonly due to ingested food allergens, but the gluten enteropathy of dermatitis herpetiformis is a more subtle example. If gluten is avoided and the bowel recovers

completely the skin disease may go into complete remission.<sup>17</sup>

Idiosyncratic susceptibility to dietary factors may be commoner than appreciated. Some cases may be due to interactions with systemic medication—for example, the severe alcohol induced flushing in some patients who take chlorpropamide<sup>18-20</sup> and the reaction to cheese or pickles in patients taking inhibitors of monoamine oxidase. Other reactions may be due to anatomical abnormalities such as the flushing that accompanies the postgastrectomy dumping syndrome. Some idiosyncratic reactions—for example, the urticaria and bronchospasm caused by bisulphites used to keep fruit and vegetables fresh—may be mediated by neural activity as the results of tests for allergy are negative and the adverse reaction is blocked by atropine.<sup>21</sup> The Chinese restaurant syndrome, in which similar symptoms occur after eating sodium glutamate, seems to be mediated by the transient release of compounds similar to acetylcholine.<sup>22,23</sup>

Clinical observations have led to many attempts to improve skin disease by changing the diet, though few have stood the test of time. Some improvements may be due to idiosyncratic reactions. Many patients think, for example, that chocolate makes their acne worse despite several studies showing that it does not affect its course.<sup>24,25</sup> The value of exclusion diets for treating atopic eczema is also controversial<sup>26,27</sup>; though these are widely used, it seems that women's magazines have more success with them than doctors.

The use of fatty acid supplements to treat atopic eczema is another idea that was fashionable in the 1930s, died out in the 1950s, and has recently been exhumed in the light of increasing knowledge of fatty acid and eicosanoid metabolism.<sup>28</sup> Patients with atopic eczema have low concentrations of essential fatty acid metabolites in their plasma,<sup>29</sup> and some plant and fish oils are particularly rich in these compounds and their precursors. Evening primrose oil and borage, for example, are rich in  $\gamma$ -linolenic acid, which is depleted in atopic eczema. Several trials have shown that oral evening primrose oil in large doses has beneficial effects in eczema,<sup>28-33</sup> and meta-analysis of several controlled trials has shown that the clinical improvement correlates with the increase in plasma fatty acid metabolites after such treatment.<sup>34</sup> Similar studies have shown modest benefit from dietary supplements with fish oil in psoriasis and eczema.<sup>28</sup> The exact mechanism of these clinical changes is uncertain, but a better understanding of the intermediary metabolism of essential fatty acid metabolites and trace metals such as zinc might pave the way