surgical treatment, which is otherwise the treatment of choice. We have made all calculations on the assumption that gastroscopy is error free. This is untrue but the error is slight, can be measured, and can easily be included in the calculations.[6]

This model of the decision problem is an accepted one and the development of a satisfactory model is usually half the battle. The other half is the estimation of values for the variables, in particular the estimation of the utilities for states of health. We can only touch on this difficult problem.

Utilities have an "as if" existence. If doctors act consistently then their actions can be thought of *as if* there were a value or utility attached to a state of health coupled with a probability of attaining it. We can elicit this implicit utility only by testing a doctor in either a real or a simulated situation and finding out with what probabilities of success or failure he would advise a particular course of action such as a surgical operation. From his replies to a group of problems, we can estimate his utility function—that is, the values or utilities he attaches to a set of states of health. The method is promising but is in its infancy.[7-10] We give this example of an "ulcer-cancer" to show that we can now envisage the possibility of measuring the cost effectiveness of a test with some degree of objectivity.

Professor W I Card acknowledges with gratitude a personal grant from the Scottish Home and Health Department.

These papers have been written by members of the Computer Workshop of the Royal College of Physicians, which seeks to study the extent of the contribution that mathematical methods of decision making can make to clinical medicine. The workshop is open to research workers, and advice is available to those who are interested in attacking the sort of problems discussed in this paper. Letters should be addressed to Dr Peter Emerson, chairman of the Computer Committee, Royal College of Physicians, 11 St Andrews Place, London NW1 4LE.

This is the third of three papers in this series.

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For Debate . . .

Migraine prodromes separated from the aura: complete migraine

J N BLAU

Summary and conclusions

Detailed questioning of 50 patients with uncomplicated migraine has shown that 17 had symptoms that preceded the headache phase by several hours. These prodromes consisted of changes in mood, behaviour, wakefulness, appetite, bowel activity, or fluid balance. The term "complete migraine" is proposed for attacks that include prodromal symptoms, whose occurrence implies an initial diffuse cerebral or hypothalamic disturbance.

Introduction

A migraine attack may start suddenly with an aura or develop slowly with a mounting headache. There is a phase, however, even before the headache or aura that is recognised by most clinicians as well as some patients. Thus George Eliot felt "dangerously well before an attack"[1] but Sir John Forbes had an "irresistible and horrid drowsiness"[2]; Lady Conway ate her supper with "a greedy appetite" and her "pain would almost certainly follow the next morning,"[3] whereas Du Bois Reymond's migraines were "in general preceded by constipation."[2]

In a recent review Friedman[4] wrote, "Some investigators believe that these changes (prodromal symptoms) are not related to the migraine syndrome . . . [my] "experience, however, indicates that these symptoms are part of the migraine attack and are therefore truly prodromal." Surprisingly, this facet of the clinical picture of migraine has received little attention. Hence I undertook this prospective study of 50 patients with migraine uncomplicated by other conditions.

Patients

All patients were seen by me in outpatient clinics in neurological or general hospitals or in a migraine clinic, having been referred by a doctor. The history was taken to establish the diagnosis of migraine, as defined,[5] and whether it was the common or classical variety. Patients were then asked if they had any indication beforehand that an attack might follow. Some gave spontaneous descriptions of preheadache symptoms. Others had to be asked directly if they noticed variations

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in mood, alertness, wakefulness, behaviour, or urinary or bowel frequency; if they yawned excessively; became unduly hungry; or developed a desire or dislike for particular foods. Patients were further questioned how long before a migraine they became aware of these symptoms, if they were initially observed by themselves or a relative or both, how long they had been aware of such symptoms compared with the age of onset of migraine, and if prodromes "always, usually, often, or occasionally" preceded an attack.

Patients with migraine only were studied excluding those with additional symptoms, such as tension headaches and other conditions. Consecutive cases were recorded, and the study was completed when 50 was reached.

Observations

Of the 50 patients (28 female, 22 male) aged from 12 to 59 (average 34), 15 had classical migraine with a visual aura while three had hemiplegic and one basilar migraine. These auras lasted 10 to 30 minutes followed by headache lasting several hours. The remaining 31 subjects had common migraine.

PRODROMES

Seventeen patients had prodromes—four with classic, one with hemiplegic, one with basilar, and 11 with common migraine. The prodromal symptoms (table) lasted 1-24 hours (average 10) before the headache ensued. Prodromal symptoms, whether single or multiple, usually remained consistent for the individual, although cases 2 and 14 noted that the initial mood elevation did not always precede the tired and yawning phase that could by itself herald an attack. Case 1 yawned only before day-time attacks.

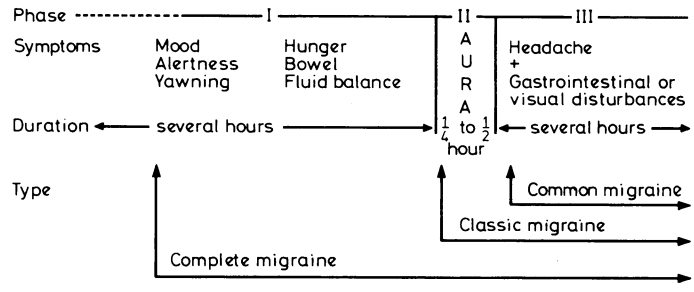
Prodromes occurred variably: thus symptoms preceded attacks "always" in two, "usually" six, "often" four, and "occasionally" four. One patient (case 11) noticed her symptoms only once before her most recent migraine.

Seven patients said that their close relatives—parents, spouse, or children—either drew their attention or commented on the prodromes. The remainder were the patients' own observations.

Thirteen of the 17 patients could estimate how long they had had migraine (a total of 241 years) and how long prodromes (96 years), giving an average of 18 years of migraine compared with seven years of awareness of prodromes. One subject (case 7) could state precisely that he had had migraine for 20 years but diarrhoea, tiredness, and yawning for only the past two years.

Discussion

Prodromes have often been mentioned in an anecdotal fashion but have not gained sufficient attention to be studied. The reasons may be that patients fail to mention symptoms that do not always herald an attack, or may be unaware of subtle changes in mood or behaviour unless attention is drawn to them by an observant and close relative; symptoms may only be recalled retrospectively in the same way as one has to point out precipitating factors in migraine; and doctors may not listen. Nevertheless, this study supports Friedman's[4] view that one or more symptoms, characteristic for the individual, may be forerunners of an attack, which in this selected series were as common as the aura.



Phases, duration, and symptoms of migraine attacks.

Confusion may have arisen in the past because prodrome and aura are synonyms (*OED*). To remove a semantic difficulty I propose the following definitions: migraine *prodromes* are symptoms with an insidious onset, which last several hours and affect mood, behaviour, wakefulness, gut motility, or fluid balance. The *aura* begins suddenly, lasts minutes, and commonly affects vision and, less often, somatic sensation, motor, speech, or brain-stem function. These temporal and qualitative differences enable the phases of a migraine attack to be represented diagramatically (see fig) and imply dysfunction at various sites of the nervous system that will be discussed.

Prodromal symptoms with timing and type of migraine

Case No	Mood variations	Somnolence and mental state	Alimentary	Fluid balance	Type of migraine	Time before headache
1		Yawning only			Common	2 hr
2	Jocular. Elated	Then tired and yawning	Hungry with desire for sweet foods		Common	Evening before
3	Feels particularly well. Bossy and noisy				Common	Day before
4	Tense. Low	Sleepy	Hungry. Eats carbohydrates	Bloated feeling	Common	Several hours
5	All systems go. On top of the world				Common	Day before
6	Snappy with brothers				Classic	2 hr
7		Tired and yawning	Diarrhoea		Classic	2 hr
8	Depressed. Quiet		Dislikes tea		Classic	Day before
9	Feels like a caged animal	Yawning			Common	1 hr
10	Irritable	Tired	Craving for food		Common	Evening or day before
11			Hunger mixed with nausea	Increased micturition frequency	Common	Several hours
12	Does too much				Hemiplegic	6-8 hr
13		Tired and yawning			Basilar	Day before
14	Ebullient. Great feeling of being well. Jocular	Then tired and yawning	Craving for sweets		Classic	Day before
15	Butterflies in stomach				Common	2 hr
16	Anxious	Fuzzy and muddle-headed. Tired and yawning			Common	Several hours
17	Irritable. Indecisive	Hazy mentally. Clumsy	Constipated		Common	Day before

SITE OF ORIGIN OF THE VARIOUS PHASES OF A MIGRAINE ATTACK

Phase 1—prodromes

The diametrically opposed variations in mood, alertness, or behaviour described here also occur in acclimatising mountaineers,[6] in hypoglycaemia, or more commonly in mild alcoholic intoxication. Such symptoms and signs indicate dysfunction, either diffusely in the cerebral hemispheres or focally in the hypothalamus.[7] Yawning, somnolence, hyperphagia or food rejection, and variations in fluid balance are characteristic of patients with hypothalamic tumours[7] or animals with surgical lesions in the ventromedial nucleus of the hypothalamus.[8]

Phase 2—aura

The classic aura is accepted as arising from stimulation or inhibition of a restricted area in the cerebral cortex or, in basilar migraine, the brain-stem.

Phase 3—headache

The headache phase accompanied by nausea, vomiting, photophobia, or phonophobia is thought to arise from extracranial vasodilation[9] or from a vasomotor instability of the meningeal circulation,[10] although the mechanism of photophobia or phonophobia remains a mystery.

New classification

One may now propose three types of migraine attacks: (1) common migraine—phase 3 only; (2) classic migraine—phases 2 and 3; and (3) complete migraine—phases 1, 2, and 3 or phases 1 and 3.

"Complete migraine" was the phrase used by Kinnier Wilson[11] 40 years ago: "In a complete attack cortex, deeper grey matter, hypothalamus, and meninges all seem to share but the lines of radiation of discharge from the primary occipital focus are unknown." One may agree with the first part of the sentence but this study indicates that the occipital cortex need not be the primary site of activation. At times prodromal symptoms occur without an ensuing headache; if we knew why or how we would be nearer a solution to the problem of migraine.

Kinnier Wilson[11] also recognised that the phases of a migraine attack may overlap or even telescope into one another, and some patients in this series noted that prodromal symptoms persisted during the headache phase. If there is such a continuum then we must seek one mechanism to account for the whole migraine episode.

Migraine: a neural hypothesis

Assuming that prodromes form an integral part of a migraine attack, we can postulate a unifying concept: a complete migraine begins with symptoms of diffuse cerebral disturbance that spread to the hypothalamus, or begins with a hypothalamic disturbance to give rise to prodromes; next, a focal area in the cortex or brain stem provokes the aura—a phase that may be absent; finally, the culmination of the attack—disturbances affecting the meningeal or other cranial vessels that are responsible for headache, vomiting, and photophobia.

This hypothesis leaves open the pathophysiology that induces dysfunction at these anatomical levels. The two major rival theories of the pathogenesis of migraine have been vascular or neural. The vascular theory assumes that cranial vessels overreact to one or more circulating agents independent of the autonomic control of the affected vasculature, and variation in calibre of these vessels provokes the attack sequence. The alternative neurogenic theory implies a primary neural stimulus that in turn induces a vasomotor response. Gowers[12] supported Liveing's[2] neurogenic hypothesis, which has lain dormant for several decades until resuscitated by Herberg,[8] Rao and Pearce,[13] and more recently by Johnson.[14]

The points in favour of a neural pathogenesis are (1) the occurrence of prodromes implicating the hypothalamus, the autonomic ganglion of the head; (2) the duration of prodromes before the headache; (3) the occurrence of an aura; and (4) the existence of sympathetic and parasympathetic innervation of pial and cerebral vessels, and that stimulation of the hypothalamus or cervical sympathetic ganglia can alter the calibre of these vessels.[15,16] Whichever theory proves correct, we are provided with a fundamental separation of migraine from other headaches—namely, additional neural disturbances, whether in the form of prodromes or aura before the onset of headache, or the autonomic disturbances that accompany the headache phase. This study further suggests that neurogenic mechanisms in migraine, particularly the hypothalamus and the neural control of cranial blood vessels, are worth reappraisal.

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Can air-freshener sprays cause urticaria?

Inhaled allergens may certainly produce urticaria. With air fresheners this would be due to the perfume content rather than the propellant.

A cleansing cream used by a cosmetic camouflage group contains borax—about 0·25%. As this is used daily to clean the skin is there any appreciable risk of toxicity? The skin is intact and the area affected will vary from perhaps less than 1% to less than 10% of body surface.

Borax may be absorbed through mucous membranes and non-intact skin, and topical preparations applied to the napkin rash of small children has given rise to serious systemic poisoning. On the other hand, intact skin is largely impervious to this material. Even daily use of cleansing cream containing no more than 0·25% borax on intact skin should cause no harm, especially if applied to only 1% to 10% of the body surface. But is borax really necessary in this context?