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REVIEW

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Feeling and seeing headaches

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Abstract The aim is to deepen our understanding of headache by three approaches. First, by trying to feel patients' total experience by eliciting their symptoms in detail, and from their reactions to these experiences. Second, by trying to remember one's own experience of headache, and observing a few patients during different headache types. Third, by attempting to see the different mechanisms of headaches by their sites of origin and their pathophysiology. Migraine, tension-type and cluster headache are the three headaches examined by these approaches. Migraine seems to arise from disturbances of the brain's cortex followed by meningeal pain – hence is intracranial in origin. Tension-type headache seems to arise from extracranial muscles, although the pain derives from the fascia or tendons of those muscles; common sites are the masticatory apparatus and the neck – hence extracranial. Cluster headache remains a mystery although vasodilatation provokes, and vasoconstriction stops, attacks – hence vasomotor control is therapeutically valuable. It is concluded that we need more adventurous ideas to deepen our understanding of these and other headaches.

Key words Migraine • Tension-type headache • Cluster headache • Intracranial and extracranial origin

Introduction

"All our knowledge has its origin in our perceptions", an inspiring and challenging aphorism from Leonardo da Vinci [1], writing secretly to himself in his notebook. It stimulates the question, can we expand our knowledge of headaches by increasing our perception of these conditions? Perception, meaning apprehending through our mind by one or more of the senses, includes our own experiences as well as those of patients who consult us.

Da Vinci is rightly famous for his paintings and sculptures. But the editor [1] in the preface to notebooks points out that "He often made use of his knowledge and experience for practical purposes" – surely applicable to medical therapy and research even now. Further, he taught young artists to look more deeply, to portray their subjects in depth and not superficially.

Here I have a dual purpose: by feeling and remembering the experience of our own headaches, we gain empathy with patients, enabling us to communicate better with them, thereby learning more from *their* experiences. Secondly, by trying to see headaches more penetratingly, we may be able to uncover the anatomical sites where headaches and associated symptoms arise, and the pathophysiological mechanisms underlying symptoms and signs.

I trust we can agree that some of our current knowledge is wrong, and all knowledge is limited; hence the importance of admitting ignorance and saying "I don't know"; then we can ask pertinent and relevant questions. I shall explore trying to feel and see the three headache types frequently encountered in clinical practice – migraine, tension headache and less often cluster headache.

Remembering our own headaches

You have at least a 98% chance of having experienced some type of headache: a study of 327 medical students, aged between 18 and 21, showed that only 7 (2%) had never had a headache in their life [2]. Their headaches arose from different causes, including hunger, alcohol, ice-cream, diverse activities particularly reading and travel, from stress, insufficient or excess sleep, exposure to excess light, noise or heat, and in females menstruation. The few who had never experienced a headache are equally important, because if we knew how they were so fortunate, we might well be a long way forward in our therapies.

Headaches arising outside the skull are easy to feel; you only have to recall hitting your head on a door. Deep seated pain is a little more difficult, but most of us have had some abdominal pain or discomfort which differs markedly from wearing a belt that is too tight round one's waist: I use this analogy when asking patients whether their pain is superficial or deep; not all can give a confident answer, but it often gives a lead in the important differentiation between intra- and extracranial headache, as discussed later.

However, before going into details, it is salutary to quote Henry Head [3] the great neurologist, who in 1926 wrote "Neurology has become frozen stiffly in the grip of pseudo-metaphorical classifications which neither explain the conditions nor correspond to the clinical facts. The dangers of such false classifications were well recognised by Jackson in 1878."

If you think things have changed, look at the latest International Headache Society (IHS) Classification and Criteria [4]: the classification has greatly expanded but I am uncertain whether it is clearer. Further I have doubts whether we diagnose illnesses by criteria; I suspect pattern recognition, which applies to recognising foods or acquaintances, is equally applicable to diagnosing diseases. (A dog recognises its owner without criteria.) Of course when we write papers we need to know that criteria were satisfied in the included cases – a different matter. This is a personal opinion to which I trust each of us is entitled.

Migraine

How do patients feel and see their migraine attacks?

Many physicians interested in headaches have migraine themselves. If so, the only point to make is that in life and in medicine, there are always individual differences in qualities and severities. However, if you have not had migraine, most of us have seen at least one person, whether a patient, relative or an acquaintance, during a severe attack. Also patients tell us they go to bed and lie still because head movement accentuates head pain; the head is buried under bed clothes or pillow, or the curtains are drawn because light hurts the eyes; they feel cold particularly in hands and feet; many have facial pallor; the bedroom door is shut to keep out noise, smells or visitors; the bathroom door is kept open, or a bowl or bucket is at the bedside in case of vomiting; above all they try to attain sleep because then head pain is not felt, and often attacks are resolved by sleeping.

What do we see in migraineurs during their attacks?

The resultant picture we see from the above symptoms is clear, from which we can draw certain conclusions. Head movement increasing pain suggests that the headache arises from the meninges, as in meningitis, or after a subarachnoid haemorrhage; photophobia, phonophobia and probably osmophobia possibly also arise from meningeal irritation. Autonomic disturbances are evident in facial pallor and cold peripheries, as well as intestinal malfunction; patients do not want to talk, perhaps the brain is not functioning normally, or they want to "sleep the attack off".

These observations, and what we deduce from them, are not seeing migraine, but are the *visible responses of subjects to their symptoms*, persisting for hours, often resolving by sleep occasionally for 1–2 hours, more frequently for a full night.

How can we learn more what patients feel and where can we help?

Further questions, when taking the history, enable us to learn more how that subject is affected, and provide details of whole episodes, including individual's modes of onset (prodromes and an aura), as well as different means of resolution, and postdromal symptoms. However, patients most often begin giving their histories by focussing on the headache, hence discussed first.

Patients may waken with an awareness in the head but are uncertain whether or not it is going to develop into migraine or not; or the headache can begin insidiously during the day; others waken with the headache fully developed. In the majority head symptoms begin slowly with a head awareness that increases to a pain taking from half to several hours, slowly building up to a maximal intensity. No one has tried or been able to explain the mechanism of this pain increase. The pain usually feels to be deep seated inside the head, can be behind the eyes, on one or both sides of the head; it can involve the whole head, or remain localised, or move from one side of the head to the other; it can be steady or intermittent pain, often briefly accentuated by coughing, sitting up or lifting objects. Most feel better lying down, but every so often one hears the converse – they go and rest on a sofa because they can sit propped up – also not satisfactorily explained.

Pain quality is the most difficult sensation to describe: a vast amount of emphasis has been place on "throbbing", all too often interpreted as arterial pulsation, when it can mean waxing and waning, or severe. As a result many eminent authorities held the view that the headache was due to vasodilatation, none more vehemently than Harold Wolff [5, p. 249], who found that his own migraine headache was reduced by pressing on his temporal or carotid artery; he gained support for this contention by tambour recordings from the temporal artery, and that ergotamine narrowed the vessel, giving some relief. However one ugly fact can destroy a beautiful theory: many patients have intense facial pallor, described by relatives as "looking as white as a sheet", "a ghost", "green" or "drained". But all generalisations have their contradictions, and occasionally a mother says that her child is flushed during a migraine; perhaps sometimes the child has a pyrexial illness that provoked a migraine.

Most migraineurs during an attack cannot eat or drink anything; even thinking about food may increase the feeling of nausea. On the other hand, a few can nibble a dry piece of toast or biscuit, which they find reduces the headache and/or nausea; some can drink a few sips of water. The majority are constipated, but the occasional patient has diarrhoea.

Attacks can end by fading away slowly; others "sleep the attack off". The amount of sleep required varies, some being helped considerably by a 1–2 hour lie down, others needing a full night's sleep even going to bed earlier than usual, and then sleep through the whole night.

In the majority, vomiting is highly unpleasant, with a temporary increase in headache. Surprising to mothers however, a youngster vomits, and 15–30 minutes later feels well enough to go and play football with others – which he does!

Further attack aspects that need eliciting from patients

Headache is the central and usually the most troublesome symptom. But there are phases before and after the headache, which often need eliciting from patients, some of which are not apparent to individuals. Premonitory symptoms are divisible into two phases: (1) prodromes that occur some hours before, even the previous evening, and (2) the aura that can have a variety of features and does not occur before every attack.

During *prodromes*, usually several hours before the headache onset, subjects can feel inappropriately tired, yawn excessively; others are euphoric and extrovert; some are pale or have "a strange look around the eyes"; craving for sweet foods especially chocolate (hence the erroneous attribution that chocolate precipitates attacks); some experience nausea even before the headache onset. Some signs can be apparent to observant partners or not infrequently by perceptive children; mothers of affected children are also good observers of these early symptoms, which are important, because when recognised can be treated so that the subject does not develop an attack. However no prophylactic trials have been conducted but seem well worthwhile, for obvious reasons.

Visual aura symptoms can only be observed by patients. There are brilliant personal drawings reproduced in papers by Airy [6], Gowers [7] and by Lashley [8], who had 4 different patterns reproduced in his paper from different stages of development. I show patients a reproduction of Airy's coloured drawings, and ask if they have a blind area, and the colour of the zigzags. Patients have rarely seen these, and are often very appreciative to see that others have similar experiences – because they fear talking about their hallucinations in case they are considered to be crazy, or more frequently that their visions are forebodings of sinister and permanent damage: they need reassurance.

Expressive dysphasia is more commonly heard from the elderly or those with mild dementia; I have not actually seen a migraineur in this phase talking "gibberish" because it is rare to see a patient during an aura. Nevertheless I have personally experienced and I suspect you have too, difficulty in naming something during a *viva voce* examination, or when under stress, or the name of a colleague whom you have not seen for a long time, and meet them in the street or at a medical meeting. Most patients have had a similar experience.

There are still more parts of attacks: resolution and postdromes, the latter often mentioned by patients, but rarely in books and unexplained. Migraineurs often add these, making attacks one or even two days longer than stated in the IHS criteria.

Attack resolution differs in a number of ways. Commonly patients say "they fade away" or "I usually

sleep the attack off". Many know from their own experience, or have been told by a partner or relative, that sleeping very deeply characterises their mode of resolving the headache phase. I wonder whether it would be interesting and perhaps teach us something if such sleep could be monitored with EEG; there may be differences in deep and REM sleep ratios compared with a normal night's sleep; my hypothesis is that deep sleep is longer than usual. However the amount of sleep required to end attacks varies: in some a 1–2-hour nap in the afternoon or early evening is sufficient, others go to bed as soon as they get home from work and then need a full night's sleep to fully recover.

Postdromes. Many, the day after an attack, feel like a "hangover", being tired, generally weak and sluggish, both physically and mentally, and even have aching muscles for half or the whole of the next day [9]; I have wondered if circulating muscle enzymes could be abnormal. Most feel relieved, and some euphoric; this is important because they "may do too much" making up for lost time, and by overdoing normal activities provoke another attack. As far as I know, postdromal symptoms, particularly the aching muscles, have not been explained.

These are the main features of attacks that we hear about, and some can be observed, thereby appreciating what patients experience. However, not all patients have all these features in all attacks. In fact the majority (85%) have never had an aura of any variety; prodromata as described above occur in about one in three subjects. Although many say at the first consultation that they are quite certain they have not experienced prodromata, are surprised when they ask their partner and hear that he or she can tell sometimes, even frequently; a close work colleague may need to be asked, because they may have noticed subtle personality changes before migraine episodes. Awareness of prodromata enables early treatment that may prevent a full-blown attack.

At the end of this overall picture we also occasionally encounter patients who develop a visual aura without ensuing headache or other symptoms. In such patients, the first attack often occurs over the age of 40, and men outnumber women, both facets that run counter to the usual migraine picture. Here I have been struck by reading or very bright lights acting as attack triggers, although lack of sleep can be an additional factor in attack provocation.

How can we deepen our understanding of migraine pathogenesis?

Localisation of attack origin

We need to consider what can we deduce about localisation and disturbance of function from observation – because they need to be incorporated in our, still incomplete, theories of migraine mechanisms.

There have been numerous theories about the underlying migraine process. Epilepsy was favoured by Hughlings Jackson and by Liveing. Vascular disturbances were in vogue at one stage, promulgated ardently by Harold G. Wolff [5, p. 269], a great headache researcher with many contributions to the experimental approach. The remains of allergic theories are still apparent in the dietetic field where patients are convinced that chocolate and cheese precipitate their attacks, although these have been disproven [10, 11]. The trigemino-vascular theory, introduced 20 years ago based on animal experiments, still has strong adherents, as has Leao's spreading depression although it has not been seen by numerous neurosurgeons operating daily on the human brain.

I begin by selecting one specific element of the migraine symptoms – dysphasia, because I know that it can only originate from the cerebral cortex, proven by Penfield and Jasper [12], and cannot arise in the brain stem, meninges, cranial nerve ganglia or blood vessels.

This view is supported by mood and behavioural changes evident during the prodromal phase of attacks, which are likely to arise from the frontal lobes and extend to the temporal lobes and amygdala, i.e., the limbic system, which is widely accepted as the cortical area controlling the autonomic nervous system. Further, the sensory disturbances that begin in the fingers, ascend the arm and manifest as dysphasia when the face is reached, also suggest that the sensory cortex is a primary site of origin. However, it is difficult to understand that dysphasia can also occur when sensory symptoms involve the *left* upper limb in a *right*-handed individual.

Next, there is no doubt that patients know that they are talking nonsense at a time when they can understand perfectly what is being said to them. This must mean that their word memory remains intact. Penfield and Jasper [12, pp. 140–146] have demonstrated by cortical stimulation in conscious human patients that patients know what they want to say but cannot express themselves while electrical stimuli are being applied, although they can as soon as the stimulus ceases.

Where and how we store even the simplest memory is a total mystery and I feel one of the major neurological problems of this century. Here are some speculations. It seems to me that memory is not going to be stored in dendrites, membranes, neurotransmitters, mitochondria or protoplasm of neurons; of course neurons need to communicate with each other for which neurotransmitters are essential, but that does not mean that memories are laid down in these chemicals. (Telephone wires or loud speakers convey but do not utter speech or thoughts.) I also feel that word memory is most unlikely to be localised in the

brain stem, or in trigeminal ganglia. However, nuclei in general with their DNA have shown that they are to be able to pass on instructions by messenger RNA and therefore must be able to store some information. This suggests that neuronal groups in the four speech cortices are potential sites of word memories. (I welcome contradictions.)

Sites of pain origin causing headache?

The first point to appreciate is that headache is stage three of the migraine attack, being preceded in about 30% by prodromata lasting hours, and in 15% by the aura lasting up to 2 h, but more commonly 20–40 min.

The evidence that the pain is due to meningeal vessels is as follows:

1. The slow build up taking minutes or hours is totally unlike neural, in particular ganglion pain. No one has explained this slow increase of headache, plus the fact that the head pain in many is not pulsating, or if it does pulsate, may only be brief.
2. Coughing, sneezing, breath holding or head shaking can all increase the pain. Here the classical experiments with histamine-induced headache by Pickering and Hess [13] showed that the pain arose intracranially. It is interesting that Pickering [14] also knew as long ago as 1939 that the increase of head pain by shaking the head from side to side indicated an intracranial origin for the pain. Dexter and I [15] used his technique, putting a blood pressure cuff round the head with the pressure elevated above systemic blood pressure. On observation of 50 patients during a migraine headache we found that the elevated pressure did not abolish the headache, but did increase with rapid lateral head rotation in 42/50, coughing in 32/50 and breath holding in 27/50. This provided support for the head pain arising from inside the skull, at least in the majority.
3. We know that meningitis is painful whether of viral, bacterial or chemical (subarachnoid blood) origin. Again nature does not always give simple answers – meningitis also giving rise to extracranial neck stiffness. Here it is interesting to note that some patients have overlying scalp tenderness on the same side as the deep headache and say they cannot lie on that side when retiring to bed.
4. Headaches can be provoked by many stimuli including hunger, alcohol, or too little sleep. All these can induce mental and physical changes which affect the brain as a whole, often progressively if the stimuli persist. Alcohol is an exception, producing cerebellar manifestations, absent in migraine. These diverse brain disturbances will not be analysed here.

It seems therefore that the pain in migraine attacks arises, at least in part, from the meninges.

Which other intracranial structures can provoke pain?

The *brain* as a whole is painless. But as always in biology there are exceptions: the thalamus (usually due to an infarct), the quinto-thalamic tract, the trigeminal ganglia, and nerve roots of the trigeminal and glossopharyngeal nerves, provoke pain of different varieties and in different areas.

Are arteries painful? Wolff [5, pp. 53–95] was deeply committed to the belief that pulsating arteries could be painful, but he admitted it was only in their proximal few centimetres. He found that the dura was painful especially near venous sinuses, which he rarely mentioned when discussing headache. However he realised that we all experience pulsation in the head with exercise, but without pain. He observed that the superficial temporal arteries became tender during migraine and postulated that they released a chemical which he called ‘headache stuff’, allied to bradykinin, which he postulated caused the extracranial pain.

Penfield and Jasper [12] had a broader focus – arteries and veins. Operating on the exposed human brain, they compressed dural meningeal arteries and produced accurately localised ipsilateral head pain. Pressure on dural sinuses, or traction on their tributary veins was also referred to the same side of the head. Manipulation of the sinus adjacent to the tentorium caused pain behind the ipsilateral eye [12, pp. 749–750]. Penfield and McNaughten [16] had shown earlier that superior sinus stimulation in addition to pain, provoked nausea, vomiting and yawning. However there was greater variability in the sensitivity of the middle cerebral arteries: in some individuals no pain was provoked, but in others ipsilateral pain ensued with electrical stimulation. They also expressed the view that perhaps it was the connective tissues around arteries which, when stretched, resulted in pain. In support, we know that harbouring an intracranial arterial aneurysm is not painful until it ruptures.

What helpful advice can we give to patients from these facts?

During lectures on migraine I often mentioned that patients frequently expressed uncertainty, particularly when they waken with some head discomfort or a mild pain, whether it is their “ordinary headache” or the beginning of a migraine. After one of these talks, a doctor came and told me his technique of differentiation: he would rapidly move his head from one side to the other, if the pain increased he took his anti-migraine medication.

A few months later, I was fascinated to read in an article by Alvarez [17], a quote from Soranus of Ephesus 125 AD, i.e., 1900 years ago (!): “A good way of telling whether or not a headache is going to be migrainous is to

sit with the head down below the knees. If the head starts to throb, it is a migraine, all right." I tell this to patients although have found the pain needs only to increase, not necessarily become throbbing. This goes to show that differentiating between an intracranial and extracranial headache is an ancient problem, and that knowledge is easily forgotten or mislaid.

The emphasis here has been on full analysis and early detection of migraine onset, to enable early and effective therapy to be taken by the patient. However, a wider approach to prophylaxis is to delineate triggers for individuals which, once recognised, may be avoided. These have been dealt with previously [18] and will not be discussed here.

Other intracranial headaches

There are many other intracranial headaches, some serious, others not. These are rarely recurrent, and many are of recent origin with progressive severity, often showing evidence of neurological deficit. I shall not deal with these here, because they are well described. However, it is essential that every patient has a complete neurological examination. In migraine, there are no abnormal physical signs; what patients need to hear: "your nervous system and brain are normal".

Tension or tension-type headache

If there are gaps in our understanding of migraine, there are oceans of ignorance about tension headache. I propose to put forward some definite syndromes in which there is extracranial muscle-fascia pain. I do not mind being wrong, because if wrong ideas stimulate correction and lead us to understand more on this topic, I have achieved something.

Both tension and tension-type headache are ambiguous because it is unclear whether the patient is tense (due to anxiety, or agitated depression, or both), or the extracranial muscles are tense due to wrong posture, joint or other local pain. Changing tension headache to tension-type headache, adds only a hyphen and 'type', but clarifies nothing. Nevertheless, I think we all agree that "This is the common type of primary headache, its lifetime prevalence in the general population ranges in different studies from 30% to 78%. At the same time, it is the least studied of the primary headache disorders, despite the fact that it has the highest socio-economic impact", as stated in the new IHS classification [4, p. 37]. The criteria laid down there deal mostly with frequency and timing, the latter ranging from 30 minutes to 7 days! It is also recommended that clinicians perform simple palpation of pericranial muscles including the frontalis, temporalis, masseter, pterygoids (!), sternomastoid and trapezius, preferably with a pressure sensitive device using controlled pressure and grading the pain severity from 0 to 3. There is no guidance on how the pains may arise, or what underlying mechanisms may provoke the pain, or where to seek possible causes. Drug overuse is mentioned and overlaps with migraine are suggested. For such a common condition, classification and criteria are prominent, but that little that can be done to understand and treat patients is apparent.

A difficulty arises from our training when there is little if any teaching on distinguishing organic from psychogenic disturbances. But we have to accept that the two come together – physical illnesses often provoking psychological reactions, and vice versa.

Seeing extracranial pain

Sitting opposite people on public transport, it is evident that some are clenching their teeth, seen in the masseter but less obvious in the temporalis muscles. Others are frowning and contracting frontalis and procerus muscles, resulting in wrinkling of the forehead, especially over the bridge of the nose. At work we see subjects staring at computers holding neck muscles in one position for hours, often for most of the day.

We talk glibly about muscle pain when the only known factor provoking pain in muscle is ischaemia, but even there the mechanism remains unknown; we have to talk about *muscle-fascia* pain, because we know from muscle biopsies that we have to anaesthetise the fascia but can cut and remove a piece of muscle without pain. Hence we have to use muscle-fascia pain, which although longer and clumsier, at least is precise. Pain can also arise from muscle tendons, or their origin and insertion into the periosteum, as well as neck and temporo-mandibular joints.

Feeling pericranial muscles

You can feel the temporalis and masseter with fingertips when clenching and relaxing the jaw a few times, on yourself and on others. Similarly the frontalis muscle can be felt to contract with the palm of the hand placed on the forehead by raising and relaxing the eyebrows. By placing fingertips on the back of the head and elevating eyebrows, you can feel the occipitalis muscles contract, transmitted by the

galea aponeurotica. Fingertips placed on the back of neck and rotating the neck from side to side, enables feeling the neck muscles. Placing the thumb anteriorly and finger tips posteriorly on the trapezius muscle and lifting the shoulder enables feeling the trapezius muscle, a common site of tenderness with neck pain, particularly near the midline.

These manoeuvres are not only useful for oneself but important to show patients so that they can localise their own sites of their pain origins. Further, they realise that their pains are extracranial and therefore not serious (see below).

Diagnosing extracranial muscle-fascia pain from patients' histories

Patients usually point the index finger to the pain site at the temple, indicating the anterior fibres of the temporalis muscle. Others draw a line in front of the ear ascending upwards to the temple, the pain arising from the temporo-mandibular joint or tendon of the temporalis muscle. Some draw a curved line over one parietal region corresponding to the origin of the temporalis muscle where pain endings are situated. These sites indicate pain arising from the temporalis muscle-fascia. The masseter is only very rarely painful.

Patients placing the palm of the hand across the frontalis may indicate pain transmitted from the temporalis, the occipitalis muscles or quite commonly from the neck.

The next most frequent site where headache originates is the neck; this has been known for a long time, but was brought to the fore by Sjaastad et al. [19]. When the pain is unilateral, patients indicate with one finger starting in the neck, moving it variable distances forwards, frequently as far as the parietal area or the forehead; or the same line may be drawn in the reverse direction. When the pain arises bilaterally from the neck, surprisingly often patients demonstrate with the palms, which they move forward or backwards, as others do with a finger. Those who indicate pain localised to the forehead need to be asked if they have neck symptoms; a positive answer is not infrequent, patients not linking the two sites.

Patients may say spontaneously or have to be asked whether any of these localised pains are helped by gentle finger rubbing, or can be increased by more pressure; they may have tried applying heat which eases the pain, or noticed that cold wind can accentuate it; similarly an ice-pack can help or hurt more. Physiotherapists have told me that in about nine out of ten subjects, heat helps and cold accentuates pain, but in one out of ten the converse applies. Analgesics usually significantly reduce or relieve the pain in 20–60 min, the pain returning to its previous severity after 4–6 h, or may not recur till the next day. All these are clear organic symptoms, which often need eliciting by direct, not leading, enquiry.

Examination for extracranial muscle-fascia pains

When we come to examine the patient, it is important to palpate all the above muscles. It is also advisable to examine the temporo-mandibular joint, listening with a stethoscope for a click or crepitus. Examining the relationship of the upper and lower teeth can show an overbite, or tips of teeth meeting in the midline, giving rise to malocclusion; this indicates that the pain may be of masticatory origin and the need for a dental opinion. Asking about nocturnal teeth grinding can be revealing: this may also have been commented upon by a dentist, who can see evidence of teeth having been worn away, or from a family member or partner sleeping in the same room as the patient.

The neck is examined by passive and active movements in six directions: flexion, extension, rotation and lateral flexion to each side, the last usually being restricted in middle age and elderly, due to neck osteoarthritis. However, some young patients with long slender necks may have joint hypermobility (Ehlers-Danlos syndrome), thereby being more liable to joint and muscle-fascial pains [20]. Recently I have discovered a new extracranial headache provoked by tying the hair too tightly in a pony-tail [21]. Pain can be felt at the site of the hair-tie only, or extend forwards to the vertex or as far as the forehead, laterally to the parietal regions or temples, or posteriorly down the neck. Consulting anatomy books I was surprised to find that the fascia of all these muscles overlap with one another, mentioned above, accounting for pain extension.

If the above views are correct, we can identify a number of different extracranial headaches depending on their site of tissue origin:

1. Masticatory apparatus, including teeth grinding, clicking jaws and malocclusion, all in the field of dental surgery.
2. Cervicogenic headache. This headache arises from the apophyseal neck joints, or neck muscle-fascia, or both, and is liable to occur in those sitting in one position for many hours, like drivers and computer operators; this too is not neurological, but in the field of rheumatology or physiotherapy.
3. Pain in one side of the face can arise from an ear infection, particularly in children: an examination with an auriscope is essential.
4. There are a host of non-neurological pains arising from the sinuses, eyes, ears, and referred pain from the chest, which will not be discussed here. However I have encountered a number of patients who said, rather diffidently, that their headache began in the lum-

bar region. These I show a diagram of paravertebral muscles, short, middle and long, running paravertebrally and therefore can occasionally reach neck muscles from where they produce a headache.

5. A diagnosis important not to miss is temporal arteritis. The patient is usually over 60, with severe pain on one side of the head, so that they cannot lie in bed on that side. They often have night sweats, anorexia and weight loss. The reason for urgent diagnosis (that needs to be checked by an immediate ESR and the patient waiting an hour for the result) is that the condition is remediable with cortico-steroids; if not treated immediately, patients may develop blindness from optic nerve involvement by the inflammatory process.

Psychogenic headache

Patients with this headache type usually say the head symptom is present from waking till going to sleep without any variation – "nothing makes it better and nothing makes it worse". They have usually tried every medication a pharmacist will sell them, as well as all their family physician's prescriptions, and none has had the slightest effect. The headache is usually all over the head, but when you ask "What is the pain like?" the prompt answer "It is not a pain doctor, its an ache". If you ask for elaboration then you hear "it's a pressure", and "it feels like a tight hat".

No one has explained these symptoms although an ENT surgeon who has patients that complain of pressure on the bridge of the nose told me that he compares the symptom with the sensation of wearing a new pair of shoes: we are aware of these, but as we wear them, we forget that they are new – unless they do not fit. I have also wondered whether the sensation transmitted by the trigeminal nerve and the second cervical nerve is the equivalent of dorsal column sensory transmission and not the spino-thalamic tract.

How therapeutic such explanations are, I do not know; further I am uncertain about the degree of benefit these patients derive from being referred to a psychologist or psychiatrist.

Difficult diagnostic headache problems

Patients with two types of headaches can be difficult to diagnose; about 1 in 3 patients coming to the City of London Migraine Clinic fall into this category. There are two approaches: One is to confuse the picture and talk about "transformed or evolved migraine", or "mixed headache". The second is to accept that patients can have two different headache types, simultaneously, and analyse each by dividing the problem into two components, initially for diagnosis and then for treatment. Rarely a patient comes with three different headaches; these are "heart sinkers", but not necessarily impossible.

First I ask the patient to tell me when the migraine started, how often attacks occurred, how long they lasted and to describe one of those attacks from its onset to its end. Usually a description of a typical attack of migraine without an aura is given, beginning in the teens or 20s.

Next I ask for a description of the present attacks – frequency, duration, location and other questions that usually produce either the picture of a muscle-fascia headache, and less frequently another headache due to lack of sleep or of psychological origin. Then it may be clear we are dealing with an additional headache.

However, it is essential to know what to do when this dual picture does not emerge. A useful question is "How many types of headache do you have?"

I find it valuable to remember that any pain in the head can provoke a migraine. And as pointed out above, the beginning of a migraine and another headache can mimic each other closely. How to differentiate the two has been dealt with, and teaching this to the patient is essential in clarifying the diagnosis. Another cause of confusion is that the headache responds to a triptan drug and many physicians maintain that this is diagnostic of migraine; but this is not true because some muscle headaches also respond, perhaps because muscles become congested with overuse, and a triptan reduces the increased blood flow. It is worth remembering that triptans also abort cluster headache, which differs completely from migraine; and secondly, that ergotamine used to be regarded as a specific anti-migraine drug when it had a profound effect on the uterus and on blood vessels.

Another helpful approach to diagnosing one or two simultaneous headaches, is to ask patients to keep a daily record of times of onset, site of pain, the effect of self-massage of the affected area, applying heat and cold, and analgesics, as indicated above. Also, to arrange a second opinion from a dentist and/or a physiotherapist depending on the site of the patient's pain. In some instances a psychological assessment can help; patients do not like being referred to a psychiatrist. The patient should then be reviewed when these above measures have been instituted and have had time to show their influence, usually 4–6 weeks later.

Cluster headache

Cluster headache is a rarity, hence should only be diagnosed when the history is typical; otherwise we are

diagnosing a rare manifestation of a rare disease, which makes the diagnosis very rare. Horton, the great American cluster headache expert, who had seen 1400 such patients, quite rightly said that once you had seen one patient in one attack you had no diagnostic difficulties.

What do patients feel and do?

They feel the headache building up from no pain to a maximum in 5–10 min. Their pain is deep seated behind one eye, and can remain there or spread to the back of the head; less frequently downwards to the upper teeth. They have an intense desire and need to get out of bed, when attacks waken them from sleep. Not only do they get up, they also want to leave the room so that the wife or partner does not see their distress, or their peculiar behaviour of walking around, hitting their own head, as well as moaning and groaning. If they sit down they continue clutching the head but rock backwards and forwards. A few kneel, moving the trunk up and down, even hitting the head on the floor. Patients are often surprised at their own behaviour [22]. They know the affected eye tears, and the ipsilateral nostril becomes blocked or discharges a clear fluid. Many sweat, not as the books say on the affected side of the face, but generally, or over the chest, or bilaterally from the neck, axillae or groins.

Attacks last 40–90 min, leaving them exhausted, drained, and above all fearful of the next attack.

What do we see?

There is no doubt that the conjunctiva of the affected eye has dilated blood vessels. The eye is often held partly closed compared with the other side. The pupil is said to be smaller but reports state a difference of 1–2 mm, which ophthalmologists allow as normal anisocoria. The upper and lower eyelids appear swollen; patients confirm this when they say that they feel there is swelling around the whole eye region. The patient is clearly in a great deal of pain, pacing around holding the head.

Dr Engel and I [23] were recently told by some patients and then asked others, that becoming hot, whether from the environment in a hot stuffy room or from a bath, or by exercise, can provoke an attack in the same manner as alcohol, presumably by increased internal blood flow.

Theory of mechanism of cluster headache and treatment

We have no explanation for the vessel dilatation in the conjunctiva of the affected eye or the ipsilateral nostril. Most patients say that the pain behind the eye feels as if the eye is being pushed out; they indicate this with a finger placed on the temple adjacent to the eye, the finger pointing forwards. Why patients feel compelled to leave the bed, go into another room or out of the house, why they have to walk around and feel intensely restless, why they want to hit their head, why attacks come in clusters, wake the sufferer up 1–2 h after going to sleep and last 20–120 min – all are mysteries. However lateral thinking shows that patients with peritonitis lie still (as in migraine), whereas those with renal colic are restless, even roll about on the floor, as well as sweat profusely (as in cluster headache).

There is no doubt that in a high proportion during a cluster spell, alcohol precipitates attacks 20–40 min after beginning to imbibe; in the UK it is not uncommon to hear "at the end of the first pint of beer". Further, glyceryl trinitrate, a powerful vasodilator, also precipitates attacks. Equally significant is the fact that vasoconstrictors, such as ergotamine in the past and triptans now, are highly effective in stopping cluster headache even when they have started; 100% oxygen also vasoconstricts, helping 60% in 5–10 min. It is also very interesting that patients keep the bedroom cool, and that many, when an attack has begun, go to the bedroom window, which they open, and repeatedly take deep breaths; we know that lowering pCO_2 provokes vasoconstriction.

Nevertheless, ignorance about cluster headache aetiology should not prevent advising highly effective therapy. Verapamil as a prophylactic during an episodic cluster spell is very effective, although not equally beneficial in the chronic variety where often an additional drug is required. How verapamil works is not clear.

Triptans, by injection or nasal spray, are excellent abortive drugs. 100% oxygen inhalation, given through a firm plastic mask with the holes covered with strapping is also effective in aborting attacks. Here, the explanation that vasoconstriction helps, makes good sense.

Conclusions

Diagnosis is only the first step – putting the condition into a category. Next we need to feel with patients to uncover each individual's need, from their different symptoms and their responses to their experiences, in order to advise and prescribe appropriate therapies. For ourselves we should look more deeply into the sites of origin as well as the

pathogenesis of these conditions. The following provisional conclusions are drawn, although I anticipate contradictions.

Migraine is an intracranial, sequential disturbance arising in regional neurons of the cerebral cortex giving rise to its full picture: the first two stages are prodromes followed by the aura. Pain is the third stage, arising from the meninges, followed by stages four and five recovery and postdromes. Many subjects have only part of this picture.

Tension-type headache is an extracranial muscle disturbance most frequently arising from the neck or masticatory apparatus. The pain originates not in muscle fibres but from their fascial coverings, from tendons or their insertion into periosteum, or from relevant joints.

Psychogenic headache is not a pain but a pressure and may be transmitted through the trigeminal and cervical 2–3 nerves by the equivalent of the spinal dorsal column.

Cluster headache is of unknown mechanism but fortunately we have highly effective prophylactic and abortive therapy.

By understanding what patients feel and by trying to look more deeply, we may be able to discard pseudolabels and keep thinking and questioning, as well as be adventurous in producing new ideas to maintain our interest in headaches, and produce ideas to advance our understanding and knowledge.

References

1. Richter IA (ed.) (1998) The notebooks of Leonardo da Vinci. Oxford University Press, Oxford, p 4
2. Blau JN (1990) Common headaches: type, duration, frequency and implications. Headache 30:701–704
3. Head H (1926) Aphasia and kindred disorders, Vol 1. Cambridge University Press, Cambridge, pp 30–31
4. – (2004) The international classification of headache disorders, 2nd edn. Headache Classification Subcommittee of the International Headache Society. Cephalalgia 24[Suppl 1]:9–160
5. Wolff HG (1963) Headache and head pain, 2nd Edn. Oxford University Press, New York, pp 53–95, 269 & 322
6. Airy H (1870) On a distinct form of hemianopsia. Phil Trans R Soc Lond 160:247–264
7. Gowers WR (1895) Subjective visual sensation, The Bowman Lecture. Trans Ophthal Soc UK 15:1–39
8. Lashley KS (1941) Patterns of cerebral integration indicated by the scotomas of migraine. Arch Neurol Psychiatry 46:331–339
9. Blau JN (1991) Migraine postdromes: symptoms after attacks. Cephalalgia 11:229–231
10. Moffett AM, Swash M, Scott DF (1974) Effect of chocolate in migraine: a double blind study. J Neurol Neurosurg Psychiatry 37:445–448
11. Moffett AM, Swash M, Scott DF (1972) Effect of tyramine in migraine: a double blind study. J Neurol Neurosurg Psychiatry 35:496–499
12. Penfield W, Jasper H (1954) Epilepsy and the functional anatomy of the human brain. Churchill, London, pp 110–111 & 140–146
13. Pickering GW, Hess W (1933) Observations on the mechanism of headache induced by histamine. Clin Sci 1:77–101
14. Pickering GW (1939) Experimental observations in headache. Br Med J 1:907–911
15. Blau JN, Dexter SL (1981) The site of pain origin during migraine attacks. Cephalalgia 1:143–147
16. Penfield W, McNaughten F (1940) Dural headache and innervation of the dura mater. Arch Neurol Psychiatry 44:43–75
17. Alvarez WC (1960) The migrainous scotoma as studied in 618 persons. Am J Ophthalmol 49:489–504
18. Blau JN, Thavapalan M (1988) Preventing migraine: a study of precipitating factors. Headache 28:481–483
19. Sjaastad O, Saunte C, Hovdahl H, Breivik H, Gronbaeck E (1983) 'Cervicogenic' headache. An hypothesis. Cephalalgia 3:249–256
20. Kerr R, Grahame R (2003) Hypermobility syndrome. Butterworth Heinemann, Edinburgh, pp 1–14
21. Blau JN (2004) Pony-tail headache: a pure extracranial headache. Headache 44:411–413
22. Blau JN (1993) Behaviour during a cluster headache. Lancet 342:723–725
23. Blau JN, Engel HO (1999) A new cluster headache precipitant: increased body heat. Lancet 354:1001–1002