PAPER

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How do I diagnose headache?

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ABSTRACT The lifetime risk of headache in the population is greater than 90% in European populations. Headache (although often mild and self-limiting) thus represents the most common neurological complaint presenting to primary care physicians as well as neurologists. The ability to recognise and manage headache is necessary for all physicians. The second *International Classification of Headache Disorders* was published in 2004;[6] although useful as a framework for research, this classification needs simplification for everyday practice. In general, most patients will have a primary (or idiopathic) headache syndrome (e.g. migraine, tension type, or cluster headache), but many patients (and doctors) are concerned about missing a sinister secondary headache.

KEYWORDS Chronic daily headache, migraine, trigeminal autonomic cephalagia.

LIST OF ABBREVIATIONS Chronic daily headache (CDH), chronic paroxysmal hemicrania (CPH), cluster headache (CH), computed tomography (CT), erythrocyte sedimentation rate (ESR), episodic paroxysmal hemicrania (EPH), hemicrania continua (HC), idiopathic intracranial hypertension (IIH), International Headache Society (IHS), magnetic resonance imaging (MRI), magnetic resonance venography (MRV), medication over-use headache (MOH), migraine with aura (MA), migraine without aura (MO), new daily persistent headache (NDPH), paroxysmal hemicrania (PH), short lasting unilateral neuralgiform headache with conjunctival injection and tearing (SUNCT), spontaneous intracranial hypotension (SIH), tension type headache (TTH), transient ischaemic attack (TIA), trigeminal autonomic cephalalgia (TAC), trigeminal neuralgia (TN)

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INTRODUCTION

Successful headache diagnosis needs recognition of the most likely diagnostic possibilities using epidemiological and phenotypic knowledge of symptoms. Like many neurological disorders, accurate history taking is fundamental as the majority of patients will not have signs. The need for further investigation is determined by 'red flag' headache symptoms, or symptoms that do not correspond to a recognised primary headache pattern.

In this brief overview I will address the important clinical aspects of headache diagnosis focusing on those most commonly encountered in clinical practice, as well as highlighting some of the rarer, but important, primary headaches that have specific treatment implications. An extensive review of the pathophysiology and management of headache is beyond the scope of this review, but readers are referred to other sources.[9]

HEADACHE EPIDEMIOLOGY

The lifetime prevalence of TTH in the population ranges between 50% and 80%. Migraine affects approximately 6% of children (of both sexes) and adult males, but rising after puberty to 18% in women. Migraine without aura is most common (80%) compared with MA, with one-third of migraineurs experiencing attacks with aura in their lifetime. Migraine onset is most common at puberty but has its highest prevalence in the fourth decade. In comparison, CH has a prevalence of approximately 1 in 1,000 (0·1%), with men more affected than women, and symptom onset usually between 20 and 40 years of age.

In comparison, approximately 3–5% of the population suffer CDH, but represent approximately 10–30% of consultations in secondary care. Chronic daily headache is not a diagnosis but a description of frequent headache (i.e. headache on more than 15 days per month for three months or more) that requires diagnostic clarification to allow appropriate optimal treatment rather than accepting the 'neuromythology' that all CDH is chronic TTH.

Individuals most commonly seek medical consultation for their headache when symptoms are new; become increasingly severe, frequent, or persistent; begin to impair normal daily or work-related activities; or become associated with systemic and/or additional neurological symptoms. Headache sufferers also desire an explanation as well as treatment. In primary and secondary care,

TABLE 1 SNOOP T red flags for secondary headache. Adapted from Silberstein SD *et al.*[10]

FLAG	Description/example
Systemic symptoms or Secondary risk factors	Fever, weight loss or known cancer, HIV, immunosuppression, or thrombotic risks.
Neurological symptoms or abnormal signs	Confusion, impaired alertness/ drowsy, or persistent focal signs (lasting more than 1 hour).
Onset	'First and worst headache', sudden or abrupt from sleep, or progressively worsening.
Older	New onset and progressive, e.g. after 50 years of age for giant cell arteritis.
Previous headache history	First headache or fundamentally different (i.e. significant change in features, frequency, or severity).
Triggered headache	By Valsalva activity, exertion, or sexual intercourse.

migraine is the most commonly encountered headache disorder but remains under-diagnosed and thus potentially under treated.

HISTORY TAKING IN HEADACHE DISORDERS

A careful and comprehensive history is the key to accurate headache diagnosis. When faced with a person complaining of headache in the clinic, the first question to answer is whether they have a primary or secondary syndrome.

Important 'red flags' that highlight the need to consider investigation for a secondary headache are summarised by the mnemonic 'SNOOP T' (see Table 1), and a detailed review is available elsewhere.[10]

As well as considering the above red flags, the following aspects of the headache history need clarification for accurate diagnosis:

- *Age of onset* of initial headache symptoms and clinical course of symptoms over time (e.g. headache beginning in adolescence or teenage years such as migraine; new onset headache at over 50 years of age, especially if progressive, may indicate the need to exclude secondary causes such as temporal arteritis, intracranial tumours and, less commonly, cerebrospinal fluid pressure or chronic meningitis).
- *Periodicity* Intermittent, e.g. migraine, TTH; daily, e.g. CH and migraine +/- MOH.
- *Duration of headache attacks* Short-lived (seconds/minutes to less than four hours) or prolonged (four hours to days).
- *Diurnal variation* Day and night, e.g. migraine and CH, or purely nocturnal, e.g. hypnic headache; worse on waking, e.g. raised intracranial pressure; headache-free on waking but worsening through the day, e.g. low intracranial pressure.
- *Frequency of the headache* More than or less than 15 days per month.
- *Duration of headache symptoms* Individuals presenting with new onset headache symptoms potentially need closer assessment (NDPH).
- *Headache onset* Sudden vs insidious, tempo and time course, e.g. progressive worsening vs bouts lasting days to weeks/months.
- *Additional symptoms* Preceding (premonitory or aura symptoms) or associated non-headache symptomatology, e.g. cranial autonomic symptoms typically in CH, TAC, and, less commonly, migraine (i.e. ptosis, miosis, eye lacrimation, conjunctival injection, nasal blockage, rhinorrhea, and facial oedema).
- *Triggering factors* Valsalva activities (e.g. cough, sneeze, lifting) suggest possible headache related to change in intracranial pressure or structural intracranial posterior fossa pathology.

TABLE 2 Diagnostic criteria for migraine in adults with or without aura. (Adapted from *The International Classification of Headache Disorders*[6])

Recurrent intermittent attacks.

Headache lasting 4–72 hours without treatment.

At least **two** symptoms of:

- Throbbing/pulsating;
- Unilateral;
- Moderate or severe;
- Worsened by movement/avoids routine physical activity.

And either:

- Nausea +/or vomiting;
  or
- Photophobia or phonophobia.

COMMON EPISODIC AND DAILY PRIMARY HEADACHE DISORDERS

Most patients will have a primary, episodic headache disorder such as migraine, CH, or TTH. Recent epidemiological data suggest 90% of headache presentations to primary care are migraine.[11] Accurate recognition of migraine is thus imperative to plan acute and/or preventative treatment dependent on headache impact.

MIGRAINE

Validated diagnostic criteria for episodic migraine are highlighted in Table 2. It is important to realise that headache can be bifrontal or holocranial, rather than unilateral, in up to 40% of sufferers. Equally, individuals often have difficulty describing pain, and non-pulsatile 'severe pressure' is commonly reported by 50%.

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Whilst the individual with a 'full house' of symptoms is easy to diagnose, it is important to realise that individuals may not report photophobia, phonophobia, nausea, or movement aggravation of headache on direct enquiry, but more focused questioning about behaviour during headache may identify such symptoms. For example, a patient may deny photophobia or phonophobia on direct questioning, only to then describe the desire to lie down in a quiet, dark place! Asking what patients do when they have a headache should elicit this sort of pattern.

Up to 70% of patients may experience prodromal symptoms within the preceding 24 hours of an attack, such as increased fatigue, impaired concentration, and neck stiffness. These symptoms may reflect activation of dopaminergic, brainstem–hypothalamic, and trigeminocervical neuronal pathways.

Aura and headache

Aura occurs in up to 30% of migraine sufferers, but rarely precedes other primary headaches, e.g. CH. Aura is characterised by gradual onset (over minutes) of focal neurological symptoms, and is thought to be due to cortical spreading depression. Aura usually lasts between 20 and 60 minutes before spontaneous resolution. Visual aura is most common, accounting for more than 90%; but other symptoms such as evolving sensory and/or motor symptoms, transient dysphasia, vertigo, or confusion may occur. Visual aura is usually binocular, and usually consists of 'positive' evolving symptoms (e.g. flashes (photopsia), shimmering, or zig-zags (fortification spectra) that may be crescentic and increase or decrease in size). There may be associated negative symptoms such as loss of vision (scotomota). Aura without headache is not uncommon especially in the fourth and fifth decades and must be distinguished from TIAs. Transient ischaemic attacks are usually shorter than aura, do not evolve, and are 'negative' rather than 'positive'.

TENSION TYPE HEADACHE

In contrast to migraine, TTH is defined as a featureless, non-throbbing, generalised headache. The 'featureless' diagnostic criteria and phenotype for TTH is based on the absence of symptoms (i.e. photophonophobia, gastrointestinal symptoms, attacks that are worsened by activity (or behaviours during attacks that suggests these symptoms)) rather than discrete positive features of the attacks themselves. Headaches often last hours, less frequently, days, and usually remit with simple over-the-counter analgesics. Most headache experts feel that TTH should not be disabling and of only mild or moderate intensity. The suspicion is that many patients diagnosed with TTH in fact have unrecognised migraine. The SPECTRUM study[7] supported such a hypothesis and proved useful when considering individuals who were initially thought to have TTH. This study reported that disabling TTH is difficult to find in the outpatient clinic. Moreover, when an individual was diagnosed in the study with disabling TTH, subsequent longitudinal diary review identified that they actually fulfilled the diagnostic criteria for migraine. Equally, patients with severe attacks of migraine were found to suffer mild featureless headaches that sometimes evolved into full-blown migraine, and these featureless headaches often promptly responded to migraine specific acute treatments. Although not a life-threatening disorder, a misdiagnosis of TTH potentially commits such patients to limited treatment options, and excludes them from effective migraine treatments.

TABLE 3 Diagnostic criteria for cluster headache (Adapted from *The International Classification of Headache Disorders*[6])

Severe unilateral, periorbital, supraorbital and/or temporal pain lasting 15–180 minutes if untreated.

Attack frequency up to eight per day.

With at least **one** of the following ipsilateral autonomic symptoms:

- conjunctival injection +/or lacrimation;
- nasal congestion +/or rhinorrhoea;
- eyelid oedema;
- forehead and facial sweating;
- miosis +/or ptosis;
- a sense of restlessness or agitation during headache.

CLUSTER HEADACHE

Cluster headache is one of the most severe headache disorders known and is sometimes nicknamed 'suicide headache' because of this severity. Cluster headache sufferers experience 'cluster bouts' lasting weeks to months before remission (typically 4–12 weeks). Cluster bouts can occur up to twice a year, often at the same time each year. The actual attacks can occur multiple times per day and often at night (within 1–2 hours of falling sleep). The diagnostic criteria are summarised in Table 3. Important differentiating characteristics from migraine are:

- Shorter attack duration, typically 45 minutes to one hour (definitely less than four hours);
- Daily nature of headaches often with multiple attacks daily (compared with migraine with attacks occurring episodically but lasting days);
- Agitated behaviour of sufferers during the attack (in contrast to a migraineur's preference to avoid movement);
- Prominence of ipsilateral cranial autonomic symptoms with CH (which can occur with migraine, but more rarely).

Most CH sufferers (up to 90%) experience episodic cluster headache, but a minority have no remission (chronic CH). Recognition of CH and differentiation

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TABLE 4 Differential diagnosis of chronic daily headache (headache occurring on more than 15 days per month).

Primary headaches		**Secondary headaches**
Headache duration > 4 hours	**Headache duration < 4 hours**	
Chronic migraine (+/- MOH)	(Chronic) CH	Inflammatory systemic disease, e.g. giant cell arteritis, vasculitis, sarcoidosis, Behçet's disease.
Chronic TTH (+/- MOH)	CPH	Post-traumatic, e.g. trauma, neurosurgical procedures, post-haemorrhage or meningitis.
HC	SUNCT	Due to chronic dural irritation, e.g. chronic meningitis.
NDPH	Hypnic headache	Due to high or low pressure syndromes.

from migraine and paroxysmal hemicrania (see later) is important as this will result in differing treatment.

CHRONIC DAILY HEADACHES

Chronic daily headache is not a diagnosis but implies that headache occurs frequently (daily or near daily; recognised international classification criteria suggest more than 15 days per month) for at least three months. There are a number of primary and secondary causes of CDH and an effective clinical approach to differential diagnosis is summarised in Table 4.

Chronic daily headache usually develops in one of two ways. The majority initially experience intermittent headache symptoms (suggesting an underlying primary headache disorder such as migraine or TTH) which insidiously increase in frequency to become daily and persistent. Less commonly, CDH can develop *de novo* without any preceding history of increasing headaches. In this situation secondary headaches must be considered.

Medication over-use headache

It is important to consider MOH as a possible cause or contributory factor in CDH. Daily use of analgesic medication may 'transform' or 'cause' chronification of a previously episodic primary headache disorder, although the biological mechanism is still unclear. Patients should be withdrawn from analgesia, and it may take several weeks before any benefit is seen. However, many patients will still have CDH despite analgesic cessation, suggesting an alternative pathophysiological mechanism.

Any form of analgesic may cause MOH if taken more frequently than 2–3 times per week for more than a few weeks. Women are more susceptible than men. Often there is a history of episodic headache (migraine or, less commonly, TTH) in earlier life that has become gradually and increasingly troublesome, mirrored by insidious increased analgesic ingestion. Triptans, when taken excessively, produce daily or more frequent 'migraine-like' headache, whereas over-the-counter and prescribed simple analgesics (paracetamol, aspirin, etc.) and combination analgesics (especially those containing codeine or caffeine-based compounds) produce a background 'featureless' TTH.

The importance of recognising MOH in CDH sufferers cannot be overestimated. It forms the cornerstone of effective management of CDH in two-thirds of patients Equally, therapeutic management strategies often fail in the context of ongoing, unrecognised MOH thus wasting resources.

Chronic (transformed) migraine

Chronic migraine describes individuals who experience symptoms of migraine and headache on more than 15 days per month for more than three months in the absence of analgesic medication overuse. Population studies from Spain, the USA, and Denmark suggest that it develops insidiously in up to 2% of sufferers of episodic migraine. Often there is initial associated MOH, but, once this has been recognised and dealt with there, is still a significant proportion of individuals with chronic migraine. A useful clinical approach to diagnosing chronic migraine has been described by Goadsby *et al*.[4]

In brief, individuals initially suffer intermittent, often infrequent, attacks of migraine. They often recall mild, but relatively frequent, headaches in the past for many years. However, over an individually variable period of time (often months to years) there is an insidious increase in headache frequency associated with the additional associated migrainous symptoms with the more severe episodes, i.e. photophonophobia, nausea, and mechanosensitivity (see above for migraine). They develop fewer headache-free days and often significant headache-related impairment. Subsequent enquiry to identify a family history of migraine or disabling headache suggestive of migraine adds indirect support to their suspected diagnosis of chronic migraine by implying an underlying genetic susceptibility to headache. The identification of known migraine triggers (e.g. change in sleep pattern, change in food pattern, female hormonal

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change, or change in emotional state or weather pattern) that induce CDH exacerbations also lends support to the clinical diagnosis of chronic migraine. In some individuals, a discrete event, e.g. neck or head trauma, or a significant life event can be identified that may have initiated chronification of previously infrequent migrainous symptoms.

NEW DAILY PERSISTENT HEADACHE

This describes the *de novo* development of new, daily or near-daily headache symptoms (symptom onset over fewer than three days), in someone previously free of headache, that does not resolve within three months. Although accepted as a diagnostic entity in the recently published IHS classification, it is probably better to use the concept that such individuals require initial investigation to exclude a secondary cause of headache before attributing symptoms to a benign primary cause. Investigations should include relevant blood tests (ESR if over 50 years of age) and brain imaging. The imaging modality of choice has not been prospectively studied to determine whether CT or MRI is diagnostically more clinically relevant. However, as low pressure (intracranial hypotension) and high pressure headache syndromes (cerebral venous sinus thrombosis, IIH) are well recognised causes of NDPH, Gd-DTPA-enhanced MRI and/or MRV are appropriate investigations to exclude the respective secondary causes that will guide treatment. In addition, lumbar puncture can be diagnostically useful when pressure syndromes are suspected and imaging is normal.

Idiopathic intracranial hypertension is characterised by daily headache with features of raised intracranial pressure and is more common in obese individuals. Headache in IIH is typically worse on awakening from sleep, or in the early morning, and is also worsened by Valsalva type activity, i.e. coughing, sneezing, straining. There is sometimes diplopia (due to partial abducens (VI) nerve palsy), and usually the only clinical sign is papilloedema. Brain imaging, including the cerebral venous sinuses and CSF analysis, must be normal except for a raised CSF opening pressure greater than 200 mm (some argue greater than 250 mm in the obese) to confirm the diagnosis.

By contrast, SIH is a rarer secondary daily headache caused by loss of CSF volume and thus lowering of CSF pressure from a dural leak. The classical early symptom is postural headache. Characteristically, the headache is minimal or absent whilst lying or on waking from sleep but develops within minutes (sometimes much longer) of adopting an erect posture. Individuals typically describe throbbing headache that can be generalised, unilateral but most commonly occipital, with associated nausea and sometimes prominent neck pain. Headache and associated symptoms are relived by lying down within 15 minutes in most cases. If unrecognised, the postural headache may be lost and becomes a milder CDH sometimes difficult to distinguish from other causes. The presence of prominent otological symptoms with headache, i.e. new whooshing tinnitus or new deafness, are other useful symptoms that are more commonly associated with headaches caused by abnormalities of CSF pressure. Yet again, the recognition that the cause of an individuals' NDPH can be an abnormality of CSF pressure determines different treatment decisions, weight loss and visual function monitoring in IIH and epidural blood patching for SIH.

For a detailed review of evaluating NDPH, see Goadsby *et al*.[4] Often, no secondary cause is found and the headache diagnosis is determined by the symptoms of the individual, i.e. primary NDPH, migrainous, or TTH-like.

HEMICRANIA CONTINUA

Hemicrania continua is a rare but important cause of CDH as it responds completely to indometacin therapy. The prevalence is unknown, but it affects women more than men, often in middle life, and may be misdiagnosed as chronic migraine, owing to a lack of awareness of the condition.

Hemicrania continua is characterised by a side-locked continuous unilateral daily, mild to moderate, dull or aching headache with exacerbations. These exacerbations are associated with ipsilateral autonomic symptoms similar to CH up to 75% of the time, especially lacrimation and conjunctival injection. Some patients may also have photophobia, phonophobia, and nausea during exacerbations. Confirmation of HC requires a complete response to indometacin therapy.

Hemicrania continua, although also having cranial autonomic symptoms (described above), differs from the other TACs (CH, episodic and chronic paroxysmal hemicrania, and SUNCT) as the paroxysmal exacerbations of side-locked pain are not separated by pain-free periods.

UNUSUAL BUT IMPORTANT PRIMARY HEADACHES

Trigeminal autonomic cephalalgias

Cluster headache, the most common form of TAC, has been described previously. Other primary headache syndromes within this group include paroxysmal hemicrania and SUNCT syndrome.

Trigeminal autonomic cephalalgias are characterised by frequent, severe, brief, unilateral headaches that have prominent ipsilateral cranial autonomic symptoms. Whilst it is the duration and frequency of attacks that largely differentiates these conditions from each other and from migraine, TAC sufferers also sometimes suffer photophobia.

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Paroxysmal hemicrania

There is both an episodic and chronic daily form of PH. Paroxysmal hemicrania does not have a nocturnal or seasonal pattern. Paroxysmal hemicrania is rarer than CH (some studies estimate 1 in 50,000). In contrast to CH, it predominantly affects females more than males, most often in their 30s. Attack duration is shorter (minutes, up to 30 minutes) than CH, and attack frequency ranges from once, to up to 40 per day. Paroxysmal hemicrania attacks are characterised by severe, abrupt-onset pain in a periorbital and temporal distribution. There is usually associated prominent ipsilateral lacrimation, conjunctival injection, nasal congestion, or rhinorrhoea, although only one autonomic symptom may be present. Triggering of attacks by rotational head movement may occur (up to 10%) but most attacks occur spontaneously. An absolute response to indometacin confirms the diagnosis. Individuals with this disorder should all have brain MR imaging as a relatively high number of symptomatic cases have been described.

SUNCT syndrome

This is an exceptionally rare headache disorder first described in 1978. It resembles TN in severity and duration, but has the following salient and clinically notable differences:

- It has prominent associated ipsilateral cranial autonomic features especially lacrimation and conjunctival injection (usually absent in TN);
- Pain is maximal in the ophthalmic division of the trigeminal nerve (whereas TN most commonly affects the maxillary and/or mandibular divisions of the trigeminal nerve);
- Like TN, attacks occur spontaneously and can be triggered (by touch, speech, chewing, the cold, washing, cleaning teeth, etc.) but SUNCT attacks can be recurrently triggered immediately after pain ceases, whereas TN has a latent period where no further attacks can be elicited by the usual trigger.

Hypnic headache

This is a rare condition sometimes also called 'alarm clock' headache due to the identical time of night at which it often strikes. It characteristically affects women more than men, occurs more commonly in adult life after the age of 50, and is probably under-diagnosed. Headache attacks occur exclusively during sleep and wake the individual. Sufferers complain of moderate or severe dull or sometimes throbbing headache, either unilateral (~40%) or bilateral (~66%). Attacks occur at least once per night, lasting more than 15 minutes and up to 1–2 hours (average one hour) in duration before resolution. Multiple attacks per night are common and, except for nausea in 20%, there are usually no other symptoms. The different site, lack of daytime attacks, and absence of prominent autonomic symptoms differentiate the headache disorder from CH. In general terms, brain imaging is recommended as increased intracranial pressure disorders may manifest as 'nocturnal waking headache'.

HEADACHE MYTHOLOGY AND OVER-DIAGNOSED HEADACHES

Whilst the general population believes refractive error and hypertension cause headache, population-based case control studies do not support such assumptions. A visit to the optician is best used to exclude the possibility of raised intra-ocular pressure and prompt referral to an ophthalmologist to exclude acute glaucoma as an ophthalmic cause of localised headache. Headache, and especially paroxysmal intermittent severe headaches with associated raised blood pressure, only becomes relevant when malignant hypertension is present. In such situations, enquiry about associated symptoms suggesting persistent or intermittent hyperadrenalism, i.e. panic attacks, hyperhidrosis, palpitations, is necessary in addition to urinary screening for adrenaline metabolites to exclude the very rare clinical syndrome associated with phaoechromocytoma. Similarly, 'sinus headache' is often unrecognised migraine, particularly if recurrent and episodic. Acute rhinosinusitis producing facial pain should be associated with green nasal discharge and relevant ENT symptoms reflecting acute sinus infection rather than simply facial pain. In general, incidental 'chronic sinus inflammation' is not considered to be a case of acute headache.

KEYPOINTS

- Primary headache disorders are diagnosed according to duration and frequency of attacks, and thus depend on accurate history taking, with headache diaries if necessary.
- Correct headache diagnosis is the start of the headache treatment pathway.
- Migraine (not TTH) is the most common episodic headache disorder that prompts consultation to a physician in secondary care, and is often misdiagnosed.
- Tension type headache is entirely featureless (based on absence of symptoms rather than features of the attacks).
- Chronic daily headache is not a diagnosis and a cause must be identified. Associated analgesic medication overuse must always be considered before attributing it to chronic TTH.
- 'SNOOP T' red flags should be used to determine the need for investigation to exclude serious secondary headache disorders.
- The presence of prominent cranial autonomic symptoms highlights a specific subgroup of headache disorders (TACs) that require specific treatment.

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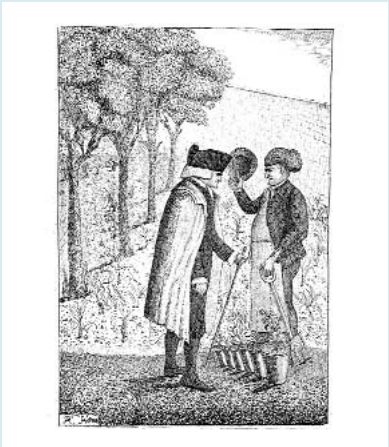

PAST PRESIDENTS

John Hope (1725–1786)

Strange as it may seem to today's doctors, until the latter half of the twentieth century, botany was a compulsory subject in the Edinburgh medical curriculum, at one time taught as part of *Materia Medica,* then, as a result of the work and influence of John Hope, in its own right. Later to become a President of the College, his fame rests on his botanical knowledge and research.

With his grandfather, Lord Hope, a senator of the College of Justice (a High Court judge in Scotland) and his father an Edinburgh surgeon, he might have been expected to train in Edinburgh, but instead he took his MD in Glasgow and did postgraduate work in Paris. It was there that he first heard of the botanist Linnaeus of Uppsala, fuelling his interest in botany. He returned to Edinburgh in 1750, became a physician at the Royal Infirmary and, in 1762, became a Fellow of the College. By this time he had already been appointed Professor of Botany at Edinburgh University, King's Botanist for Scotland, and Superintendent of the Physic Garden, using the now established Linnaean system of classification.

This garden had been started by two of the founding fathers of our College – Sir Andrew Balfour and Sir Robert Sibbald. The first site was near Holyrood House, the Edinburgh residence of the monarch. From there, it was moved to unsuitable, swampy ground where Waverley Station stands today. In 1776, it was moved again, this time to Gayfield Square, off Leith Walk, a site far too small but better than its previous sites. In 1820, it was incorporated into the Royal Botanic Garden being developed at Inverleith, and today regarded as one of the world's greatest such gardens. The miniature Physic Garden built in a courtyard of our College in 1996 is tended by the gardeners of the Royal Botanic Garden.

One of Hope's students, William Roxburgh (1751–1815) FRCP Edin, founded the Indian Botanic Garden in Calcutta. No fewer than eight species of rhododendron introduced into Britain are named after Edinburgh doctors, Roxburgh naming a tree genus *hopea* after his teacher.

Hope was President of the College 1784–1786.

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