IN-DEPTH REVIEW

Vestibular assessment: a practical approach

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Abstract
This paper presents an evidence-based introduction to assessing and managing the dizzy patient. It is based upon a literature review highlighting current best practice and the clinical experience of the author. Dizziness is common, often chronic and largely untreated, resulting in great personal and economic loss. Serious pathological causes of dizziness may be quickly and effectively screened by a simple ocular motor examination, differentiating these from peripheral vestibular lesions. A triage, differentiating dizziness into either acute onset, intermittent episodes or continuous symptoms is proposed, along with management strategies for the commonest causes of dizziness. Vestibular suppressant medication should be restricted to the acute phase of a vestibular episode. Vestibular rehabilitation therapy is the most effective treatment for continuous or chronic dizziness.

Key words
Dizziness; vestibular assessment; vestibular rehabilitation.

Introduction

There can be few physicians so dedicated to their art that they do not experience a slight decline in spirit when they learn that their patient's complaint is dizziness.


Dizziness is one of the most common problems among patients in primary health care [1]. Neuhauser et al. [2] estimate the prevalence of dizziness within the general population to be over 5%, currently >3 million people in the UK. They report that 41% of people complaining of dizziness take time off work and that 80% limit their usual daily activities due to the dizziness. The incidence of dizziness increases with age, ranging from 20 to 30% in the 50–65 age range [3], to over 40% in the over 70s. In the over 75s, dizziness is the most common reason to seek a general practice consultation [4]. Despite this, treatment is haphazard and largely ineffective. Yardley et al. [5] reported that one-third of patients with dizziness remained symptomatic after 5 years and only one in four ever received treatment. Studies from a number of different countries all conclude that dizziness is common, often becomes chronic and is largely untreated.

This article will not attempt to undertake a systematic review of the literature but rather aims to be a practical guide on how to manage the dizzy patient.

What is dizziness?

Dizziness is often used as a general term to describe many sensations, such as light-headedness, imbalance, vertigo and presyncope [6]. Specifically, it may refer to a feeling of unsteadiness or mild intoxication as if the ground is moving. True vertigo refers to the illusion of movement, usually rotation, of the self or of the environment around the self [7]. In practice, all of these symptoms can be thought of as symptoms of a balance system disorder.

The balance system is a complex multicomponent system, comprising peripheral and central neurological pathways, the vestibular end organ (labyrinth), vision, the musculoskeletal system, perception, cognition and psychological factors such as emotion and belief. The cerebellum plays a crucial role in the integration of all these components.

In order to balance, the brain needs sensory information comprising vision, somatosensory feedback from muscles, joints and the skin, and vestibular inputs from the labyrinth of the inner ears. The incoming sensory information is processed in the brain stem and cerebellum, organized and integrated with cognitive and psychological inputs, such as memory, belief, expectation and emotions. If there is an absence of information, for example in the case of an elderly diabetic patient, with poor vision and a vestibular loss, then the result is imbalance or unsteadiness. If there is a conflict or mismatch in the information, then the result is dizziness. This will be familiar to passengers waiting in a stationary train at

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a station, watching a moving train through the train window. The perception of movement results from the mismatch between visual inputs (indicating movement) and somatosensory/vestibular inputs (indicating a stationary position). If there is a significant difference in the activity levels of the two sides of the balance system, then the result will be a rotary vertigo. This is a typical symptom of children’s playgrounds. Riding on a revolving roundabout will stimulate vestibular signals on one side of the system. When the child jumps off the roundabout there is a difference between the activity levels of the two sides of the balance system that takes a few seconds to subside. During this time, a spinning feeling results due to the conflict with the visual/propioreceptive inputs.

In order to maintain balance, the sensory integration and organization within the brain triggers vestibular motor outputs. These involve stabilization of gaze, maintenance of head position with respect to midline, tonic spinal cord reflexes enabling us to hold ourselves upright against gravity, phasic balance righting reactions in response to sudden balance disturbances, and a perception of where we are in space projected up to the thalamus and cortex. The cerebellum is the site for anticipatory or feed-forward balance motor outputs, for example in necessary postural adjustments as we walk from flat ground onto a slope.

The demands upon the balance system are highly dependent upon the task in question. Sitting still in a chair and walking down a crowded high street require different levels of sensory information, sensory organization and motor outputs. Additionally, the environment or context in which the task takes place also affects the outcomes. Patients typically find that walking at home is significantly easier than walking outdoors. History and expectations also affect the outcome. If a patient previously experienced a dizzy attack in a supermarket, the next time they return to a supermarket they are already ‘primed’ to suffer a further attack. These processes are summarized in Figure 1.

The vestibular (balance) system is a remarkably complex multifactorial system which is usually taken for granted and is only really noticed when something goes wrong.

**Practical diagnosis**

Dizziness and vertigo may be symptoms of central nervous system pathology, so it is important that patients are carefully screened to exclude serious pathology such as stroke, tumours or multiple sclerosis. Cases of central nervous system pathology rarely present with dizziness as the only complaint [8,9]; other signs and symptoms of brain stem or cerebellar dysfunction will usually be evident. If the patient reports symptoms of dysarthria, dysphagia, double vision, loss of consciousness, sudden onset severe headache, muscle weakness or anaesthesia/paraesthesia associated with the dizziness, then central pathology should be considered. If there is a new gait abnormality, cerebellar signs or cranial nerve dysfunction then referral to a neurologist is indicated. Any unilateral loss of hearing should be referred on to an ear, nose and throat (ENT) specialist for further investigation.

When assessing the dizzy patient it is important to consider the characteristics of the symptoms. Broadly, dizziness may be categorized as follows:

1. **acute initial onset,**
2. **intermittent/episodic attacks,** or
3. **chronic/continuous symptoms [10,11].**

**Acute onset dizziness**

If the patient presents with an initial acute attack of dizziness, the most important differential diagnosis is between peripheral vestibular dysfunction and vertebrobasilar ischaemia of the brain stem or cerebellum [8,11]. The examination of eye movements is a more sensitive method than magnetic resonance imaging for the diagnosis of acute vestibular syndromes and for the differentiation of peripheral from central lesions [12]. Kattah et al. [12] outline a simple three-step bedside clinical oculomotor examination that will differentiate between unilateral vestibular lesion and brain stem stroke. An examination showing a normal head impulse test, direction-changing nystagmus in eccentric gaze and vertical skew deviation of the eyes is sensitive and specific to brain stem stroke [13]. An acute peripheral vestibular disorder such as labyrinthitis or vestibular neuronitis will more likely show a positive head impulse test, unidirectional gaze-evoked nystagmus and no vertical skew deviation of the eyes. It is worth noting that some brain stem ischaemias, such as proximal anterior inferior cerebellar artery infarcts, may mimic peripheral vestibular lesions with positive head impulse tests, but will still have other central signs such as vertical skew deviations or direction-changing

![Figure 1. Sensory inputs and motor outputs of the balance system.](image-url)
gaze-evoked nystagmus [9,14]. Figure 2 illustrates the head impulse test.

Management of vestibular neuronitis

Once the clinician is assured that the problem is peripheral in nature, management is more straightforward. If the dizziness is associated with unilateral hearing loss then an inner ear infection, or labyrinthitis, is likely and referral to an ENT specialist is advised. If, as is more common, no hearing loss is reported, then a peripheral vestibular neuronitis, an inflammation of the eighth cranial (vestibular) nerve, is most likely. The cause is usually viral, the herpes simplex virus being the most likely [15,16]. The condition should be considered as an acute reversible peripheral neuropathy, much like a Bells’ palsy of the seventh facial nerve with which it is often associated. Strupp et al. [16] have shown that early treatment with anti-inflammatory corticosteroids is the most effective medical intervention. Vestibular suppressant medication should be limited to the first few days only, to help reduce nausea and vomiting [17]. The severe symptoms should subside over a few days. The majority of patients will experience a good resolution of symptoms in under 3 months; however, up to one-third may well go on to experience chronic dizziness [5].

Episodic/intermittent dizziness

Patients who present with repeated attacks of dizziness will have symptoms of varying duration, and this is helpful in diagnosis.

The commonest cause of vestibular dysfunction is benign paroxysmal positional vertigo (BPPV) [18]. The condition is characterized by episodes of intense vertigo, lasting <1 min, triggered by position changes such as lying down, rolling over in bed, looking up or bending forward. The mechanism is thought to be due to dislodged deposits of otoconia from the wall of the otolith becoming trapped within one of the semicircular canals of the labyrinth. Head movements in the plane of the affected canal initiate movement of the otoconia within the canal triggering a false signal from the affected side. Although there are different types of BPPV, the majority affect the posterior canal and are readily identified and treated [18].

Intermittent attacks of dizziness lasting up to a few hours are most likely to be due to either Ménière’s disease or migraine-associated vertigo (MAV). The episodes often come in clusters of random, extremely debilitating attacks, but the patient may be symptom-free between episodes. A residual sensitivity to motion may remain in-between attacks. Ménière’s disease is a pathology affecting the whole of the labyrinth, and as such there will be a specific low-frequency hearing loss associated with the dizziness. Patients often complain of tinnitus and a feeling of ‘fullness’ or pressure within the ear. If Ménière’s disease is suspected, then a referral to ENT should be initiated. If there are no reports or signs of hearing loss, then MAV is the most likely cause, being present in over 10% of dizziness patients [19–21].

Management of BPPV

The commonest form of BPPV occurs when mobile otoconia become trapped within the posterior semicircular
canal of the labyrinth. The condition is diagnosed by the symptom and nystagmus response to the Dix–Hallpike positional vertigo test [18], during which the head is moved in the plane of the posterior canal. Free-moving otocoria within the canal will trigger an excitatory response, causing strong vertigo, along with torsional/up-beating paroxysmal nystagmus, easily seen if viewed through Frenzel lenses or video-ocular goggles. The test position involves ipsilateral head rotation of 45°, along with cervical extension. The patient is moved back into a supine position with the head hanging down 20° below the horizontal [18]. The test position is held for up to 1 min. In a positive test, there is normally a slight delay, then strong torsional nystagmus and vertigo which fades away in 5–20 s. A similar response with opposite direction nystagmus is found on returning the patient to the upright position. The test is repeated on the other side. Generally, BPPV is a unilateral condition but the author has occasionally seen bilateral BPPV. Figure 3 illustrates the Dix–Hallpike test.

Treatment is by particle repositioning manoeuvres, whereby the debris is manoeuvred out of the posterior semicircular canal and returned into the otolith. Epley’s manoeuvre is the most common method of treatment. Beginning with the Dix–Hallpike to the positive side, the patients head is moved in 90° steps round towards the contralateral side, resting for 1 min in the Dix–Hallpike position, then 1 min in the contralateral Dix–Hallpike, then 1 min in the side-lying nose-down position, before sitting up slowly. The Epley manoeuvre is summarized in Figure 4.

The success rate of the manoeuvre is extremely high with over 90% of patients achieving resolution of symptoms within one or two treatment sessions [22–24]. Despite this, patients rarely obtain effective treatment early on. A study by Fife and FitzGerald [25] reviewed the path from initial complaint to diagnosis and symptom resolution in a group of ultimately confirmed BPPV suffers. They reported a mean wait time of 92 weeks from initial consultation to successful treatment, and 85% of these patients had immediate symptom resolution at the first treatment session.

**Management of MAV**

MAV is often a diagnosis of exclusion but may be suspected if the patient has a history of migraine [20]. In between attacks, the patient may be symptom-free without signs of a vestibular condition, or present with signs of a residual vestibular imbalance [26]. It is managed much the same as common migraine, with emphasis on managing anxiety and stress, avoidance of dietary or environmental triggers and anti-migraine medication. Vestibular rehabilitation therapy (VRT) is often very helpful [26–30].

**Management of Ménière’s disease**

Hearing is affected in a characteristic low-frequency pattern of hearing loss, therefore patients should be referred to ENT for specialist assessment. During periods of frequent attacks, rehabilitation potential is limited. However, attacks often lead to a gradual build-up of vestibular asymmetry/imbalance. During periods of remission, VRT is very effective in helping the brain to compensate for the peripheral vestibular asymmetry [30,31]. Management should aim at reducing disability, and limiting salt, sugar, caffeine and alcohol intakes as these often trigger attacks due to the diuretic effects. There is evidence to support the use of beta-histine in order to increase blood perfusion to the labyrinth and help central compensation [32].

**Chronic continuous dizziness**

For patients complaining of a continuous, chronic form of dizziness or imbalance, the history is helpful in differentiating between cerebellar ataxias and non-compensated peripheral vestibular dysfunction. Gradual onset with age or a history of alcohol abuse, along with other signs and symptoms of central nervous system and cerebellar involvement should help identify ataxias.

There are a large number of patients who suffer from a peripheral vestibular dysfunction, for example, vestibular neuritis or Ménière’s disease, who unfortunately never fully recover. The central nervous system should compensate for any peripheral loss of function, but this will only occur if the brain receives sufficient feedback after the lesion. In this case, the feedback required is sensory information from movement of the head, neck and eyes. Many vertigo sufferers do not generate this feedback due to the severity of the symptoms provoked, thus are unable to compensate centrally. They remain with sensitivity to motion, positions, environments, visual stimuli and complain of a daily dizziness. Often, testing fails to reveal signs of pathology and this group are vulnerable to being labelled psychosomatic or anxious. This group, more than any other, will benefit from VRT.

![Figure 3](image-url). A left-side Dix–Hallpike test for positional vertigo.
The differential diagnoses for vertigo are summarized in Table 1.

**Vestibular rehabilitation therapy**

The use of vestibular exercises to treat patients with persistent symptoms of positional- and movement-related vertigo has been around for many years. The Cawthorn–Cooksey exercises, developed in the 1940s, consist of a series of head and body movements, aiming to provoke the patient’s dizziness and thus hasten the recovery process. Better understanding of the function and adaptation of the vestibular system, has led to customized VRT, adapted to the specific needs of the individual, becoming the mainstay of treatment [33].

Vestibular rehabilitation is most effective in patients with a unilateral peripheral vestibular loss with incomplete central compensation [34–36]. Even long-standing complaints may be managed successfully with an appropriate VRT programme.

Following acute vestibular dysfunction, for example, vestibular neuronitis, the signs and symptoms are due to the tonic asymmetry within the vestibular nuclei. Acute compensation to the static situation (head still) will occur at the vestibular nuclei through cerebellar inhibition within 24–72 h. Central compensation to dynamic situations enabling accurate vestibular responses to head movements, takes much longer, usually several weeks. This occurs through neuroplastic reorganization of the cerebellar and brain stem pathways and requires vestibular stimulation and thus dizziness provocation. Due to the symptoms, patients are reluctant to stimulate the system enough, fail to ensure adequate central compensation and therefore use alternative strategies in order to balance. Substitution with ocular and cervical proprioceptive sensory feedback is commonplace. This often leads to a dependency upon, and sensitization to, visual sensory stimuli. This may be associated with increased upper cervical muscle tone, with complaints of neck pain and headaches, secondary to the peripheral dysfunction.

Vestibular rehabilitation has demonstrated improvements in both subjective and objective measures, in patients with bilateral vestibular loss [37–39], and with central vestibular disorders [40]. In a recent review of
the literature, Hansson et al. [30] found that VRT can be strongly recommended as treatment for vestibular hypo-function, for multisensory dizziness, for Ménière’s disease and after vestibular surgery. The results of research into vestibular rehabilitation for neurological causes of dizziness, BPPV, dizziness associated with whiplash and migraine-associated dizziness are promising enough for recommending vestibular rehabilitation for these diagnoses. Although the early studies were of small scale and limited applicability, more recent research has focussed upon specific subgroups of patients and randomized large-scale trials. There is now a sufficient body of high-quality, large-scale research to justify VRT for these conditions[41,42].

The main aims of VRT are to improve gaze stability, reduce dizziness, improve postural and gait stability, restore normal movement patterns, return patients to normal function and activity and to increase general conditioning [33]. An experienced vestibular therapist will develop a programme of exercises reflecting the specific needs of the patient. Exercises should be task, context and environment specific in order to ensure effective compensation.

Gaze stability is improved by a process of central vestibular adaptation, through inducing movement of the visual image across the retina, known as retinal slip. This error signal sharpens the vestibular–ocular reflex. Eye and head coordination exercises with stationary, moving or multiple targets, against a variety of backgrounds will facilitate this process [35,36].

Loss of vestibular coordination leads to a motion-provoked vertigo. Movements of the head or body typically provoke symptoms of dizziness. In addition, movements in the environment may also trigger dizziness due to sensitivity to incoming visual sensory information. Habituation exercises reduce the symptoms through repeated exposure to the specific dizziness-provoking movement. Persistent exposure to provoking movements leads to suppression of vertigo and desensitization of the synaptic pathways from the peripheral sensory cells to the vestibular nuclei and brain stem. Typically, three or four movements are chosen to provoke a mild reaction. Symptoms are allowed to settle, and then the process is repeated. Gradually, movements are made more rapidly and in more challenging circumstances.

Postural instability is improved by exercises challenging the whole balance system. Simply put, in order to improve your balance, you need to wobble. By varying the vestibular, visual and somatosensory inputs, emphasis can be placed upon the dysfunctional part of the system. Balance exercises may incorporate standing or walking upon different surfaces, head movement, eye movement, eyes closed, altered lighting, stimulating or moving visual backgrounds, throwing and catching a ball and moving in different environments.

Dizzy patients often develop compensation strategies in order to avoid provoking further symptoms. These may result in altered patterns of movement, both of the head and body. For example, head turning is avoided, leading to significantly increased cervical muscle tone and neck stiffness. Gait patterns become more rigid and wide-based, resembling that of the toddler, with the head and gaze pointing firmly forwards. Abnormal movement patterns lead to aberrant proprioceptive feedback into the cerebellum. This in turn further sensitizes the central vestibular system and increases symptoms of dizziness and imbalance. Therapy must be directed at restoring normal movement patterns, for example, getting the neck moving again through manual therapy and exercise, or gait education. A return to normal movement leads to better proprioceptive feedback and easier balance coordination.

| Table 1. Differential diagnosis of vestibular vertigo |
|---------------------------------------------|------------------|------------------|
| Vertigo characteristics | Possible condition | Presentation |
| Sudden acute onset | Vestibular neuronitis | Initial symptoms last for days |
| Brain stem ischaemic stroke | | Positive head impulse test |
| Brain stem ischaemic stroke | | Negative head impulse test |
| Vertical occular deviation | | Strong direction-reversing gaze-evoked nystagmus |
| Intermittent/episodic | BPPV | Duration of seconds |
| Migraine | | Positional triggers |
| Ménière’s disease | | Positive Dix–Hallpike test |
| Duration of hours | | Duration of hours |
| Associated hearing loss | | Motion and visual sensitivity |
| Continuous/chronic | Cerebellar ataxias | Central nervous system signs |
| Non-compensated vestibular loss | | No central signs |
| | | Motion and visual sensitivity |
The high levels of disability resulting from dizziness, mean that many patients become anxious or depressed regarding their situation, reduce or stop their usual activities and many end up on long-term sick leave. It is vital to work with the patient in order to develop a graded return to activity. This may incorporate normal household activities, fitness programmes and a structured and graded return to work. A daily walking programme is very effective at encouraging independence, raising confidence and restoring lost cardiovascular fitness. It is important to realize that during recovery from vestibular dysfunction, tiredness and loss of concentration are typical symptoms, and that the body requires sufficient recovery time while undergoing a vestibular training programme.

Early return to work is advisable wherever possible in order to minimize long-term disability and help overcome feelings of depression, anxiety and isolation. Problems with concentration and tiredness mean that a graded return is usually necessary. Typically, dizzy clients report issues with computer use due to visual overload. Manual workers may report issues with regards to working at heights and head movements, for example, bending forwards or looking up. Patients should be reassured that they will desensitize to the particular demands of their employment so long as they do not overdo the activity in the early stages. An early return to activity hastens the recovery, a process that may be as short as 2 weeks in some, but more typically occurs over a period of 2–6 months. Special mention should be made for workers in visually disorientating environments, for example, pilots, fire fighters, underwater divers. Vestibular rehabilitation programmes need to incorporate specific exercises in order to ensure that these clients do not remain dependent upon vision in order to balance.

Use of vestibular suppressant medication

The use of medication to suppress symptoms should be limited to the acute phase of a sudden vestibular loss (3–5 days). Vestibular suppressants help to reduce activity in the vestibular nuclei and cerebellum [17] and reduce nausea and vomiting. It is the tonic asymmetry within the vestibular nuclei that causes the symptoms of vertigo and in order for accurate central compensation to occur, the brain needs to be made aware of this asymmetry. Although most vestibular experts agree that vestibular suppressant medication hinders central compensation and hence recovery [43,44], this continues to be the mainstay of medical treatment, with 61–89% of patients receiving vestibular suppressants at the initial medical consultation. Li et al. [45] report that 90% of patients correctly diagnosed with BPPV were given suppressant medication as their treatment. Many patients reporting with generalized dizziness do not have vestibular dysfunction, but have symptoms due to other conditions. Prescribing vestibular suppressants will hinder vestibular system function precisely when they most depend upon it.

Conclusion

Dizziness represents a major challenge to health professionals. It is common, often becomes chronic and remains largely untreated. By the time that the majority of patients seek help from a specialist centre, medical examination reveals remarkably little sign of peripheral or central vestibular pathology. Despite this, the symptoms of dizziness and the resultant disability may be distressing for the patient and problematic for the clinician. Accurate clinical ocular examination will differentiate between central and peripheral vestibular lesions. Symptoms usually begin with a dysfunction in the peripheral vestibular system. The brain should compensate centrally for the resultant vestibular tonic imbalance. In order for this to occur, the brain requires feedback, in the form of head, eye and neck movements. Unfortunately, due to the unpleasant symptoms many patients refrain from movement, hindering central compensation. In order to avoid chronic long-term complications, vestibular rehabilitation is extremely effective in kick starting the process of central compensation, reducing symptoms and restoring patients to full activity. Dizzy patients are extremely satisfying to work with, because if they receive the correct treatment, they recover.

Key points

- Negative head impulse test, direction-reversing gaze-evoked nystagmus and vertical skew deviation suggest central vestibular lesions.
- Positive head impulse test, unilateral nystagmus and no vertical skew deviation indicate peripheral vestibular lesions.
- Peripheral vestibular neuronitis may be considered as an acute reversible peripheral neuropathy.
- Benign positional vertigo is the most common form of vestibular dizziness. It is diagnosed with a Dix–Hallpike test and treated with appropriate particle repositioning manoeuvres.
- Vestibular suppressant medication should be restricted to the acute phase of a vestibular episode.
- Vestibular rehabilitation therapy is the most effective treatment for continuous or chronic dizziness.

Conflicts of interest

None declared.
References

Fifty years ago: ‘Free enterprise and public service’

But what happens with occupational health? The fate of the Slough Occupational Hygiene Service is the latest instalment of a sorry tale of Governmental indifference and the misguided application of a political slogan in this field. It began as part of the Slough Industrial Health Service, and was described in the first volume of this journal [Nash, 1951, 1, 123]. Its laboratories were established at the London School of Hygiene, where they remained until 1956, when they were moved to Slough. In 1961, with the assistance of a grant from the Nuffield Foundation, it was established as an independent unit, aiming to serve industry in the whole of the British Isles, and equipped with the men and facilities for investigating and advising on the industrial environment. It had to become self-supporting, by means of the fees charged to industry for its services. In March 1964, it died. It had committed the unforgivable sin of the Admass world—it was not a commercial success.

Repeatedly, we have been told that the Government favours the maximum possible voluntary effort in the field of occupational health. What this has meant in effect, both for the Occupational Hygiene Service and for the majority of the group occupational health services which exist, is that having been launched by charity, which is none the less so for bearing a respected name, they must sell themselves to industrialists just as motor cars and television sets are sold to the public. Thus, doctors and their co-workers are forced into a field for which they are untrained and must undertake, in addition to their proper work, a kind of sales promotion which must be amateurish, is distasteful and distracting, and which may at times bring them perilously near to offending against the accepted ethical code, which precludes advertising.

This state of affairs stems directly from Government policy. There are quite genuine practical difficulties in the way of establishing a national occupational health service, even given the will; its financing and its staffing would both present major problems. But no such considerations need have prevented the Government from saving the Slough Occupational Hygiene Service from extinction, either by underwriting its deficit or by taking it over and assigning it to the Factory Inspectorate, which possesses no comparable facilities of its own. Countries with a mere fraction of our population and wealth, such as Finland, put us to shame in this respect, but Her Majesty’s advisers were unmoved.

A time is bound to come when we cease to apply the standards of the world of commerce to occupational health services. Meanwhile, tragically, the team is already disbanded, and the equipment sold; but at least the pioneering achievements of the Service will be remembered, and something at least of its experience will reach its successors. Its epitaph should mark its demise as a sacrifice to an economic doctrine applied in a context where it is supremely irrelevant.

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