

## CHAPTER 12 - AVIATION NEUROLOGY

### [1 INTRODUCTION

Despite the increasing automation of modern civil aircraft, human pilots will remain a necessity just as long as their brain continues to be more comprehensive and flexible than aircraft systems. The effectiveness of a pilot as a control system is normally assessed operationally during his/her training and subsequent flying career. This assessment is subjective, giving few comparable parameters by which performance change can be recognised and so 'pilot error' remains the single major reported cause for aircraft accidents. Given that 'pilot judgmental error' is such an important aspect of aviation safety with so few specific parameters it is not surprising that those areas of cerebral activity that can be measured and examined receive very close scrutiny by the aviation medical physician, frequently out of proportion to the risk involved. The neurological assessment for aviation fitness must therefore be assessed not only in terms of what can be measured and examined but also regarding the aviation risk involved.]

### 2 PATHOLOGY OF THE NERVOUS SYSTEM

Pathology of the nervous system may:

- a Reduce or distort the sensory input from, and appreciation of the external and internal environment [(flight instruments and aids)].
- b Impair assessment, judgement and decision making.
- c [Affect the motor skills necessary for good piloting. Effects of such pathology may be episodic, potentially recurring, static or progressive. Neurological assessment should include careful history and physical examination with particular attention being paid to those areas mentioned in the standards and particularly recognised as aviation problems. Consultation with appropriate specialists is essential in doubtful cases or when questionable findings are noted.]

### 3 NEUROLOGICAL FITNESS

A satisfactory assessment may be achieved if:

- a [There is no demonstrable abnormality of history, examination or performance];
- b Any abnormality noted has an acceptable risk of hazard to the safety of the flight operation concerned. Such abnormality may be congenital or acquired, it may additionally be a single event, static, progressive or intermittent but potentially recurrent. The condition may improve but subsequently relapse. Neurological 'fitness' for aviation purposes must therefore be demonstrated at initial examination and maintained throughout the defined period of medical certificate validation].

### 4 EXAMINATION TECHNIQUES

#### 4.1 GENERAL

The neurologic examination begins with observations of the patient while the history is being obtained. The manner in which the patient tells the story of his illness may betray confusion or incoherence in thinking, impairment of memory or judgment, or difficulty in comprehending or expressing ideas. A common error is to pass lightly over inconsistencies in history and inaccuracies about dates and symptoms, only to discover later that these flaws in memory were the essential features of the illness. Asking the patient to give his own interpretation of the possible meaning of symptoms may sometimes expose unnatural concern, anxiety, suspiciousness, or even delusional thinking. One then generally proceeds from an examination of the cranial nerves, neck, and trunk to the testing of motor, reflex, and sensory functions of the upper and lower limbs. This is followed by an assessment of the function of sphincters and the autonomic nervous system and suppleness of the neck and spine (meningeal irritation).

Gait and station (standing position) should be observed before or after the rest of the examination. In addition, it is often instructive to observe the patient in the course of his natural activities, such as walking or dressing; this may disclose subtle abnormalities of gait and movement that might not be evident in formal testing

<b>Brief neurologic examination in the general medical patient (performed in 5 minutes or less)</b>	
1.	Orientation, insight into illness, language assessed during taking of the history
2.	Size of pupils, reaction to light, visual and auditory acuity
3.	Movement of eyes, face, tongue
4.	Examination of the outstretched hands for atrophy, pronating or downward drift, tremor, power of grip, and wrist dorsi flexion
5.	Biceps, supinator, and triceps tendon reflexes
6.	Inspection of the legs during active flexion and extension of the hips, knees, and feet
7.	Patellar, Achilles, and plantar (Babinski) reflexes
8.	Vibration sensibility in the fingers and toes
9.	Finger-to-nose and heel-to-shin testing of coordination
10.	Gait

#### 4.2 TESTING OF HIGHER CORTICAL FUNCTIONS

These functions are tested in detail if the patient's history or behaviour during the general examination has provided a reason to suspect some defect. Questions should then be directed toward determining the patient's orientation in time and place and insight into his current medical problem. Attention, speed of response, ability to give relevant answers to simple questions, and the capacity for sustained and coherent mental effort all lend themselves to straightforward observation. Useful bedside tests of attention, concentration, memory, and clarity of thinking include the repetition of a series of digits in forward and reverse order, serial subtraction of 3s or 7s from 100, recall of three items of information or a short story after an interval of 3 min, and naming the last six presidents or prime ministers. The patient's account of his recent illness, medical consultations, dates of hospitalization, and his day-to-day recollection of medical procedures, meals, and other incidents are excellent tests of memory; the narration of the illness and the patient's choice of words (vocabulary) provide information about his intelligence and coherence of thinking. Many other tests can be devised for the same purpose. Often the examiner can obtain a better idea of the clarity of the patient's sensorium and soundness of intellect by using these few tests and noting the manner in which he deals with them than by relying on the score of a formal intelligence test. If there is any suggestion of a speech or language disorder, the nature of the patient's spontaneous speech should be noted. In addition, his ability to read, write, and spell, execute spoken commands, repeat words and phrases spoken by the examiner, name objects and parts of objects, and solve simple arithmetical problems should be assessed. The ability to carry out commanded tasks (praxis) has great salience in the evaluation of several aspects of cortical function. Bisecting a line, drawing a clock or the floor plan of one's home or a map of one's country, and copying figures are useful tests of visuospatial perception and are indicated in cases of suspected cerebral disease.

#### 4.3 TESTING OF CRANIAL NERVES

The function of the cranial nerves must be investigated more fully in patients who have neurologic symptoms than in those who do not. If one suspects a lesion in the anterior fossa, the sense of smell should be tested in each nostril; then it should be determined whether odors can be discriminated. The olfactory defect can be verified readily enough by presenting a series of nonirritating olfactory stimuli (vanilla, peanut butter, coffee, tobacco, etc.), first in one nostril, then in the other, and asking the patient to sniff and identify them. Ammonia and similar pungent substances are unsuitable stimuli because they do not test the sense of smell but have a primary irritating effect on the mucosal free nerve endings of the trigeminal nerves. Unilateral gustatory impairment can be identified by withdrawing the tongue with a gauze sponge and using a moistened applicator to place a few crystals of salt or sugar on discrete parts of the tongue; the tongue is then wiped clean and the subject is asked to report what he or she had sensed.

Visual fields should be outlined by confrontation testing, in some cases by testing each eye separately; if any abnormality is suspected, it should be checked on a perimeter and scotomas sought on the tangent screen or, more accurately, by computed perimetry. Pupil size and reactivity to light and accommodation during convergence, the position of the eyelids, and the range of ocular movements should next be observed. To examine the eye movements, the patient may be asked to look quickly to each side as well as up and down (saccades) and to follow a moving target (pursuit of a light, the examiner's or the patient's finger, or an optokinetic drum).

Sensation over the face is tested with a pin and wisp of cotton; also, the presence or absence of the corneal reflexes may be determined. Facial movements should be observed as the patient speaks and smiles, for a slight weakness may be more evident in these circumstances than on movements to command. The auditory meati and tympanic membranes should be inspected with an otoscope. A 256 double-vibration tuning fork held next to the ear and on the mastoid discloses hearing loss and distinguishes middle-ear (conductive) from neural deafness. Audiograms and other special tests of auditory and vestibular function are needed if there is any suspicion of disease of the eighth nerve or the cochlear and labyrinthine end organs. The vocal cords must be inspected with special instruments in cases of suspected medullary or vagus nerve disease, especially when there is hoarseness. Voluntary pharyngeal elevation and elicited reflexes are meaningful if there is a difference on the two sides; bilateral absence of the gag reflex is seldom significant. Inspection of the tongue, both protruded and at rest, is helpful; atrophy and fasciculations may be seen and weakness detected. Slight deviation of the protruded tongue as a solitary finding can usually be disregarded. The pronunciation of words should be noted. The jaw jerk and the snout, buccal, and sucking reflexes should be sought, particularly if there is a question of dysphagia, dysarthria, or dysphonia.

#### 4.4 TESTS OF MOTOR FUNCTION

In the assessment of motor function, it should be kept in mind that observations of the speed and strength of movements and of muscle bulk, tone, and coordination are usually more informative than the state of tendon reflexes. It is essential to have the limbs fully exposed and to inspect them for atrophy and fasciculations. The next step is to watch the patient maintain the arms outstretched in the prone and supine positions; perform simple tasks, such as alternately touching his nose and the examiner's finger; make rapid alternating movements that necessitate sudden acceleration and deceleration and changes in direction, such as tapping one hand on the other while alternating pronation and supination of the forearm; rapidly touch the thumb to each fingertip; and accomplish simple tasks such as buttoning clothes, opening a safety pin, or handling common tools. Estimates of the strength of leg muscles with the patient in bed are often unreliable; there may seem to be little or no weakness even though the patient cannot arise from a chair or from a kneeling position without help. Running the heel down the front of the shin, alternately touching the examiner's finger with the toe and the opposite knee with the heel, and rhythmically tapping the heel on the shin are the only tests of coordination that need be carried out in bed. The maintenance of both arms against gravity is a useful test; the weak one, tiring first, soon begins to sag, or, in the case of a corticospinal lesion, to resume the more natural pronated position ("pronator drift"). The strength of the legs can be similarly tested, either with the patient supine and the legs flexed at hips and knees or with the patient prone and the knees bent. Also, abnormalities of movement and posture and tremors may be exposed.

#### 4.5 TESTS OF REFLEX FUNCTION

Testing of the biceps, triceps, supinator (radial-periosteal), patellar, Achilles, and cutaneous abdominal and plantar reflexes permits an adequate sampling of reflex activity of the spinal cord. Triggering tendon reflexes requires that the involved muscles be relaxed; underactive or reflexes difficult to trigger can be facilitated by voluntary contraction of other muscles (Jendrassik maneuver). The plantar response poses special difficulty because several different reflex responses can be evoked by stimulating the sole of the foot along its outer border from heel to toes. These are:

- (1) the quick, high-level avoidance response;
- (2) the slower, spinal flexor nocifensive (protective) reflex (flexion of knee and hip and dorsiflexion of toes and foot, "triple flexion") — dorsiflexion of the large toe as part of this reflex is the well-known Babinski sign;

- (3) plantar grasp reflex; and;
- (4) support reactions. Avoidance and withdrawal responses interfere with the interpretation of the Babinski sign and can sometimes be overcome by utilizing the several alternative stimuli that are known to trigger the Babinski response (squeezing the calf or Achilles tendon, flicking the fourth toe, downward scraping of the shin, lifting the straight leg, and others). An absence of the superficial cutaneous reflexes of the abdominal, cremasteric, and other muscles are useful ancillary tests for detecting corticospinal lesions. The finding of absent Achilles reflexes and diminished vibratory sense in the feet and legs alerts the physician to the possibility of diabetic or alcoholic-nutritional neuropathy even when the patient has no symptoms referable to these disorders.

#### 4.6 TESTING OF SENSORY FUNCTION

This is undoubtedly the most difficult part of the neurologic examination. Usually sensory testing is reserved for the end of the examination and, if the findings are to be reliable, should not be prolonged for more than a few minutes. Each test should be explained briefly; too much discussion of these tests with a meticulous, introspective patient may encourage the reporting of useless minor variations of stimulus intensity. It is not necessary to examine all areas of the skin surface. A quick survey of the face, neck, arms, trunk, and legs with a pin takes only a few seconds. Usually one is seeking differences between the two sides of the body (it is better to ask whether stimuli on opposite sides of the body feel the same than to ask if they feel different), a level below which sensation is lost, or a zone of relative or absolute analgesia (loss of pain sensibility) or anesthesia (loss of touch sensibility). Regions of sensory deficit can then be tested more carefully and mapped out. Moving the stimulus from an area of diminished sensation into a normal area enhances the perception of a difference. The vibration sense may be tested by comparing the thresholds at which the patient and examiner lose perception at comparable bony prominences. We usually record the number of seconds for which the examiner appreciates vibration at the malleolus or toe after the patient reports that the fork has stopped buzzing. The finding of a zone of heightened sensation ("hyperesthesia") calls attention to a disturbance of superficial sensation.

Variations in sensory findings from one examination to another reflect differences in technique of examination as well as inconsistencies in the responses of the patient.

#### 4.7 TESTING OF GAIT AND STANCE

The examination is completed by observing the patient stand and walk. An abnormality of stance and gait may be the most prominent or only neurologic abnormality, as in certain cases of cerebellar or frontal lobe disorder; and an impairment of posture and highly automatic adaptive movements in walking may provide the most definite diagnostic clues in the early stages of Parkinson disease and progressive supranuclear palsy. Having the patient walk tandem or on the sides of the soles may bring out a lack of balance and dystonic postures in the hands and trunk. Hopping or standing on one foot may also betray a lack of balance or weakness, and standing with feet together and eyes closed will bring out a disequilibrium that is due to deep sensory loss (Romberg test). When confronted with a disorder of gait, the examiner must observe the patient's stance and the attitude and dominant positions of the legs, trunk, and arms. It is good practice to watch patients as they walk into the examining room, when they are apt to walk more naturally than during the performance of special tests. They should be asked to stand with feet together and head erect, with eyes open and then closed. A normal person can stand with feet together and eyes closed while moving the head from side to side, a test that eliminates both visual and vestibular cues and induces certain compensatory trunk and leg movements that depend on proprioceptive afferent mechanisms. As already mentioned, the Romberg sign - marked swaying or falling with the eyes closed but not with the eyes open - usually indicates a loss of postural sense, not of cerebellar function, although with vestibular or cerebellar dysfunction there may be a less pronounced exaggeration of swaying with eyes closed and feet together. Swaying due to nervousness may be overcome by asking the patient to touch the tip of his nose alternately with the forefinger of one hand and then the other. Next, the patient should be asked to walk, noting in particular any hesitation in starting and negotiating turns, width of base, length of stride, foot clearance, arm swing, and cadence. A tendency to veer to one side, as occurs with unilateral cerebellar disease, can be brought out by having the patient walk around a chair. When the affected side is toward the chair, the patient tends to walk into it; when it is away from the chair, there is a veering outward in ever-widening circles. More delicate tests of gait are walking a straight line heel to toe

("tandem walking test"), walking backward, and having the patient arise quickly from a chair, walk briskly, stop and turn suddenly, walk back and then sit down again. It is instructive to observe the patient's postural reaction to a sudden push backward and forward or to the side. With postural instability there is a delay or inadequacy of corrective actions. Finally, the patient should be asked to hop on one leg and to jog. If all these tests are successfully executed, it may be assumed that any difficulty in locomotion is not due to impairment of a proprioceptive, labyrinthine-vestibular, basal ganglionic, or cerebellar mechanism.

## 5 HEADACHE-FACIAL PAIN

Of all the painful states that afflict humans, headache is undoubtedly the most frequent. Overall, more than 90% of the headache patients examined annually suffer from the primary headache disorders — migraine, tension-type, or cluster headache. The remaining patients have headache secondary to tumor, meningitis, giant cell arteritis, sinusitis, or other medical conditions. Intracranial pathology is extremely uncommon among patients with primary headache disorders. Only 0.18% of patients with migraine and a normal neurological examination will have a significant intracranial abnormality.

<b>Clinical classification and prevalence of different types of headache in the general population (%)</b>	
<i>(Primary %)</i>	
Muscle contraction/tension-type headache	69
Idiopathic stabbing headache (ice cream/ice pick headache)	33
Migraine	16
Exertional headache	1
Cluster headache	0.1
<i>(Secondary %)</i>	
Systemic infection	63
Head injury	4
Drug induced headache	3
Vascular disorders	1
Subarachnoid haemorrhage	<1
Brain tumour	0.1

<b>Aetiology of headache</b>	<i>Adults (17–65 years)</i>	<i>Elderly (65+ years)</i>
Tension headache	+++	+
Migraine	+++	+
Idiopathic stabbing headache	+	+
Exertional headache	++	–
Cluster headache	++	+
Post-traumatic	+++	+
Drug-induced headache	++	++
Cervicogenic (referred from neck)	+	+++
Cranial arteritis	+	+++
Subarachnoid haemorrhage	++	+
Subdural haematoma	++	++
Brain abscess	+	+
Brain tumour	++	++
Idiopathic intracranial hypertension	++	–
Glaucoma	+	++
Paget's disease of the skull	+	++
Cerebral venous sinus thrombosis	+	+
Arnold–Chiari malformation	+	+/-

## 5.1 CLINICAL HISTORY

- Types of headache: how many types of headache do you suffer from?
- Length of history of headache: is this a new headache or not; has it changed in character, severity or frequency?
- Features of the headache itself:
  - Location/site of headache.
  - Onset.
  - Type/quality of headache (throbbing, steady).
  - Timing.
  - Severity.
  - Radiation.
  - Associated features (e.g. nausea, photophobia, phonophobia, symptoms of aura, fever, neurological symptoms such as weakness, diplopia, clumsiness, disturbance of balance, altered cognitive function; altered consciousness).
  - Exacerbating factors (e.g. physical activity, bright light, noise).
  - Relieving factors.
  - Duration of headache.
  - Predisposing factors.
  - Premonitory features.
- Family history.
- Medications/drugs.

Most often, secondary headache can be suspected when 1 or more of the features shown in Table 1 are present.

**Table 1: Red flags symptoms**

Systemic symptoms/signs (fever, myalgias, weight loss)
Systemic disease (malignancy, acquired immune deficiency syndrome)
Neurologic symptoms or signs
Onset sudden (thunderclap headache)
Onset after age 40 years
Pattern change
– Progressive headache with loss of headache-free periods
– Change in type of headache

## 5.2 SELECTED DIAGNOSTIC TESTING

Physical examination

Metabolic evaluation

- Hematological
- ESR/CRP
- Endocrinological
- Chemistry
- Toxicology (drug screens, etc.)

Standard x-rays

Neuroimaging

- CT
- MRI/MRA/MRV

Dental and otological exam

Lumbar puncture

Diagnostic blockades

## 5.3 MIGRAINE

A symptom complex, or syndrome, that manifests as discrete episodes of headache associated with other features of sensory sensitivity.

### 5.3.1 **Migraine variants**

#### 5.3.1.1 **Ophthalmoplegic migraine:**

Paralysis of the >1 ocular cranial nerves, usually the IIIrd nerve, at the height of a migraine headache. The paralysis usually resolves but may persist after recurrent episodes.

#### 5.3.1.2 **Vertebrobasilar migraine**

Gradual onset and evolution over several minutes of brainstem, cerebellar and visual disturbances, often accompanied or followed by headache and syncope.

#### 5.3.1.3 **Hemiplegic migraine**

Hemiparesis preceding or occurring with a migraine headache.

#### 5.3.1.4 **Migrainous infarction**

Permanent focal neurological symptoms persisting beyond 24 hours after the cessation of migraine headache.

### 5.3.2 **Aeromedical Status**

Migraine cannot be treated in pilots with ergotamine and other drugs, because such medications are unacceptable in aviation due to side effects. Known migraineurs should not be selected for professional aircrew training due to the unpredictability and disabling nature of the condition, but those who present after qualification either through worsening of the condition or an in-flight incident should be neurologically assessed and any causal factor addressed. If no other structural abnormality is found and the individual is migraine free for 3 to 6 months, a return to flying may be approved with a multi-pilot (Class 1 'OML') limitation. A fit assessment for solo operations must be evaluated against the history and type of operation. Some prophylactic treatments such as propranolol, may be acceptable with each case being reviewed individually.

A similar assessment applies to Class 2.

The following must be considered:

- Frequency of headaches
- Degree of incapacitation caused by the headache.
- Drugs used to treat the headache.

Adverse factors for aeromedical certification include:

- Sudden significant neurological symptom such as loss of vision, weakness and incoordination with no warning
- Failure or of prophylactic treatment with frequent attacks
- Requirement for intensive treatment
- Short prodrome that does not allow effective use of acute treatment before symptom onset.

### 5.4 **MUSCLE CONTRACTION/TENSION TYPE HEADACHE**

An episodic or chronic continuous headache due to sustained muscle contraction.

#### 5.4.1 **Aeromedical Status**

Most applicants should be considered fit. Special Aeromedical assessment should be considered in case of use of chronic tension headaches that require treatment such as anxiolytics or other drugs likely to cause a decreased state of alertness or diminished performance. The chronic use of medication is against fitness to fly.

## 5.5 CLUSTER HEADACHE

A form of primary headache marked by recurrent episodes, lasting 15–180 minutes, of excruciating unilateral periorbital pain and associated autonomic features, that tend to occur once or twice a day in bouts or clusters, lasting from weeks to months at a time, separated by remission periods of months or 1–2 years.

### 5.5.1 Aeromedical Status

Cluster headaches are extremely painful and unpredictable – they require neurological assessment and pharmacological treatment is often unacceptable for flying. Individuals with such conditions need an extended pain free period of temporarily unfit assessment before being considered for a return to multi-pilot flying. A similar assessment applies to Class 2 'OSL' and Class 2.

## 5.6 TRIGEMINAL NEURALGIA

Specialised neurosurgical assessment may be needed. Consideration must be given to the side effects of medications commonly used in its treatment

### 5.6.1 Aeromedical Status

(see under cluster headache)

## 6 ACQUIRED METABOLIC DISEASES OF THE NERVOUS SYSTEM

### 6.1 HYPOXIC ENCEPHALOPATHY

Brain dysfunction caused by a lack of oxygen to the brain as a result of failure of the circulation or respiration.

#### 6.1.1 Aeromedical Status

The applicants should be considered unfit. However, in case of mild disease each case should be considered individually by the AMS considering:

- Causal factor
- Clinical features of encephalopathy
- Presence of neurological defects
- Present cognition status

### 6.2 HEPATIC ENCEPHALOPATHY

A clinical neuropsychiatric syndrome characterized by abnormal mental status occurring in patients with severe acute, subacute, or chronic hepatocellular insufficiency.

#### 6.2.1 Aeromedical Status

The applicants should be considered unfit.

## 7 AUTONOMIC NERVOUS SYSTEM DISORDERS

### 7.1 AUTONOMIC NEUROPATHY

#### 7.1.1 Disorders affecting CNS

Primary autonomic failure  
Spinal cord lesions above T6

Cerebrovascular disease.  
Brainstem tumours.  
Multiple sclerosis.  
Tabes dorsalis.

#### 7.1.2 Disorders affecting the peripheral nervous system

Diabetes.  
Acute inflammatory radiculoneuropathy (Guillain–Barré syndrome).  
Acute intermittent porphyria.  
Alcoholism and nutritional diseases.  
Metabolic disorders (vitamin B12 deficiency, chronic renal failure, chronic liver disease).  
Charcot–Marie–Tooth disease.  
Malignancy.  
Rheumatoid arthritis.  
Systemic lupus erythematosus.  
Chronic inflammatory neuropathy.  
HIV infections.

#### 7.1.3 Drugs

- Antidepressants.
- Anti-hypertensive drugs.
- Barbiturates.
- Phenothiazines.
- Atropine.
- Epidural anaesthesia.

#### 7.1.4 Clinical Features

The most common clinical manifestations of autonomic dysfunction are:

- Postural (orthostatic) hypotension; impotence.
- Disorders of bladder function.
- Abnormalities of sweating.
- Vasomotor disturbances.

##### *Sympathetic adrenergic failure*

- Postural hypotension.
- Ejaculatory failure.

##### *Sympathetic cholinergic failure*

- Anhidrosis.

##### *Parasympathetic failure*

- Fixed heart rate.
- Sluggish urinary bladder and bowel.
- Erectile failure.

#### 7.1.5 Aeromedical Status

The applicants should be considered unfit. However, in cases with minor manifestations each case should be considered individually by the AMS considering:

- Flight safety
- Causal factor
- Clinical symptoms

## 8 DISORDERS OF CONSCIOUSNESS

### 8.1 NARCOLEPSY

A syndrome of excessive sleepiness and abnormalities of rapid-eye-movement (REM) sleep.

### 8.1.1 Clinical Features

Excessive sleepiness  
Cataplexy  
Hypnagogic hallucinations

### 8.1.2 Aeromedical Status

The applicants should be considered unfit.

## 8.2 SYNCOPE

A transient loss of postural tone and consciousness resulting from an acute reduction in blood flow to the brain.

History of the event is paramount in differentiation of the causes.

Specific features that will help in differentiating the physiological system involved are:

- Prodrome: absence or present.
- Posture at the time of the episode.
- Period: ie, duration of attack.
- Postictal orientation.
- Activity before, immediately and within 24 hr preceding.
- Head trauma.
- Frequency.
- Urinary incontinence.
- Tongue biting.
- Observer report: confirmation of patient's account, particularly concerning convulsive movements. Time course to any convulsive movement is important ie, did it occur at the same time as LOC, or seconds later?
- Bystanders' action: e.g., promptly placing patient in prone or coma position, or keeping patient sitting/upright.
- Family and/or past history.
- Known cardiovascular history or risks.
- History of infection such as recent viral infection that may support labyrinthitis.

Depending on the historical features elicited, the need for referral to relevant specialist/s can be determined.

Prognosis depends on the cause.

### 8.2.1 Aeromedical Status

Each case should be considered individually by the AMS considering:

Cause  
Clinical symptomatology  
Flight safety  
Remission rate  
Precipitating factors

## 9 CRANIAL NEUROPATHIES

### 9.1 OLFACTORY (CRANIAL NERVE I) NEUROPATHY

Disorder of the 1<sup>st</sup> cranial, or olfactory, nerve resulting in a disturbance of smell sensation (anosmia).

## 9.2 OPTIC (CRANIAL NERVE II) NEUROPATHY

Special concern should be given to optic neuritis as a multiple sclerosis early sign. About 15% of patient will develop clinically definite MS within the next 2 years, 22% within 5 years, 35% in 10 years, and about 50–60% of patients with optic neuritis will eventually develop MS.

Following optic neuritis *MS is less likely to occur*

- in childhood than in adults.
- With bilateral simultaneous onset.
- With absence of pain.
- With marked disc oedema.
- If there are no oligoclonal bands in the CSF.
- If the brain MRI is normal

*MS is more likely to occur*

- If previous ill-defined non-specific neurological symptoms are present.
- If there is an history of previous optic neuritis.
- With increased CSF IgG.
- If the brain MRI is abnormal with three or more lesions suggestive of demyelinating disease:
- Possibly, if there is a family history of MS.

## 9.3 HORNER'S SYNDROME

Horner's syndrome presents as unilateral ptosis, miosis and anhidrosis due to damage to the ipsilateral oculosympathetic pathway. Prognosis depends on the cause. Generally benign in isolated, new onset postganglionic Horner's syndrome.

## 9.4 CRANIAL NERVE III, IV, V (OCULAR MOTOR) NEUROPATHIES

These disorders, along with disorders of the extraocular muscles, that are innervated by these nerves, cause dysconjugate eye movements and thus binocular diplopia (unless either eye is closed, blind or amblyopic). Prognosis depends on the cause.

## 9.5 TRIGEMINAL (CRANIAL NERVE V) NEUROPATHY

Prognosis depends on the cause.

## 9.6 FACIAL (CRANIAL NERVE VII) NEUROPATHY

Differential diagnosis should be made between Upper and lower motor neurone facial weakness. Prognosis depends on the cause.

Acute Idiopathic Facial Paralysis (Bell's Palsy) is a unilateral, lower motor neurone facial paralysis that is probably due to acute viral inflammatory demyelination of the facial nerve causing swelling and secondary nerve ischaemia within the facial canal. 60–80% of patients recover completely. In these cases, recovery usually begins within 8 weeks and is complete by 6–12 months. The most favourable prognostic sign is an incomplete rather than complete facial palsy. If weakness is severe or complete, recovery commencing within 3 weeks is a favourable sign. The longer the delay in return of movement the poorer the recovery.

Predictors of incomplete recovery are:

- Complete facial weakness.
- Pain other than in or around the ear (i.e. back of head, cheek, other).
- Systemic hypertension, diabetes or psychiatric illness.
- Older age.
- Hyperacusis.
- Decreased tearing.

## 9.7 VESTIBULAR-COCHLEAR (CRANIAL NERVE VIII) NEUROPATHY

An ENT assessment will be needed. Prognosis depends on the cause.

## 9.8 AEROMEDICAL STATUS

These disorders are assessed on the basis of the nature and degree of deficit. Each case should be considered individually by the AMS considering:

- Cause
- Residual neurological defects
- Associated clinical-laboratory findings
- Flight safety concerns

## 10 DEGENERATIVE DISEASES OF THE NERVOUS SYSTEM

### 10.1 DEMENTIA

Dementia is characterized by progressive intellectual deterioration that is sufficiently severe to interfere with social or occupational functions. Memory, orientation, abstraction, ability to learn, visuospatial perception, language function, constructional praxis, and higher executive functions, such as planning, organizing, and sequencing, are all impaired in dementia.

#### 10.1.1 Causes of dementia

- Alzheimer's disease : 50–55%.
- Vascular dementia : 15–20%.
- Diffuse Lewy body disease : 15–25%.
- Parkinson's disease : 5–10%.
- Brain injury: alcohol, head trauma : 5%.
- Other causes : 5%:
  - Normal pressure hydrocephalus .
  - Intracranial mass lesion: frontal or temporal lobe tumour, chronic subdural haematoma.
  - Metabolic/toxic: chronic drug intoxication (e.g. alcohol, barbiturates, sedatives), chronic hepatic encephalopathy.
  - Endocrine: hypothyroidism, Cushing's syndrome.
  - Autoimmune: SLE.
  - Nutritional: vitamin B12 deficiency ; Wernicke– Korsakoff syndrome .
  - Syphilis (general paresis of the insane) ; HIV.
  - Creutzfeldt–Jakob disease : myoclonus and rapidly progressive dementia.

Multiple causes (i.e. combinations of the above): 10–15%


Mini-Mental Scale (MMSE) is a useful tool for clinical practice to identify in early stages some cognition decline It should be applied in pilots over 40's. Mild Cognitive Impairment (MCI) is an early stage of Alzheimer disease. In most studies, 10 to 20 percent per year of such affected patients will be found to have acquired Alzheimer disease.

MMSE $\leq$ 27 should needed further evaluation

#### 10.1.2 Aeromedical Status

Applicants with dementia are permanently unfit. In the small number of cases where the cause of the dementia is known and treatable and the condition has been resolved, applicants may be considered for a fit assessment at revalidation / renewal.

**“Mini-Mental” status test of Folstein, Folstein, and McHugh**

TASK	INSTRUCTIONS	SCORING	
Date orientation	“Tell me the date?” Ask for omitted items.	One point each for year, season, date, day of week, and month	5
Place orientation	“Where are you?” Ask for omitted items.	One point each for state, county, town, building, and floor or room	5
Register three objects	Name three objects slowly and clearly. Ask the patient to repeat them.	One point for each item correctly repeated	3
Serial sevens	Ask the patient to count backwards from 100 by 7. Stop after five answers. (Or ask them to spell “world” backwards.)	One point for each correct answer (or letter)	5
Recall three objects	Ask the patient to recall the objects mentioned above.	One point for each item correctly remembered	3
Naming	Point to your watch and ask the patient “what is this?” Repeat with a pencil.	One point for each correct answer	2
Repeating a phrase	Ask the patient to say “no ifs, ands, or buts.”	One point if successful on first try	1
Verbal commands	Give the patient a plain piece of paper and say “Take this paper in your right hand, fold it in half, and put it on the floor.”	One point for each correct action	3
Written commands	Show the patient a piece of paper with “CLOSE YOUR EYES” printed on it.	One point if the patient’s eyes close	1
Writing	Ask the patient to write a sentence.	One point if sentence has a subject, a verb, and makes sense	1
Drawing	 Ask the patient to copy a pair of intersecting pentagons onto a piece of paper.	One point if the figure has ten corners and two intersecting lines	1
<b>Scoring</b>	<b>A score of 24 or above is considered normal.</b>		<b>30</b>

**10.2 PARKINSON’S DISEASE**

A slowly progressive, age-related, degenerative disorder of the CNS, characterized clinically by tremor, bradykinesia, rigidity, and disturbed postural reflexes (parkinsonism).

**10.2.1 Aeromedical Status]**

Most applicants with Parkinsonism suffer physical and/or cognitive deficits which render them unfit. Individuals who have minimal Parkinson’s disease, such that they do not require L-dopa or an L-dopa agonist, may [be assessed as fit]. All patients licensed with Parkinson’s disease must have a detailed neurological assessment at each [revalidation /renewal] and more frequently if clinically indicated. This assessment applies to Class 1 and Class 2.

### 10.3 **MULTIPLE SYSTEMS ATROPHY (MSA)**

A sporadic, adult-onset progressive, degenerative disease of unknown aetiology which is clinically protean, characterized by autonomic dysfunction, parkinsonism, and ataxia in any combination,

#### 10.3.1 **Aeromedical Status**

Applicants with MSA are permanently unfit.

## 11 **DEVELOPMENTAL DISEASES OF THE NERVOUS SYSTEM**

### 11.1 **ARNOLD–CHIARI MALFORMATION**

A number of developmental anomalies of the hindbrain, base of skull, and upper cervical canal characterized by caudal displacement of the cerebellar tonsils, and sometimes more of the cerebellum and lower brainstem, through the foramen magnum into the cervical spinal canal.

#### 11.1.1 **Aeromedical Status**

Applicants are permanently unfit.

### 11.2 **NEUROFIBROMATOSIS**

The neurofibromatoses are a group of neurocutaneous syndromes primarily affecting tissues derived from the neural crest. NF-1 type more common type, with better prognosis

#### 11.2.1 **Aeromedical Status**

Each case should be considered individually by the AMS considering:

- Symptomatology
- Number of lesions

## 12 **EPILEPSY**

### 12.1 **PROGNOSIS**

Risk of a second seizure after a first seizure

- 31–71%, depending on other risk factors:

EEG: epileptiform discharges: 80% risk; non-specific abnormalities: 40% risk; normal: 12% risk.

### 12.2 **GENERALISED TONIC-CLONIC SEIZURES (GRAND MAL)**

- Age group: any age.
- Symptoms:
  - Premonitory irritability, headache, elation, depression in some; others have no warning.
  - Focal onset (an aura) indicates initial partial seizure.
  - Tonic phase (15–30 seconds): contraction of facial, jaw, limb, chest, trunk muscles, initial cry sometimes as air is expelled loss of muscle tone (fall) cyanosis; tonic convulsions.
  - Clonic phase: rhythmic jerking/convulsions of limbs.
  - Post-ictal phase: muscle relaxation slow deep breathing through clenched jaws deep sleep, stupor, confusion for minutes to hours awareness of headache and muscle soreness later.
- Duration: 1–3 minutes.
- Special features:
  - Tongue biting and urinary incontinence sometimes.

- A succession of tonic-clonic seizures, without regaining consciousness for more than 30 minutes, is known as convulsive status epilepticus

### 12.3 ABSENCES (PETIT MAL)

- Age group: children and juveniles.
- Symptoms: sudden brief loss of consciousness, staring and blinking; immediately regain consciousness.
- Duration: few–30 seconds.
- Special features:
  - Long absences may be accompanied by: simple actions (automatisms) upward rolling of the eyes lip smacking, chewing, fiddling, fumbling with clothing slight jerking of limbs (myoclonus), passing urine, falling to the ground (akinetic attack)

### 12.4 JUVENILE MYOCLONIC EPILEPSY

This is the most common form of idiopathic generalized epilepsy in older children and young adults. It begins in adolescence, typically about age 15, with a range that essentially spans all of the teenage years. The patient comes to attention because of a generalised seizure, often upon awakening or because of myoclonic jerks in the morning that involve the entire body; sometimes absence seizures are prominent. The family reports that the patient has occasional myoclonic jerks of the arm and upper trunk that become prominent with fatigue, during early stages of sleep, or after alcohol ingestion. The disorder does not impair intelligence and tends not to be progressive, but a proclivity to infrequent seizures usually continues throughout life. Valproic acid in particular and some other anticonvulsants have been highly effective in eliminating the seizures and myoclonus; they should be continued indefinitely. Withdrawal of anti-epileptic medication is associated with a high relapse rate (in 80% of patients).

### 12.5 PARTIAL EPILEPTIC SEIZURES

#### 12.5.1 Simple partial seizures (focal)

- Age group: any age.
- Symptoms: depend on location of seizure focus; no loss of consciousness and no postictal confusion.
- Duration: seconds to minutes.
- Special features: an 'aura' is a simple partial seizure.
- Subtypes:
  - Focal motor seizures (Jacksonian epilepsy): usually start with twitching of face or thumb which spreads over several seconds to the ipsilateral arm and leg as epileptic activity 'marches' over the contralateral motor cortex.
  - Focal sensory seizures: arise from primary sensory cortex and can take the form of contralateral paraesthesia (somatosensory cortex), unformed visual hallucinations, e.g. light flashes (visual cortex), transient vertigo and simple auditory hallucinations such as crackling, buzzing (auditory cortex-superior temporal gyrus).

#### 12.5.2 Complex partial seizures

- Age group: any age.
- Symptoms: depend on location of seizure focus impaired consciousness; postictal confusion.
- Duration: minutes.
- Subtypes:
  - Olfactory and gustatory hallucinations: usually unpleasant smells and taste, and dreamy state, arise from uncus of temporal lobe.
  - Visual and auditory hallucinations: formed visual hallucinations of objects, animals and people originate in the visual association cortex; complex hallucinations of music and conversation may arise in the auditory association cortex.
  - Emotional disturbances: rapid onset of unexplained fear/terror or elation, may arise in the temporal lobe.

- Memory disturbances (transient): amnesia, *déjà vu* (a false sensation of familiarity), *jamais vu* (a false feeling of unfamiliarity and strangeness), illusion of time passing more quickly or slowly.
- Automatism: episodes of movements and apparently confused behaviours, with impaired awareness and consciousness (e.g. lip smacking, chewing movements, picking at clothes, fumbling with objects, and driving a car), ictal or post-ictal, patient cannot remember them.

### 12.5.3 **Benign Childhood Epilepsy with Centrottemporal Spikes (Rolandic Epilepsy, Sylvian Epilepsy) and Epilepsy with Occipital Spikes**

This type of focal motor epilepsy is unique among the partial epilepsies of childhood in that it tends to be a self-limited disorder, transmitted in families as an autosomal dominant trait. The convulsive disorder begins between 5 and 9 years of age and usually announces itself by a nocturnal tonic-clonic seizure with focal onset.

The seizures are readily controlled by a single anticonvulsant drug and gradually disappear during adolescence.

## 12.6 **FEBRILE SEIZURES**

The well-known *febrile seizure*, peculiar to infants and children between 6 months and 5 years of age (peak incidence 9 to 20 months) and with a strong tendency to be inherited, is generally regarded as a benign condition. Usually it takes the form of a single, generalized motor seizure occurring as the temperature rises or reaches its peak. Seldom does the seizure last longer than a few minutes; Recovery is complete. These patients' risk of developing epilepsy in later life is only slightly greater than that of the general population.

## 12.7 **SEIZURES WITH ONSET IN ADULT LIFE AND SECONDARY TO MEDICAL**

### 12.6.1 **Withdrawal Seizures**

The possibility of abstinence seizures in patients who had chronically abused alcohol, barbiturates, or benzodiazepine sedative drugs must always be considered when seizures occur for the first time in adult life

### 12.6.2 **Infections**

Bacterial meningitis, acute herpes simplex encephalitis

### 12.6.3 **Endogenous Metabolic Encephalopathies**

### 12.6.4 **Medications as a Cause of Seizures**

### 12.6.5 **Global Arrest of Circulation and Cerebrovascular Diseases**

### 12.6.6 **Seizures with Acute Head Injury**

## 12.7 **MANAGEMENT OF FOCAL OR GENERALIZED SEIZURES IN LATE ADULT LIFE**

A person in the later age group who begins to have seizures of either partial or generalised type is always to be suspected of harboring a primary or secondary tumor or an infarct that had not declared itself clinically. This is a matter usually settled by the neurologic examination and by CT or MRI. Tumor, either primary or secondary, will be found to account for about half the cases of seizures occurring for the first time in late adult life. Previous infarcts are by far the most common lesions underlying status epilepticus in late adult life, an old trauma is as common as well. Cortical and subcortical encephalomalacia, the result of previous traumatic contusions, is a particularly important cause of seizures among alcoholics; the lesions are revealed by brain imaging and are typically located in the anterior frontal and temporal lobes.

Brain abscess and other inflammatory and infectious illnesses are less common except in tropical regions. Seizures as a result of Alzheimer and other degenerative diseases do occur but are uncommon. In the not infrequent cases of an adult with a first seizure that remains unexplained after thorough evaluation, it is a

practice to administer an anticonvulsant and to re-evaluate the situation in a matter of 6 to 12 months, with the goal of eventually discontinuing medication. Usually, a second MRI and EEG are performed to exclude focal abnormalities that were not appreciated during the initial evaluation, but often these studies are again unrevealing. One-third of patients with a single unprovoked seizure will have another seizure within 5 years; the risk is even greater if there is a history of seizures in a sibling, a complex febrile convulsion in childhood, or a spike-and-wave abnormality in the EEG. Moreover, the risk of recurrence is greatest in the first 24 months. In patients with two or three unexplained seizures, three-quarters have further seizures in the subsequent 4 years.

## 12.8 DISCONTINUATION OF ANTICONVULSANTS

Withdrawal of anticonvulsant drugs may be undertaken in patients who have been free of seizures for a prolonged period. There are few firm rules to guide the physician in this decision. A safe plan, applicable to most forms of epilepsy, is to obtain an EEG whenever withdrawal of medication is contemplated. If the tracing is abnormal by way of showing paroxysmal activity, it is generally better to continue treatment. After 2 years on a single anticonvulsant during which no seizures had occurred, the rate of relapse was 40 percent 2 1/2 years later and 50 percent at 5 years after discontinuation; this compared to the seizure recurrence rate of 20 percent for patients remaining on medication. Patients with juvenile myoclonic epilepsy, even those with long seizure free periods, should probably continue with medication lifelong, but there have been no thorough studies to our knowledge to support this dictum. The appropriate duration of treatment for postinfarction epilepsy has not been studied, and most neurologists continue to use one drug indefinitely.]

### [12.9] Aeromedical Status

Those which are alcohol-provoked and have no demonstrable EEG abnormality would seem to have the best prognosis and may be considered [with a multi-pilot (Class 1 'OML') or a safety pilot (Class 2 'OSL') limitation] after full consideration of any alcohol abuse problem. Ultimately, any single unprovoked seizure after the age of five [must be considered disqualifying (vide infra) particularly if associated with EEG abnormality with the consideration that a little more latitude in the area of 'provocation' can be allowed for established aircrew. An applicant with a history of a single, uncomplicated febrile convulsion between the age of 1 and 5 years will still be eligible for pilot training. If, however, the convulsion was complicated, the applicant will no longer qualify, i.e.

- i. A convulsion before the age of 1 year. This holds the risk for mental retardation and epilepsy later in life.
- ii. Multiple febrile convulsions.
- iii. Duration of convulsions longer than 5 minutes.
- iv. Lateralising signs during febrile convulsions.

An individual with a single epileptiform seizure is initially unfit for medical certification. A case may be reconsidered five years from a seizure, if the following conditions are met:

- Specialist neurological examination is normal
- Repeated EEGs, including sleep-deprived EEGs, do not reveal any significant abnormalities
- Studies incorporating additional nasopharyngeal or minisphenoidal electrodes, if relevant, do not reveal any significant abnormalities
- Neuro imaging, preferably by MRI, has demonstrated normal brain structure.

In case of Benign rolandic seizures the applicant may be assessed as fit by the AMS.]

### [12.10] THE ELECTROENCEPHALOGRAPH IN AVIATION NEUROLOGY

The EEG is a clinical tool of value to neurological specialists and although widely used for screening aircrew applicants, its sensitivity and specificity under such circumstances remains ill-defined. [EEG is required when indicated by the history of the applicant or on clinical grounds.]

### [12.10.1] Electroencephalograph technique.

In order to reduce variation in interpretation, the technique used must be standardised where possible. The national aeromedical department shall ensure EEG recording facilities are to a high standard [and that the reading and interpretation follows standardised, international procedures.]

#### Recommended procedure

- a 20 leads with 10/20 (international) placement.
- b The montage and machine settings shall be indicated on the tracing.
- c Calibration is required at the beginning and end of each complete tracing.
- d Each montage recorded should include eyes open as well as closed.
- e There should be 2–3 minutes of hyperventilation.
- f Photic stimulation should be carried out in a darkened room with at least 10 exposures between 1 and 30 Hz.
- g A minimum of 20 minutes of recording on a 16 channel machine (or equivalent) is required.
- h If a subject falls asleep during the recording, it should be continued through the progressive phases of sleep, with intermittent arousal as appropriate.

#### Interpretation of EEGs.

There has been much discussion regarding the significance of various wave forms, particularly in predicting convulsive episodes. There is general agreement that paroxysmal phenomena (epileptiform or seizure patterns), the photoconvulsive response and spike-and-wave complexes (2–4 Hz, irregular, generalised or focal) are significant. Although such cases appear to be only 0.5% of apparently normal applicants, the published data [indicate] a risk of convulsion exceeding 1% per annum and therefore beyond that acceptable for professional aircrew or solo private pilotage.

### 12.9.2 Definitions

#### Epileptiform pattern.

Interpretive term. Applies to distinctive waves or complexes, distinguished from background activity, and resembling those recorded in a proportion of human subjects suffering from epileptic disorders and in animals rendered epileptic experimentally. Epileptiform patterns include spikes and sharp waves, occurring singly or in bursts lasting at most a few seconds. Comments:

- (1) This term refers to interictal paroxysmal activity and not to seizure patterns.
- (2) The probability of association with clinical epileptic disorders is variable.

#### Seizure pattern.

Phenomenon consisting of repetitive EEG discharges with relatively abrupt onset and termination and characteristic pattern of evolution, lasting at least several seconds. The component waves or complexes vary in form, frequency and topography. They are generally rhythmic and frequently display increasing amplitude and decreasing frequency during the same episode. When focal in onset, they tend to spread subsequently to other areas. Comment: EEG seizure patterns unaccompanied by clinical epileptic manifestations detected by the recordist and/or reported by the patient should be referred to as subclinical'. (cf. epileptiform pattern.)

#### Paroxysm.

Phenomena with abrupt onset, rapid attainment of a maximum and sudden termination, distinguished from background activity. Comment: commonly used to refer to epileptiform patterns and seizure patterns. (cf. epileptiform pattern; seizure pattern, EEG.)

#### Spike.

A transient, clearly distinguished from background activity, with pointed peak at conventional paper speeds and duration from 20 to under 70 ms, i.e. 1/50–1/14 s, approximately. Main component is generally negative relative to other areas. Amplitude is variable.

- Comments:
- (1) EEG spikes should be differentiated from sharp waves, i.e. transients having similar characteristics but longer durations. However, it is well to keep in mind that this distinction is largely arbitrary and serves primarily descriptive purposes. Practically, in ink written EEG records taken at 3 cm/s, spikes occupy 2 mm or less of paper width and sharp waves more than 2 mm.
  - (2) EEG spikes should be held in clear contradistinction to the brief unit spikes recorded from single cells with microelectrode techniques. (cf. sharp wave.)

### **3 Hz spike-and-slow-waves.**

Characteristic paroxysm consisting of a regular sequence of spike-and- slow-wave complexes which:

- (1) repeat at 3–3.5 Hz (measured during the first few seconds of the paroxysm),
- (2) are bilateral in their onset and termination, generalised and usually of maximal amplitude over the frontal areas,
- (3) are approximately synchronous and symmetrical on the two sides of the head throughout the paroxysm. Amplitude is variable but can reach values of 1000  $\mu$ V (1 mV).

### **Photoconvulsive response.**

A generalised discharge of spikes or spike wave activity consistently elicited by intermittent photic stimulation, which is autonomous occurring asynchronously with respect to the stimulus, and self-sustaining outlasting the stimulus train by at least 100 msec.

## **[13 DISORDERS OF THE CSF CIRCULATION**

### **13.1 HYDROCEPHALUS**

Hydrocephalus is defined as an increase in volume of the CSF in association with dilatation of the cerebral ventricles.

#### **13.1.1 Clinical features**

##### *Raised intracranial pressure causing*

- Headache, often worse in the early morning.
- Nausea and vomiting.
- Blurred vision, occasionally.
- Diplopia, due to VIth nerve palsy may occur.
- Papilloedema sometimes; its absence does not exclude raised intracranial pressure.
- Optic atrophy with progressive visual loss and poor pupillary reaction to light: a late complication of chronic papilloedema due to raised intracranial pressure.
- Upgaze paresis.
- Increasing unsteadiness of gait occurs, culminating in frequent falls due to a combination of ataxia, spasticity and dyspraxia of gait.
- Urgency of micturition and eventual incontinence

### **13.2 NORMAL PRESSURE HYDROCEPHALUS (NPH)**

A chronic hydrodynamic hydrocephalus that occurs in adults and is associated with a delay in the circulation or absorption of CSF and progressive neurological deficit comprising Hakim's triad of gait apraxia, urinary incontinence, and progressive dementia.

### **13.3 AEROMEDICAL STATUS**

These applicants will be unfit for flying duties.]

## **14 INFECTIONS OF THE NERVOUS SYSTEM**

### **14.1 MENINGITIS AND ENCEPHALITIS**

#### **14.1.1 ACUTE PYOGENIC (BACTERIAL) MENINGITIS**

Fever, headache, meningismus (neck stiffness), and signs of cerebral dysfunction (confusion, delirium, vomiting or declining consciousness) are found in about 85% of patients at presentation. Cranial nerve palsies, particularly nerves III, IV, VI and VII (30% of cases). Epileptic seizures (30%) or focal neurological signs (10–20% of cases), due to inflammation and thrombosis of cortical arteries and veins and venous sinuses (causing cerebral infarction), or subdural effusion.

#### **14.1.2 SUBACUTE AND CHRONIC MENINGITIS AND/OR ENCEPHALITIS**

A syndrome characterized by various combinations of fever, headache, lethargy, stiff neck, confusion, nausea, and vomiting with accompanying CSF pleocytosis, of greater than 4 weeks duration.

#### **14.1.3 TUBERCULOUS MENINGO-ENCEPHALITIS**

Neurological impairments persist in 20–30% of survivors, most commonly cognitive dysfunction, epileptic seizures, visual and oculomotor disorders, deafness and hemiparesis.

#### **14.1.4 ACUTE ASEPTIC MENINGITIS**

Prognosis determined by the underlying cause. Most cases do not progress; resolution begins within a few days and is complete within 2 weeks in most patients. A few will have persistent malaise and myalgia for some weeks.

#### **14.1.5 VIRAL ENCEPHALITIS**

At 10 year follow-up: almost half the survivors have motor dysfunction and educational dysfunction; well over a third have neurological dysfunction, and almost a fifth have behavioural, self-care and sensory dysfunction.

#### **14.1.6 Aeromedical status**

All applicants diagnosed with meningitis should not engage in flight duties for six months. Return to flight duties depends on the nature of the infecting agent or cause of meningitis, eg, viral, bacterial or fungal, and the degree of recovery of resultant deficit and risk of development of epilepsy or hydrocephalus.

### **14.2 INTRACRANIAL ABSCESS**

The usual presenting features are the subacute onset and progressive evolution of:

- Fever.
- Headache.
- Lethargy and malaise.
- Seizures.
- Focal neurological signs.
- Symptoms and signs of raised intracranial pressure. Epilepsy is a complication in more than 50% of survivors.

#### **14.2.1 Aeromedical status**

Assessment is based on the underlying cause and whether the lesion is:

- Supratentorial, in which case the risk of epilepsy and the degree of deficit must be considered, or
- Infratentorial, where the nature and degree of deficit must be considered.

### **14.3 NEUROSYPHILIS**

Symptoms include headache, nausea and vomiting, neck stiffness, seizures and changes in mental status; patients are often afebrile.

- Ocular or cranial nerve abnormalities, especially VII and VIII, may occur due to involvement at the base of the brain.
- Argyll Robertson pupils.

Antimicrobial therapy can cure meningovascular syphilis and arrest tabes dorsalis, but lightning pains and fixed neurological deficits are likely to remain.

#### 14.3.1 **Aeromedical status**

Return to flight duties depends on the degree of recovery of the resultant deficit.

#### 14.4 **HUMAN IMMUNODEFICIENCY VIRUS (HIV)-ASSOCIATED COGNITIVE/MOTOR COMPLEX (HIV-CMC)**

A distinct neurological syndrome of subcortical dementia characterised by slowness and imprecision of cognition and motor control, and called the AIDS dementia complex or HIV-1-associated cognitive/motor complex. It is the most important 'primary' neurological complication of HIV infection. Progressive, may be insidious or rapid.]

##### [14.4.1] **Aeromedical status**

Once symptoms of the AIDS-related complex have appeared a temporarily unfit assessment would appear inevitable as, despite remissions, the usual course is of progressive deterioration. The psychological trauma of HIV sero positivity is major and formal psychiatric opinion may be necessary before any return to flying can be considered. More recent publications would indicate that damage to the individual immune response can be staged (see Chapter on Sexually Transmitted Diseases and Other Infections), therefore making assessment somewhat easier. This assessment applies to Class 2. Class 2 'OSL' may be appropriate if immune staging not available.

#### [15] **INFLAMMATORY DISORDERS OF THE NERVOUS SYSTEM**

##### [15.1] **MULTIPLE SCLEROSIS**

About two-thirds of cases of MS have their onset between 20 and 40 years of age. Of the remainder, most cases begin before the age of 20; in a smaller number, the disease appears to develop in late adult life (late fifties and sixties). Episodic neurological symptoms, often with full recovery, give rise to suspicion of multiple or disseminated sclerosis. Any part of the central nervous system may be affected. Weakness or numbness, sometimes both, in one or more limbs is the initial symptom in about half the patients. In about 25 percent of all MS patients (and in a larger proportion of children), the initial manifestation is an episode of optic neuritis. About half of patients with optic neuritis recover completely, and most of the remaining ones improve significantly. The main point to be made here is that one-half or more of adult patients who present with optic neuritis will eventually develop other signs of MS. Investigations showed that MS developed in 74 percent of women and 34 percent of men by the 15th year after onset of visual loss; the risk is considerably lower (22 percent at 10 years) if the cranial MRI fails to reveal demyelinating lesions. Other initial symptom of MS is acute myelitis. The cumulative probability of developing MS after 2 years is similar after either optic neuritis or transverse myelitis. There are no screening tests for MS but a family history does increase the risk. The duration of the disease is exceedingly variable. A small number of patients die within several months or years of the onset, but the average duration is in excess of 30 years. A relapsing and remitting course occurs in about 80% of patients. Relapse may occur at any time. The average relapse rate is about 0.5 attacks per year but is very variable. A chronic progressive course occurs from onset in the other 20%, particularly if onset occurs after 40 years of age with spastic paraparesis due to spinal cord dysfunction. These patients also tend to have a worse prognosis.

### **[15.1.1] Aeromedical status**

Initial applicants with an established history must therefore [be assessed as unfit. At revalidation / renewal applicants may be assessed as fit with a multi-pilot (Class 1 'OML') of safety pilot (Class 2 'OSL') limitation]. Any neurological event of any note requires specialist neurological assessment.

If multiple sclerosis is considered a strong diagnostic possibility investigation should include cerebrospinal fluid (protein bands) MR scans and evoked responses. [The mean of symptom recurrence is approximately four years with only 5% being sudden and 20% severe. The in-flight risk is therefore small (less than 1% per annum or one in 10-7 flying hours)]. Should the probability [of a multiple sclerosis] remain high, but symptoms are fully recovered an individual may be [assessed as fit with a multi-pilot (Class 1 'OML') of safety pilot (Class 2 'OSL') limitation] after six months, [requiring] a six monthly review:

- a ophthalmologically to look for colour contrast acuity phosgenes, after images and post fixation blindness;
- b operationally (simulator) to assess attention overload and judgement.

Any individual who is left with a neurological deficit after an exacerbation must be [assessed as] unfit. A similar assessment is appropriate for Class 2 'OSL'.

[In all cases, [the] assessment depends upon:

- Nature of symptoms
- Time between exacerbations
- Residual deficit
- Likelihood of sudden incapacitation
- Activity of the disease.

A flight test may be necessary to determine the effect of any residual deficit.]

## **16 INHERITED METABOLIC DISEASES OF THE NERVOUS SYSTEM OF ADULT ONSET**

### **16.1 WILSON'S DISEASE**

Pseudosclerotic, with tremor of the limbs (postural and intention) that closely resembles that seen in multiple sclerosis and which can be severe enough to be described as 'wing-beating', titubation of the head, incoordination, limb ataxia, and dysarthria

### **16.2 MITOCHONDRIAL DISEASES**

Generalized seizures. Myoclonus: spontaneous and stimulus-sensitive. Ataxia

### **16.3 ADRENOLEUCODYSTROPHY**

Pseudobulbar palsy: dysarthria; dysphagia; quadriparesis; emotionalism. Dementia (i.e. progressive learning difficulties and decline in scholastic performance). Personality change.

### **16.4 AEROMEDICAL STATUS**

These applicants should assessed as unfit.

## **17 MONONEUROPATHIES**

### **17.1 RADIAL NEUROPATHY**

Partial lesions tend to recover spontaneously and over about 6-8 weeks.

## 17.2 **CARPAL TUNNEL SYNDROME**

Paraesthesia (e.g. tingling), with or without numbness and pain, involving the palmar surface of the hand (particularly the thumb, index, middle and ring fingers innervated by the median nerve) and often extending proximally into the forearm, arm and even neck. The symptoms are frequently worse at night weeks.

## 17.3 **ULNAR NEUROPATHY**

Partial lesions tend to recover spontaneously and over about 6–8 weeks. Severe ulnar neuropathy caused by compression at the elbow may take 6–12 months after operation to recover, and may not recover at all.

## 17.4 **SCIATIC NEUROPATHY**

Wasting and weakness of knee flexion and all movements of the foot and toes.  
Diminished sensation of the posterior thigh and calf.

## 17.5 **TIBIAL NEUROPATHY**

Wasting and weakness of plantar flexion and inversion and toe flexion. Diminished sensation of the heel, sole of the foot and dorsal aspect of the toes.

## 17.6 **PERONEAL NEUROPATHY**

Foot drop Altered sensation (numbness or paraesthesia) of the lateral part of the lower leg and dorsum of the foot

## 17.7 **Aeromedical status**

These disorders are assessed on the basis of the nature and degree of deficit.

## 18 **MOVEMENT DISORDERS**

### 18.1 **DYSTONIA**

A syndrome of intermittent or continuous sustained muscle contractions, frequently causing twisting and repetitive movements or abnormal postures of virtually any part of the body. About 1 in 20 (5%) patients with any form of idiopathic dystonia may experience a spontaneous improvement or even resolution.

### 18.2 **ESSENTIAL TREMOR**

A low frequency postural tremor which is absent at rest and not associated with the clinical signs of parkinsonism or other neurological deficits. ET is slowly progressive but seldom becomes severe.

### 18.3 **CHOREA**

Involuntary, abrupt, irregular, arrhythmic and purposeless movements of variable amplitude and usually involving the face, hands and feet, which flow randomly from one body part to another. The prognosis depends on the cause. Hemichorea and hemiballismus due to stroke usually resolve within a few weeks or months.

#### 18.4 **MYOCLONUS**

Sudden, very brief, shock-like involuntary movements caused by muscular contractions (positive myoclonus) or inhibitions (negative myoclonus). Myoclonus is a primarily descriptive term; it is not a diagnosis. Depends on the cause; myoclonus may occur as a transient or persistent phenomenon in many conditions such as viral encephalitis, suppurative meningitis, intoxications with strychnine and tetanus, and metabolic disorders such as uraemia and anoxic encephalopathy.

#### 18.5 **AEROMEDICAL STATUS**

These disorders are assessed on the basis of the nature and degree of deficit.

### 19 **MUSCLE DISORDERS**

#### 19.1 **MYOTONIC DYSTROPHY**

The most common form of muscular dystrophy in adults. Skeletal muscle wasting and weakness Sudden death is well recognized, and may be due to heart block or arrhythmia.

##### 19.1.1 **Aeromedical status**

These applicants should be unfit for flying duties.

#### 19.2 **DERMATOMYOSITIS**

An idiopathic inflammatory myopathy with characteristic cutaneous manifestations. Initial symptoms include subacute onset of myalgias, fatigue and weakness, manifested as difficulty climbing stairs, raising the arms for actions such as shaving or brushing hair, rising from a squatting or sitting position, or a combination of these features. If treated early, most patients will respond well, with many showing full recovery of muscle function. In most cases the disease will burn itself out, although this may take many years during which time treatment has to be continued.

##### 19.2.1 **Aeromedical status**

This disorder is assessed on the basis of the nature and degree of deficit.

#### 19.3 **POLYMYOSITIS**

An acquired inflammatory myopathy characterized by progressive muscle weakness and the presence of inflammatory infiltrates in muscle. Slow onset (weeks to months). Usually symmetrical weakness of proximal limb muscles, typically involving the pelvic more than the shoulder girdle, occasionally with pain and muscle tenderness. Weight loss, neck weakness, dysphagia and voice change are common. The response is less favourable than in dermatomyositis, particularly in those with a long history at presentation. Immunosuppressive therapy usually prevents further progression but significant improvement may not occur.

##### 19.3.1 **Aeromedical status**

These applicants should be unfit for flying duties.]

### [20] **NEUROMUSCULAR JUNCTION DISORDERS**

#### [20.1] **MYASTHENIA GRAVIS**

The clinical hallmarks are muscular weakness and fatigability. The weakness tends to increase with repeated activity and improves with rest. Weakness usually occurs in a characteristic distribution. The eyelid and extraocular muscles are the first muscles to be involved in about 65% of patients, and are affected at some stage of the disorder in >90% of patients, causing ptosis and diplopia, which are typically asymmetrical. Remission or substantial improvement can be expected in 80% of patients and most patients lead normal lives but take immunosuppressive medication indefinitely.

**[20.1.1] Aeromedical status**

These applicants should be unfit for flying duties.

**[21 NUTRITIONAL DEFICIENCY AND THE NERVOUS SYSTEM**

**21.1 WERNICKE–KORSAKOFF SYNDROME**

Wernicke's disease (thiamine deficient encephalopathy) is a disorder [characterised] by rather abrupt onset of any combination of nystagmus, gait ataxia, conjugate gaze palsy and mental confusion in association with nutritional deficiency, especially due to alcoholism. Korsakoff's psychosis is a mental disorder, also associated with alcoholism and malnutrition, in which retentive memory is enormously impaired due to a defect in learning and memory, in an otherwise responsive patient. The Wernicke–Korsakoff syndrome is a symptom complex comprising the manifestations of both Wernicke's disease and the Korsakoff amnesic state.

**21.1.1 Aeromedical status**

These applicants should be unfit for flying duties.

**21.2 VITAMIN B12 DEFICIENCY**

Vitamin B12 deficiency is a nutritional disorder of the nervous system that may be characterized by a symmetrical, distal, predominantly sensory peripheral neuropathy due to axonal degeneration, autonomic neuropathy, subacute combined degeneration of the spinal cord (or combined systems disease), optic neuropathy, dementia and other disturbances of higher mental function. The most important factor influencing the neurological response to treatment is the duration of symptoms before treatment is started. If treatment is given early enough, it may not only prevent progression but also reverse some neurological symptoms and signs besides paraesthesia in the feet and optic atrophy.

**21.2.1 Aeromedical status**

This disorder is assessed on the basis of the nature and degree of deficit.]

**22 DISEASES OF THE PERIPHERAL NERVE**

**22.1 PERIPHERAL NEUROPATHY**

Numbness and tingling, usually beginning in the feet (supplied by the longest nerves) are common symptoms of patients with sensory axonal neuropathies, particularly those involving damage to the cell body (e.g. vitamin B12 deficiency). Pain beneath the sole, and a feeling as if the socks are ruffled are other common symptoms. Upper limb symptoms, if present, generally include difficulty picking up small objects such as pins, numbness or a sandpaper feeling on the fingers.

**22.2.1 Aeromedical status**

These disorders are assessed on the basis of the nature and degree of deficit.

## **22.2 HEREDITARY NEUROPATHIES**

### **22.2.1 Charcot–Marie–Tooth disease**

Progressive, predominantly distal muscle wasting and weakness involves mainly the legs. Depending on the type, the disease has the potential to cause significant disability if ignored and prevention measures are not initiated early and maintained. The level of disability therefore depends on how early the patient is diagnosed and whether there has been consistent use of splints and compliance with exercise.

#### **22.2.1.1 Aeromedical status**

These applicants should be unfit for flying duties.

## **22.3 GUILLAIN–BARRÉ SYNDROME**

Progressive symmetrical weakness of the limbs which develops acutely (within days) or subacutely (up to 4 weeks), and progresses over a period of 1–8 weeks in an ascending fashion (caudal to rostral), reaching a plateau, and then spontaneously resolving. Paraesthesiae in the hands and feet: not as prominent as motor signs. The interval from onset to peak disability may vary from hours to weeks. About 30% reach their maximum deficit within 7 days; others progress for up to 4 weeks. About 60% of cases are unable to walk at the height of their illness. Good recovery (includes paraesthesiae, mild weakness): 80%. Unsteady gait with or without orthosis: 5% Walk with callipers: 5%. Wheelchair-bound: 3%. Chronic or relapsing course: 3%. Mortality: 5%.

#### **22.3.1 Aeromedical status**

These disorders are assessed on the basis of the nature and degree of deficit.

## **22.4 CHRONIC INFLAMMATORY DEMYELINATING POLYNEUROPATHY (CIDP)**

Typically subacute onset and progressive (over >8 weeks) asymmetrical weakness and/or numbness of the distal and proximal limbs, with pain, sensory ataxia, and areflexia. About 80% of patients respond to treatment, which needs to be continued for many years. About 13% of patients deteriorate sufficiently to become permanently dependent, bed-bound or chair-bound. About 87% of prevalent patients are able to walk without walking aids or other assistance. About half of patients have a relapsing remitting course.

#### **22.4.1 Aeromedical status**

These applicants should be unfit for flying duties.

## **22.5 DIABETIC NEUROPATHY**

May be acute, but more commonly insidious. Distal symmetrical predominantly sensory (and motor) loss characterized by sensory impairment in a glove and stocking distribution and distal motor weakness. The sequelae of longstanding severe distal sensory loss, such as neuropathic joints, may be present. Autonomic neuropathy. The cranial nerves III and VII are affected particularly. Poor glycaemic control and low plasma concentrations of insulin independent of concentrations of glucose are associated with increased risk of development and progression of neuropathy. Autonomic neuropathy in diabetes probably carries a poor prognosis (i.e. increased risk of death).

#### **22.5.1 Aeromedical status**

These applicants should be assessed as unfit.

## **23 SPINAL CORD DISEASES**

### **23.1 SPINAL MUSCULAR ATROPHY (SMA)**

SMA's are a group of common inherited disorders characterized by degeneration of lower motor neurones, leading to progressive paralysis with muscular atrophy.]

#### 23.1.1 **Aeromedical status**

These applicants should be assessed as unfit.]

### [23.2] **MOTOR NEURONE DISEASES**

Motor neurone diseases are a heterogeneous group of inherited and sporadic disorders of upper and lower motor neurones which lead to progressive weakness of bulbar, limb, thoracic, and abdominal muscles with relative sparing of oculomotor muscles and sphincter function.

#### [23.2.1 **Aeromedical status**

These applicants should be assessed as unfit.]

### [23.3] **SYRINGOMYELIA**

A chronic, progressive, degenerative disorder of the spinal cord, which causes progressive neurological symptoms, usually brachial amyotrophy and segmental dissociated sensory loss, as it expands. Usually slowly progressive and ultimately severely disabling but some patients experience a stepwise deterioration, and others 'plateau' and do not progress.

#### 23.3.1 **Aeromedical status**

These applicants should be assessed as unfit.]

### [23.4] **CERVICAL SPONDYLOTIC MYELOPATHY AND RADICULOPATHY**

A condition in which the spinal cord (myelopathy) and/or nerve roots (radiculopathy) are damaged, either directly by traumatic compression and abnormal movement, or indirectly by ischaemia due to arterial compression, venous stasis, or other consequences of the proliferative bony changes that characterize spondylosis.

- Neck pain in some.
- Numb, clumsy hands

The natural history is not well known because most patients undergo some form of surgical treatment. Spontaneous

regression and complete remission is unusual. About one third of patients experience a recurrence during a median

time of follow-up of 5 years.

### [23.5] **ACUTE 'SLIPPED DISC'**

Back pain with or without radiculomyelopathy. Acute neck and low back pain, in the absence of tumour and other serious underlying disease, usually resolves rapidly within 4–6 weeks. Lumbar discectomy is successful in 80–90% of patients, if properly selected. Patients with severe cauda equina syndrome due to massive midline disc herniation have a guarded prognosis for neurological recovery, even with prompt disc removal and neural decompression.

### [23.6] **SPINAL EPIDURAL HAEMATOMA**

Sudden and severe neck or back pain, followed in minutes to hours by progressive motor, sensory and sphincteric disturbances referable to radicular spinal cord or cauda equina origin.]

### 23.7 SPINAL EPIDURAL ABSCESS

Subacute onset. Systemic upset: fever, headache, malaise. Back pain and tenderness to palpation/percussion: local and severe. Neck/spine rigidity. Nerve root pain.

### 23.8 SPINAL CORD INFARCTION

Sudden onset. Flaccid paraparesis and weakness of myotomes below the level of the lesion. Recovery is impeded by autonomic dysfunction, pain, paraesthesia, depression. Poor prognosis if extensive deficits are present without initial improvement.

### 23.9 ACUTE TRANSVERSE MYELITIS

An acute loss of sensory, motor and bladder function due to inflammation of a transverse ( $\pm$  rostral-caudal) segment of the spinal cord. Extremely variable: about two-thirds recover to variable degrees and one-third remain paraplegic.

### 23.10 SPINAL CORD TUMOURS

Prognosis depends on the underlying cause and duration and degree of spinal cord and nerve root compression/ infiltration. In contrast to brain tumours, many spinal tumours are benign and produce their effects mainly by compression of the spinal cord rather than by invasion.

### 23.11 Aeromedical status

These disorders are assessed on the basis of the nature and degree of deficit. A simple test of the type of movement

and loads involved is the ability to step up onto a kitchen chair or wooden box 40 cms high. If this can be completed without pain, the pilot is considered fit for flying. Surgical procedures such as laminectomy require a similar degree of recovery before clearance to fly and will usually remain unfit for about 3 months

## 24 TRAUMATIC DISEASES OF THE NERVOUS SYSTEM

Head injury with associated brain damage is common but varies considerably in severity and extent. The basic problem in craniocerebral trauma is at once both simple and complex: simple because there is usually no difficulty in determining causation—namely, a blow to the head—complex because of a number of delayed effects that may complicate the injury. Brain damage occurs following penetration/laceration, deformation and accelerations. Secondary complications may occur subsequent to the loss of cerebrovascular autoregulation and formation of intracranial haematoma or traumatic subarachnoid haemorrhage. Further difficulties may ensue associated with infection, surgery or CSF fistulae. Long term one may see the development of post traumatic epilepsy and hydrocephalus. The aviation neurological assessment of head injury should consider firstly whether any significant CNS injury has occurred. A scalp laceration may appear severe but without loss of consciousness is unlikely to be significant, conversely any scalp injury may be associated with an alteration of consciousness and this possibility should be carefully explored in the history.

The severity of head injury should be assessed by:

- i the presence of demonstrable neurological deficit;
- ii duration of pre and post traumatic amnesia (the use of strong narcotic analgesics may produce amnesia which is not brain injury related);
- iii the presence of cranial fracture and any associated meningeal rupture.

### 24.1 SUBDURAL HAEMATOMA

Haemorrhage into the subdural space, usually caused by rupture of bridging veins which pass from the pia-arachnoid over the brain to a dural sinus. Usually initial loss of consciousness (from the head trauma),

which may persist or be followed by a short lucid interval of several hours where consciousness is regained, followed by progressive headache, confusion and deterioration of conscious state. Lateralizing neurological signs may be present. 85–90% of patients make a good functional recovery with appropriate treatment.

#### 24.1.1 Aeromedical status

##### **Mild head injury**

- Loss of Consciousness/Post Traumatic Amnesia (LOC) / PTA < 30 min
- No neurological deficit
- No compounding factors (skull #, vertigo, headache)

It is recommended that all applicants who sustain a head injury and have impaired consciousness (no LOC) be grounded for at least 7 days, as even they may develop post-traumatic epilepsy. Those who have even a fleeting LOC and amnesia should be assessed as temporarily unfit for a period of 6 weeks. These applicants tend to recover fully, and may then fly without limitations.

##### **Moderate head injury**

- LOC / PTA >30 min but <24h
- Focal neurological deficits
- Skull base #
- Surgical penetration of the dura

Following a moderate head injury (particularly if the duration of post-traumatic amnesia is >12h) the applicant should be assessed as temporarily unfit for a period of 2 years (this decision is usually made/confirmed by the AMS.) After 2 years, the applicant may apply for fit assessment. The examination should preferably be coordinated by the designated body or institution and a series of special investigations are required (e.g. sleep deprivation / photostimulation EEG, CT / MRI scans of the brain, neuropsychological evaluation etc.). In addition to these special investigations, a practical flight test is usually required. Pilots may then be assessed as fit, assessed as fit with limitations, or unfit by the AMS.

##### **Severe head injury:**

- LOC / PTA 1 to 7 days
- Neurological/intellectual impairment
- Traumatic penetration of the dura
- Depressed skull #
- Traumatic intracranial haemorrhage
- EEG abnormalities persisting for >2 years

These applicants will most likely be assessed as unfit. Exceptional cases with a full clinical recovery may be considered for a fit assessment after 5 years following rigorous assessment (with several specialist reports and special investigations) co-ordinated from the designated body or institution.

##### **Very severe head injury**

- LOC / PTA >7 days
  - Missile penetration of the brain
  - Brain abscess
  - Debilitating neurological deficit
- Very severe head injury is disqualifying.

#### 24.2 POSTTRAUMATIC EPILEPSY

Epilepsy is the most common delayed sequela of craniocerebral trauma, with an overall incidence of about 5 % in patients with closed head injuries and 50 % in those who had sustained a compound skull fracture and wound of the brain. The basis is nearly always a contusion or laceration of the cortex. As one might expect, the risk of developing posttraumatic epilepsy is also related to the overall severity of the closed head injury. The risk of seizures after severe head injury (loss of consciousness or amnesia for more than 24 h, including subdural hematoma and brain contusion) was 7 % within 1 year and 11.5 % in 5 years. If the injury was only moderate (unconsciousness or amnesia for 30 min to 24 h or causing only a skull fracture), the risk fell to 0.7 and 1.6 percent, respectively. After mild injury (loss of consciousness or

amnesia of less than 30 min), the incidence of seizures was not significantly greater than in the general population.

The interval between the head injury and the first seizure varies greatly. Some 4 to 5 % of hospitalized head-injured individuals are said to have one or more seizures within the first week of their injury (*early epilepsy*). The immediate seizures have a good prognosis and we tend not to treat them as if they represented epilepsy; on the other hand, late seizures are significantly more frequent in patients who had experienced epilepsy in the first week after injury (not including the convulsions of the immediate injury)

In medical writings, the term *posttraumatic epilepsy* usually refers to late epilepsy, i.e., to seizures that develop several weeks or months after the head injury (1 to 3 months in most cases). Approximately 6 months after injury, half the patients who will develop epilepsy have had their first episode; by the end of 2 years, the figure rises to 80 percent. The longer the interval, the less certain one is of its relation to the traumatic incident. Data derived from a 15-year study of military personnel with severe (penetrating) brain wounds indicate that patients who escape seizures for 1 year after injury can be 75 % certain of remaining seizure-free; patients without seizures for 2 years can be 90 % certain; and for 3 years, 95 % certain. For the less severely injured (mainly closed head injuries), the corresponding times are 2 to 6 months, 12 to 17 months, and 21 to 25 months.

#### 24.2.1 Aeromedical status

The diagnosis of epilepsy is usually made after the second convulsion, but the applicant is unfit to fly after the first convulsion. If there are 3 or more convulsions in the first year, the incidence of persistent epilepsy is as high as 85%. After a head injury, the applicant is seen after 7 days, one month, and then 3 monthly for 2 years to observe for post-traumatic epilepsy and the posttraumatic syndrome. If an applicant does develop convulsions, he / she is seen weekly until they are controlled.

## 25 TUMOURS OF THE CENTRAL NERVOUS SYSTEM

**Benign tumours:** complete removal achieves a cure. Even with incomplete removal, prolonged survival is possible with repeated operations and adjuvant therapy.

**Malignant tumours:** prognosis is poor, despite surgery and radiotherapy; therefore, palliation of distressing symptoms is often the goal of therapy.

- Anaplastic astrocytoma: median survival time with radiotherapy and chemotherapy: 36–48 months.
- Mixed anaplastic astrocytoma and glioblastoma: median survival time with brain irradiation: 9–11 months; 10% of patients with glioblastoma survive 2 years.
- Metastases from the bronchus, gastrointestinal tract and melanoma have a worse prognosis than breast or renal metastases to the brain.

#### **Favourable prognostic factors for adult supratentorial tumours**

- Epileptic seizure as the initial presenting symptom.
- Young age (<45 years).
- Absence of focal neurological signs (i.e. hemiparesis).
- Absence of mental signs (confusion, altered awareness, personality change).
- Absence of contrast enhancement on cranial CT scan.
- Presence of cystic change on CT (a circular low density area before enhancement, with clear cut margin).
- Presence of diffuse low density on CT (diffuse, poorly demarcated low density without contrast enhancement).
- Presence of calcification on CT.

## 25.1 Aeromedical status

Tumours of the Central Nervous System are disqualifying. Potential exceptions are:

- **Supratentorial meningioma**
  - a These applicants should be assessed as temporarily unfit upon diagnosis.
  - b Following successful surgery, they must be asymptomatic, and have no neurological deficit for a period of 2 years before being considered for re-certification by the AMS.
  - c They will require a MR scan of the brain that shows no tumour, and an oncologist's report which states that:
    - i) The applicant is in remission.
    - ii) That he/she never had convulsions.
  - d The AMS may assess the applicant as fit, an annual medical examination (including specialist's report) is required.
- **Infratentorial meningioma, acoustic neuroma, pituitary adenoma, and benign extraaxial tumours:**
  - a Require the same conditions as a supratentorial meningioma.
  - b Except that the stipulated minimum period before a fit assessment is considered is 1 year.
- **Pseudotumour Cerebri:**  
 [These applicants are assessed as temporarily unfit for a period of at least 6 months, until they have been headache free, and have had normal visual fields.]

Whenever [a fit assessment] is considered, the AMS [has to consider, whether any eventual] neurological deficit is compatible with [flying] (medical flight test), and [whether] the risk of epilepsy is [only] minimal (less than 1% per annum). [A fit assessment] should [require a] multi-pilot (Class 1 'OML') [limitation] for an extended period. Reference should also be made to the oncology chapter. This assessment also applies to Class 2

## 26 VASCULAR DISEASES OF THE NERVOUS SYSTEM

Vascular lesions cause ischaemia or infarction with a variable degree of brain damage and, although the effects may appear reversible, there may be long term sequelae.

Lesions may be:

- a Haemorrhagic (aneurysms, arteriovenous malformation (AVMs) and spontaneous (intracerebral) bleeds.
- b Vasospastic (as in the initial phase of migraine).
- c Vaso-occlusive (embolism, thrombosis or vascular distortion).

Clinically all such conditions are potentially incapacitating, such incapacitation may or may not be reversible and may recur unpredictably.

### Major types of cerebrovascular diseases and their frequency

	HARVARD STROKE	
	SERIES <sup>a</sup> (756 SUCCESSIVE CASES)	BCH AUTOPSY SERIES <sup>b</sup> (179 CASES)
Atherosclerotic thrombosis	244 (32%)	21 (12%)
Lacunae	129 (18%)	34 (18.5%)
Embolism	244 (32%)	57 (32%)
Hypertensive hemorrhage	84 (11%)	28 (15.5%)
Ruptured aneurysms and vascular malformations	55 (7%)	8 (4.5%)
Indeterminate		17 (9.5%)
Other <sup>c</sup>		14 (8%)

[As a group, TIA patients have an increased risk of stroke and other serious vascular events of about 8–10% per year. The risk of stroke is about 4–5% in the first month, 12% in the first year, 29% over 5 years. The risk of a coronary event is about 3% per year.

The average risk of recurrent stroke in patients with a first ever stroke is about:

- 13% in the first year (15 times the risk in the general population).
- 4% per year for subsequent years, so that by 5 years, about 30% will have suffered a recurrent stroke.

#### *Epileptic seizures*

- 2% of patients with a first-ever stroke have a seizure at stroke onset.
- 11% have a later seizure in the first 5 years of follow-up, but nearly half of these patients have only one seizure.
- The risk of seizures is increased in survivors of intracerebral and subarachnoid haemorrhage, and total anterior circulation infarction.
- Stroke survivors who are independent at 1 month after stroke have a very low risk of future seizures. Hence, stroke patients who are functionally competent may return to driving after 30 days.

### 26.1 INTRACRANIAL HAEMORRHAGE

**Intracranial hemorrhage** is the third most frequent cause of stroke. The frequency of seizures after each type of hemorrhage has not been established, but it is lower than for ischemic strokes. In patients who survive there can be a surprising degree of restoration of function, since, in contrast to infarction, the hemorrhage has to some extent pushed brain tissue aside rather than destroyed it. Function may return very slowly, however, because extravasated blood takes time to be removed from the tissues. About 7% of 30-day survivors suffer a recurrent stroke in the first year, of which at least 25% are haemorrhagic. About 70% of recurrences are fatal.]

#### [26.1.1 Spontaneous Subarachnoid Haemorrhage]

Spontaneous Subarachnoid Haemorrhage is associated with:

- i aneurysm (80%)
- ii arteriovenous malformation (15%)
- iii unidentified cause (<5%).

Convulsive seizures, usually brief and generalized, occur in 10 to 25 percent of cases. When a diagnosis of (i) or (ii) is confirmed the late epileptic risk would make revalidation extremely unlikely. Surgical repair of aneurysm brings an additional post craniotomy risk of epilepsy, however, where published data demonstrates post-operative criteria for an incapacitation risk <1%, [a fit assessment] may be considered. Patients with the typical clinical picture of spontaneous subarachnoid hemorrhage, in whom an aneurysm or arteriovenous malformation cannot be demonstrated angiographically, have a distinctly better prognosis than those, in whom the lesion can be [demonstrated]. Where no cause is identified and recovery complete, [a fit assessment with a multi-pilot (Class 1 'OML') or safety pilot (Class 2 'OSL') limitation] may be considered in normotensive individuals after 9 months. Class 2 [without limitation] may be considered after 2 years.

#### [26.1.2 Unruptured Intracranial Aneurysms]

Not infrequently, cerebral angiography, MRI, MRA, or CT scanning performed for an unrelated reason, discloses the presence of an unruptured saccular aneurysm. The only clinical feature of significance relative to rupture is aneurysmal size. Studies found an extremely low rate of rupture (aneurysms smaller than 7 mm in diameter: annual risk about 0.1 % yearly; aneurysms between 7 and 10 mm: 0.5 %; lesions between 13 and 24 mm (depending on location): ranging from 0.6 to 3.5 %; aneurysms > 25 mm diameter: up to 10 %; the yearly rates for rupture were higher in all categories if there had been prior bleeding from another site)].

## 26.2 Cerebral decompression sickness]

Cerebral decompression sickness is postulated as the formation of bubbles in nitrogen supersaturated body fluids following a reduction in ambient pressure. Such bubbles may coalesce and produce local symptoms or, if in the blood, circulate throughout the body including the brain. Decompression sickness is rare in normal aircraft operations but should be considered when unpressurised aircraft are flying above 15,000 feet. It occurs at lower cabin altitudes when flying after SCUBA diving. Individuals who have experienced this condition as divers or in previous military flying should be carefully reviewed as permanent damage may be caused by repeated exposure.

### 26.2.1 Aeromedical status

Aviation Medicine Section assesses all cases individually.

## 26.3 TRANSIENT MEMORY LOSS

Loss of memory concerning a period of time (minutes to hours) is not uncommon. Causes include alcohol, epilepsy, migraine, TIA's, certain drugs (e.g. benzodiazepines) and psychiatric disturbances (e.g. psychogenic fugue).

### 26.3.1 Aeromedical status

Applicants must be assessed according to the underlying cause. The vast majority will be assessed as unfit.

## 26.4 GIANT CELL ARTERITIS

A systemic angiitis that involves a wide variety of medium and large arteries, and tends to affect older people (over 50 years) causing two main clinical syndromes: temporal (cranial) arteritis and polymyalgia rheumatica, which respond rapidly to corticosteroid therapy. Ophthalmic GCA starts as a unilateral condition but may become bilateral after days, months, or years. Between one-third and one-half of patients can stop steroids after 2 years. Relapses are most likely during the initial 18 months of treatment and within 1 year of withdrawal of steroids. Patients should be urged to report back immediately if arteritic symptoms occur.

### 26.4.1 Aeromedical status

These disorders are assessed on the basis of the nature and degree of deficit.

## 26.5 CEREBRAL VENOUS THROMBOSIS

The prognosis for recovery of function is generally favourable and much better than in arterial occlusion.

### 26.5.1 Aeromedical status

These disorders are assessed on the basis of the nature and degree of deficit.

## 27 VERTIGO

### Peripheral vertigo (most common)

*Inner ear (semicircular canals, utricle, saccule)*

- Benign paroxysmal positional vertigo (25% of cases).
- Vestibular neuronitis ('viral' labyrinthitis).
- Ménière's disease.
- Benign recurrent vertigo.
- Trauma (including perilymph fistula): head injury.
- Infection: otitis media, syphilis.
- Vascular lesions.

#### *Vestibular nerve*

- Meningitis.
- Acoustic neuroma and other cerebello-pontine angle tumours (usually cause unsteady gait and rarely cause vertigo, particularly if there is no deafness).
- Ototoxins: aminoglycosides, frusemide (cause imbalance rather than vertigo).

#### **Central vertigo**

- Tumours (usually posterior fossa).
- Vertebro-basilar ischaemia (but may also cause infarction of the labyrinth): cerebellar or brainstem infarction.
- Vascular malformation in brainstem (VIII nucleus)
- Multiple sclerosis involving brainstem.
- Trauma to brainstem
- Basilar migraine (may also cause end organ or peripheral involvement).
- Arnold–Chiari malformation.
- Syringobulbia.
- Drugs (alcohol, anti-epileptic drugs, barbiturates).
- Complex partial seizures can cause vertigo but almost always with other more typical symptoms.
- Vertigo is rarely, if ever, due to cervical spondylosis.

Sudden unilateral loss of vestibular function usually causes acute, severe vertigo that persists for hours to days. Even without recovery of the underlying vestibular deficit, resolution of the severe disabling symptoms occurs as a result of equilibration of the tonus in the brainstem vestibular nuclei via the CNS process known as compensation. During this recovery period the spontaneous symptoms resolve, but patients may continue to complain of shortlived episodes of vertigo induced by head or body motion. Persistent motion-induced symptoms following an acute vestibular insult reflect incomplete central compensation. In contrast, recurrent spontaneous episodes of vertigo indicate an unstable vestibular lesion resulting from active underlying pathology. Such conditions demand specific medical or surgical treatment.

#### **27.1 Aeromedical status**

These disorders are assessed on the basis of the nature and degree of deficit.

#### **27.2 BENIGN PAROXYSMAL POSITIONAL VERTIGO**

Episodic rotational vertigo of brief duration induced by head movement. Spontaneous resolution occurs in most cases. Episodes may last only a few days, but some patients can experience recurrent episodes for 2 months or more. In some cases, episodes occur recurrently for more than a year, and in others it can persist chronically.

##### **27.2.1 Aeromedical status**

Assessment is based on the frequency of occurrences, their duration and severity.

#### **27.3 MÉNIÈRE'S DISEASE**

Sudden onset of intense vertigo which may take many hours to resolve. Unilateral tinnitus and decreased hearing associated with a sensation of fullness and increased pressure. Recurrence of attacks is a hallmark of the disease. Many patients experience decremental hearing loss with recurrent episodes. Initially hearing low in the low frequencies is observed. As the disease progresses, high frequency hearing loss is seen

##### **27.3.1 Aeromedical status**

Applicants with these conditions are usually unable to meet the standard for certification, but require individual assessment.]

## **[28] EPISODIC NEUROLOGICAL PROBLEMS**

### **[28.1] EPISODIC IMPAIRMENT OF CONSCIOUS LEVEL FATIGUE**

Episodic Impairment of Conscious Level Fatigue or sleep loss can produce micro-sleep episodes which are immediately resolved by rest. Any other episodes need to be investigated neurologically as the differential diagnosis is wide – a diagnosis needs to be made and the potential risk of recurrence assessed. Narcolepsy, even when treated, is incompatible with flying. A history of recurrent fainting, whether vasovagal or syncopal is unacceptable as precipitating factors may well arise when flying. Hypoglycaemia is a popular diagnosis but rarely proven – if it is, the condition should be disqualifying. Occasionally an episode may follow alcohol withdrawal, under these circumstances, provided any alcohol abuse is treated and the individual remains asymptomatic, [a fit assessment with multi-pilot (Class 1 'OML') limitation] may be considered.

### **[28.2] SLEEP APNOEA SYNDROME**

The sleep apnoea syndrome most commonly affects overweight males, especially between the ages of 40 and 60 years. The syndrome consists of excessive daytime sleepiness and frequent apnoeas during sleep, associated with loud intermittent snoring. Sleep recordings reveal apnoeic episodes in REM and non REM sleep. There may be an absence of respiratory effort with cessation of diaphragmatic movement. The upper airway can remain open even without airflow (central apnoea) or there may be excessive respiratory effort due to [obstruction of the upper airways]. The chronically disturbed nocturnal sleep and hypoxaemia causes excessive daytime sleepiness. This leads to inappropriate and unrefreshing naps, an obvious safety hazard in a pilot who may also have circadian disruption to deal with. The sleep apnoea syndrome evolves gradually and may not be fully described by the sufferer. It should be considered with any presentation of excessive sleepiness which is not improved by a period of undisturbed sleep. Investigation should include respiratory studies and sleep recordings. The condition can be treated but a diagnosis will require flight crew to be assessed [as] temporarily unfit until all aspects of the recovery and treatment can be considered by the AMS.

## **[29] CONGENITAL NEUROLOGICAL PATHOLOGY**

Congenital conditions include hamartoma and arachnoid cysts discovered incidentally and spinal disorganisation including minor degrees of spina bifida. Each case must be considered individually but usually the decision is one of operational competence rather than risk of incapacitation.

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