Essential neurosurgery andrew kaye pdf



Essential Neurosurgery. Second Edition.Edited by andrew h kaye. (Pp 448 £23.50). Published by Churchill Livingstone, Edinburgh 1996. ISBN 0443053472. The second edition of this basic textbook gives a clear and concise introduction to neurosurgery. It is mainly written by one author with contributions from his neurological and neurosurgical colleagues at the Royal Melbourne Hospital. The text is well written, beginning with an excellent chapter on neurological assessment and examination, and proceeding to covering the whole of neurosurgical controversies are well argued with balanced approaches to difficult areas—for example, the treatment of spontaneous intracerebral haematomas. Advances in some treatments such as gene and immunotherapy are well covered, but the sections on other new techniques—for example, endoscopy, image-guidance and interventional radiology are brief. I have no major criticisms, but the chapter on head injury could have had more detail on head injury as a component of multiple head injury (there is no mention alcohol or drugs as indication for admission. Also, the book would benefit from some more recent references. The review of the first edition of the book published in thisJournal in 1991 suggested that the author preferred intracranial work as he expressed less strong opinions regarding spinal surgery. However, I think the second edition has redressed the balance. The author now includes anterior approaches to the thoracic and lumbar spine. The section on AIDS is still brief, and the driving regulations relate to Australian law. Overall, however, although the author admits the book describes practice in the Asia-Pacific rim region, it is equally applicable to European neurosurgery. In summary, I enjoyed reading the book and considered that it gives an accurate and useful guide to neurosurgical management. I would strongly recommend it to medical students, general and orthopaedic surgeons, and junior neurosurgical trainees. Page 2 Dr. Andrew H. Kaye, an Elsevier Author, serves as Director of the Melbourne, and Director of the Melbourne, and Director and Professor of Surgery at the University of Melbourne, and Director of the Melbourne Neuroscience Centre, The Royal Melbourne Hospital. He holds particular research interest in the mechanisms of brain tumor cell invasion, intracellular signaling, and development of gene therapy treatment programs. He is an author of the Elsevier publication Brain Tumors.For more information, visit Skip to content 您是否正在寻找由 作者 Andrew H. Kaye写的Essential Neurosurgery?如果你想买 它,这本书Essential Neurosurgery通常售价是143.83¥。但是,在某些国家/地区,由于供应不足,此书可能已不再可供购买。但是,由作者 Andrew H. Kaye写的Essential Neurosurgery的pdf版本,只需点击以下的下载按钮即可。您将被前往到含有此pdf文件的合作伙伴服务器以进行下载。Preface to the third edition vii Preface to the first edition ix 1 Neurological assessment and examination 1 2 Neurosurgical investigations 14 3 Raised intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial pressure and hydrocephalus 27 4 Head injuries 40 5 Traumatic intracranial p MD FRACP 11 Developmental abnormalities 158 12 Infections of the central nervous system 170 13 Low back pain and leg pain 185 14 Cervical disc disease and cervical spondylosis 197 15 Spinal cord compression 206 16 Spinal injuries 225 17 Peripheral nerve entrapments injuries and tumours 234 18 Facial pain and hemifacial spasm 248 19 Painneurosurgical management 254 20 Movement disorders-neurosurgical aspects 263 21 Epilepsy and its neurosurgical aspects 269 Christine Kilpatrick MD FRACP Index 281 About the AuthorProfessor Andrew H. Kaye, MBBS MD FRACS James Stewart Professor of Surgery and Head of Department of Surgery, The University of Melbourne, Australia Director of Neurosurgery and Director, The Melbourne Neuroscience, which is the official journal of the Neuroscience, which is the official journal of the Neurosurgical Society of Australasia and the Australian Association of Neurologists. Reviews "What it lacks in colour, it more than makes up with some fantastic diagrams, pictures and scans showing the important pathology of a number of core problems. It guides the reader from clinical presentation all the way through to postoperative care. Definitely worth a look." - Black Bag. Bristol Medical School Magazine. Winter 2005 Review quotations from the previous edition 'flowing and well highlighted text keeps the reader interested in the subject' British Journal of Neurosurgery 'an excellent text...well organised and clearly set out' Journal of Neurology, Neurosurgery and Psychiatry You're Reading a Free Preview Pages 15 to 41 are not shown in this preview. You're Reading a Free Preview Pages 58 to 81 are not shown in this preview. You're Reading a Free Preview Pages 90 to 118 are not shown in this preview. You're Reading a Free Preview Pages 130 to 135 are not shown in this preview. You're Reading a Free Preview Pages 12 to 225 are not shown in this preview. You're Reading a Free Preview Pages 154 to 202 are not shown in this preview. You're Reading a Free Preview Pages 154 to 202 are not shown in this preview. You're Reading a Free Preview Pages 154 to 202 are not shown in this preview. You're Reading a Free Preview Pages 154 to 202 are not shown in this preview. 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You're Reading a Free Preview Pages 154 to 202 are not shown in this preview. You're Reading a Free Preview Pages 154 to 202 are not shown in this preview. You're Reading a Free Preview Pages 154 to 202 are not shown in this preview. You're Reading a Free Preview Pages 154 to 202 are not shown in this preview. You're Reading a Free Preview Pages 154 to 202 are not shown in this preview. You're Reading a Free Preview Pages 154 to 202 are not shown in this preview. You're Reading a Free Preview Pages 154 to 202 are not shown in this preview. You're Read Preview Pages 229 to 239 are not shown in this preview. You're Reading a Free Preview Pages 243 is not shown in this preview. You're Reading a Free Preview Pages 247 to 249 are not shown in this preview. You're Reading a Free Preview Pages 247 to 249 are not shown in this preview. in this preview. You're Reading a Free Preview Pages 285 to 287 are not shown in this preview. You're Reading a Free Preview Pages 291 to 306 are not shown in this preview. Dr. Andrew H. Kaye, an Elsevier Author, serves as Director and Professor of Neurosurgery and James Stewart Professor of Surgery at the University of Melbourne, and Director of the Melbourne Neuroscience Centre, The Royal Melbourne Hospital. He holds particular research interest in the mechanisms of brain tumor cell invasion, intracellular signaling, and development of gene therapy treatment programs. He is an author of the Elsevier publication Brain Tumors. For more information, visit Accounting Economics Finance Management Marketing Classics Readers Romance Thriller Skip to main contentDiscover VDOC.PUB Download Embed This document was uploaded by our user. The uploader already confirmed that they had the permission to publish it. If you are author/publisher or own the copyright of this documents, please report to us by using this DMCA report form. Report DMCA Essential Neurosurgery provides a comprehensive introduction to neurosurgery for junior surgical diagnosis and management of the more common central nervous system problems, including an understanding of neurology and the pathological basis of neurological disease. There is also coverage of neurosurgical techniques and postoperative patient management. This new edition brings the text fully up to date and includes many of the biological advances made in the field of neurosurgery that have improved surgical possibilities and patient outcomes. Review quotations from the previous edition 'flowing and well highlighted text keeps the reader interested in the subject' British Journal of Neurosurgery 'an excellent text...well organised and clearly set out' Journal of Neurosurgery and Psychiatry E-Book Content Essential Neurosurgery THIRD EDITION Essential Neurosurgery Andrew H. Kaye MBBS, MD, FRACS James Stewart Professor of Surgery and Head of Department of Surgery, The University of Melbourne Neuroscience Centre, The Royal Melbourne Director, The Melbourne Director, The Melbourne Director, The Melbourne Neuroscience Centre, The Royal Melbourne Director, The Melbourne Director, The Melbourne Neuroscience Centre, The Royal Melbourne Director, The Melbourne Direc Limited © 2005 Andrew Kaye Published by Blackwell Publishing, Inc., 350 Main Street, Malden, Massachusetts 021485020, USA Blackwell Publishing, Inc., 350 Main Street, Malden, Massachusetts 021485020, USA Blackwell Publishing Asia Pty Ltd, 550 Swanston Street, Carlton, Victoria 3053, Australia The right of the Author to be identified as the Author of this Work has been asserted in accordance with the Copyright, Designs and Patents Act 1988. All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, except as permitted by the UK Copyright, Designs and Patents Act 1988, without the prior permission of the publisher. First publisher. 1641-2 1. Nervous system — Surgery. [DNLM: 1. Neurosurgical Procedures. 2. Central Nervous System — surgery. 3. Central Ne British Library Set in 9/12 Palatino by SNP Best-set Typesetter Ltd., Hong Kong Printed and bound in India by Replika Press Pvt., Ltd. Commissioning Editor: Vicki Noyes Development Editor: V paper from mills that operate a sustainable forestry policy, and which has been manufactured from pulp processed using acid-free and elementary chlorine-free practices. Furthermore, the publisher ensures that the text paper and cover board used have met acceptable environmental accreditation standards. Contents Preface to the third edition, vii Preface to the first edition, ix 1 Neurological assessment and examination, 1 2 Neurosurgical investigations, 14 3 Raised intracranial haematomas, 56 6 Brain tumours, 64 7 Benign brain tumours, 93 8 Pituitary tumours, 109 9 Subarachnoid haemorrhage, 125 10 Stroke, 140 Stephen M. Davis MD, FRACP 11 Developmental abnormalities, 158 12 Infections of the central nervous system, 170 13 Low back pain and leg pain, 185 14 Cervical spondylosis, 197 15 Spinal cord compression, 206 16 Spinal injuries, 225 17 Peripheral nerve entrapments, injuries and tumours, 234 18 Facial pain and hemifacial spasm, 248 19 Pain — neurosurgical aspects, 263 21 Epilepsy and its neurosurgical aspects, 263 21 Epilepsy and 26 enabled better imaging of central nervous system disease, understanding of disease processes and the consequent development of rational treatments. Magnetic resonance imaging has now become the standard radiological technique to investigate central nervous system disease, and this has further demystified the diagnostic process in neurosurgery. However, it has entailed a new learning process not only for students, but also for practising clinicians. Magnetic resonance angiography. Improved understanding of the biology of the central nervous system and tumour biology, has led to the introduction of more rational treatment regimes, with improved outcomes. Molecular biology techniques, the introduction of biological therapies including gene therapies including gene therapies including the provide the horizon for the management of a wide range of neurological diseases. Technological advances in the operating theatre have increased the surgical possibilities, particularly combining stereotactic techniques with microneurosurgery. Our patients have benefited considerably from these advances, and over the next two decades biological and technical advances will continue to provide considerably from these advances. Essential Neurosurgery has essentially been based on the first and second editions, but has incorporated many of the advances described. Modern neurosurgical practices still differ considerably in North America and Europe, and despite the 'global village' there continues to be substantive differences in the philosophical approach to the management of clinical problems. The author has described his own practice, which hopefully continues to utilize the best of both systems, as well as incorporating the unique advances and philosophies of the Asia-Pacific rim region. It is not possible to list and acknowledge all the many people who have helped in the preparation of this third edition. However, I particularly acknowledge my neurological and neurosurgical colleagues at The Royal Melbourne Hospital. Stephen Davis and Christine Kilpatrick have again provided chapters on their own areas of expertise. I am very grateful to Nicholas Maartens for his considerable help with chapters on their own areas of expertise. Laidlaw for his assistance with a chapter on Subarachnoid Haemorrhage and Bhadu Kavar for his input into the rewriting of the Spinal Injuries chapter. I would like to especially thank Kate Lagerewskij for the many hours she spent preparing the manuscript and to Helen Harvey at Blackwell Publishing for making this edition possible. As always I am especially grateful to the encouragement and patience of my wife Judy and son Ben. Andrew H. Kaye, Melbourne, 2004 vii Preface to the first edition Clinical neurosciences, particularly neuropathology and neurophysiology. In the past the mystique of neurosurgery has inadvertently prevented both medical trainees and physicians from a proper appreciation of even basic neurosurgical illnesses. The improvements in medical technology have markedly improved the accuracy of the diagnosis, the efficacy of neurosurgical treatment and the range of diseases that can be diagnosed and treated. In particular, the exciting advances in neurosurgery more accessible. This book is intended as an introduction to neurosurgery. It is hoped that it will be useful for physicians in training, neurosurgical trainees and medical students. The book is not intended to be an exhaustive coverage of neurosurgical principles, pathological basis and relevant investigations that form the basis of the diagnosis are emphasised. The neurosurgical management is outlined but the surgical techniques are only briefly mentioned, so that the reader will understand the postoperative problems likely to be encountered in the management of the patient. Modern neurosurgery has evolved principally from North American and European practices and there are often significant differences in the philosophical approach in the management of clinical problems. The author has in general described his own practice, which hopefully utilises the best of both systems. The references have been chosen for their general coverage of the topics, ease of access, historical interest and, in some cases, because they will provide thought provoking alternatives that give a different perspective to the subject. It is not possible to list and acknowledge all the many people who have helped in the preparation of this book, both knowingly and as a result of their influence on my own neurosurgical education but on that of many other Australian neurosurgeons. I particularly acknowledge the help of my neurological and neurosurgical colleagues at the Royal Melbourne Hospital in the preparation of this book. Stephen Davis and Christine Kilpatrick have provided chapters on their own areas of expertise. Professor Brian Tress, Director of Radiology at the Royal Melbourne Hospital, has always been accessible and helpful and I am indebted to him for his expert teaching over many years and for assistance with the details on magnetic resonance is stance with the details on magnetic resonance scans (Figsnance is stance). 7.9, 12.7, 13.5). Professor Colin Masters, Department of Pathology, University of Melbourne Hospital, gave assistance with the pathologist at the Royal Melbourne Hospital, gave assistance with the pathology details and illustrations. Il parix x ticularly acknowledge the assistance of Drs John Laidlaw and Michael Murphy, registrars in neurosurgery, who proof read the manuscript and offered constructive criticism. I thank Sue Dammery for the manuscript and offered constructive criticism. I thank Sue Dammery for the manuscript and offered constructive criticism. I thank Sue Dammery for the manuscript and offered constructive criticism. I thank Sue Dammery for the manuscript and offered constructive criticism. I thank Sue Dammery for the manuscript and offered constructive criticism. I thank Sue Dammery for the manuscript and offered constructive criticism. not have been possible without the guidance and stimulus from Peter Richardson at Churchill Livingstone. I am especially grateful to the encouragement and patience of my wife Judy and son Ben. Andrew H. Kaye, Melbourne, 1990 CHAPTER 1 1 Neurological assessment and examination An accurate neurological assessment is fundamental for the correct management of the patient. The basic aim of the neurological examination is to solve the following four questions: 1 Is there a neurological problem? 2 What is the site of the lesion (or lesions) in the neuroanatomical site and the pathological cause from the history, what is the most likely diagnosis? Answering these four questions in turn will indicate the type of investigation necessary to confirm the diagnosis. The neurological history As in general medicine and surgery the neurological history is the key to the diagnosis. The history involves not only questioning the patient's general manner, mood, posture, gait, facial expression and speech are all vital clues to the final diagnosis. In addition, patients who do not have an organic disease may present in a characteristic manner, particularly with an exaggeration of the complaint. The history and examination commences with observation, and this should begin when first meeting the patient walks into the examination room, sits on the chair, answers questions and climbs on to the examination couch will provide vital clues in the search for the diagnosis. Initially it is important to allow the patient adequate opportunity to explain their symptoms in an unstructured and unprompted manner. neurological symptoms are in essence a verbal examination of the neurological system. It is not just the content of the answer that is important but the way in which the patient responds to the questions. The following is a general classification of neurological symptoms. 1 General neurological symptoms: (a) headache (b) drowsiness (decreased conscious state) (c) vertigo (d) seizures, blackouts. 2 Symptoms of meningismus: (a) headache (b) photophobia (c) neck stiffness (d) vomiting. 3 Symptoms related to the special senses: (a) vision (b) hearing (c) taste (d) smell. 4 Symptoms related to speech and comprehension. 5 Motor symptoms: (a) power (b) coordination. 1 2 6 Sensory symptoms. 7 Cognitive symptoms, e.g. memory. 8 Symptoms of other systems which may relate to diseases of the nervous system. Careful questioning will ascertain the important details concerning each symptom. These include: • The time, mode of onset, progression and duration of the symptom. The symptom of the symptom of the symptom. process. Sudden onset of a neurological disturbance is usually due to a vascular or epileptiform cause; a sudden severe headache is characteristic of subarachnoid haemorrhage whereas a slowly progressive headache is characteristic of subarachnoid haemorrhage whereas a slowly progressive headache is characteristic of subarachnoid haemorrhage whereas a slowly progressive headache is characteristic of subarachnoid haemorrhage whereas a slowly progressive headache is more in keeping with a cerebral tumour. slowly progressive weakness may be due to a compressive or infiltrative cause. • What factors result in alleviation or exacerbation of the symptom? Headache from raised intracranial pressure is characteristically worse in the morning and on coughing and straining. Patients find the hand pain associated with carpal tunnel syndrome is often worse at night and is alleviated by shaking the hand over the side of the bed. • Is there a past history of any similar event? It is often helpful to obtain details of the nature of epileptic seizures should always be obtained from a relative or friend who has witnessed an event. A thorough understanding of the nature of the illness and symptomatology should be undertaken in a systemic fashion in the following order. 1 Mental state. 2 Speech. 3 Cranial nerves. 4 Examination of limbs and trunk: CHAPTER 1 (a) posture (b) wasting (c) tone (d) power (e) reflexes (f) sensation (g) coordination and gait. Mental state Examination of the mental state Examination of the mental state environment of: • conscious state • orientation in time, place and person • memory • emotional state • presence of delusions or hallucinations. A correct assessment of the mental state is essential prior to the evaluation will be undertaken within the context of the patient's mental state. The accurate assessment of conscious state is especially important in state — is the most important neurological sign and indicates major intracranial pathology. As with all neurological symptoms and signs it is essential to obtain an assessment of the progression of the drowsiness by questioning the patient's friends or relatives. A deteriorating conscious state is a neurosurgical emergency. Memory disturbances should be tested formally for both short-term and long-term preservation. Short-term memory should be tested by listing a name, address and type of flower and asking the patient to recall it after 5 minutes. Loss of short-term memory with relative preservation of memory for long-past events is typical of dementia, e.g. Alzheimer's disease. In Korsakoff's psychosis the disturbance of recent memory and disorientation may be so severe that the patient NEUROLOGICAL ASSESSMENT AND EXAMINATION will make up stories to provide a convincing answer to the questions. This is confabulation and is classically associated with alcoholism, although it may rarely be seen as a result of anterior hypothalamic lesions due to trauma or following subarachnoid haemorrhage and vasospasm. Speech disorders: 1 Mutism Mutism is characterized by the patient being alert but making no attempt to speak. It may result from lesions affecting the medial aspect of both frontal lobes, classically occurring as a result of vasospasm following subarachnoid haemorrhage from a ruptured anterior communicating artery aneurysm. Aphonia is said to occur when the patient is able to speak but is unable to produce any volume of sound. It is due to a disturbance of the vocal cords or larynx. If the patient is able to cough normally then it is usually hysterical. Dysarthria is due to impaired coordination of the lips, palate, tongue and larynx and may result from extrapyramidal, brainstem or cerebellar lesions. The volume and content of the speech will be normal but the enunciation will be distorted. Spastic dysarthria. This is due to bilateral upper motor neurone disease due to pseudobulbar palsy, motor neurone disease or brainstem tumours. Ataxic dysarthria is seen in cerebellopontine angle tumours, cerebellar lesions, multiple sclerosis and phenytoin toxicity. 3 Dysarthria may result from lesions of the lower motor neurones and the muscles, such as occur in palatal palsies or paralysis of the tongue. 'Rigid dysarthria'. This is characteristic of Parkinson's disease. In severe cases the phenomenon of palilalia is seen, in which there is a constant repetition of a particular syllable. Dysphasia Dysphasia may be either expressive or receptive. Patients with expressive dysphasia cannot understand speech but cannot formulate their own speech. Patients with receptive dysphasia results from lesions of the dominant hemisphere, which is the left hemisphere in right-handed people as well as in a high proportion of left-handed people. Expressive dysphasia. This is due to a lesion affected the patient may have a nominal dysphasia, in which the ability to name objects is lost but the ability to speak is retained. Receptive dysphasia. This results from lesions in Wernicke's area, which is the posterior part of the superior temporal gyrus and the adjacent parietal lobe. agraphia (inability to write) is due to a lesion in the left angular gyrus. The patient is unable to read or write spontaneously and the condition is often accompanied by nominal dysphasia, acalculia, hemianopia and visual agnosia. Gerstmann's syndrome consists of finger agnosia for both the patient's own finger and the examiner's finger, acalculia right/left disorientation and agraphia without alexia. It is found in lesions of the dominant hemisphere in the region of the angular gyrus. 4 CHAPTER 1 Motor activity Sensory Leg Thumb Ankle Neck Brow Toes Eyelid Nose Lips Jaw Larynx Primary motor area Supplementary motor area Precentral gyrus Central sulcus Primary somatosensory area Secondary visual area Broca's motor speech area Examination of the cranial nerves Olfactory nerve The sense of smell should be tested by the patient sniffing through each nostril as the other is compressed. The common causes of anosmia are olfactory prove meningiomas. It is important to use non-irritant substances when testing olfaction, as irritating compounds (e.g. ammonia) will cause irritation of the nasal mucosa. The stimulus is then perceived by the general sensory fibres of the trigeminal nerve. Optic nerve The optic nerve should be tested by: • measuring the visual acuity and colour vision • charting the visual fields • fundal examination with an ophthalmoscope • the pupillary light reflex. Primary visual area Fig. 1.1 Major areas of somatotopic organization of the cerebrum. Visual acuity should be tested using the standard Snellen type charts placed at 6 m. The acuity is recorded as a fraction, e.g. 6/6 or 6/12, in which the numerator indicates the distance in metres from the chart splaced at 6 m. vision. Refractive errors should be corrected by testing with the patient to view the chart through a pinhole. Visual fields can be charted by confrontation, with the patient facing the examiner and objects of varying size being moved slowly into the visual fields can be charted by confrontation, with the patient facing the examiner and objects of varying size being moved slowly into the visual fields can be charted by confrontation. should be undertaken in all cases of visual failure, pituitary tumour, parasellar tumour, other tumours possibly involving the visual pathways and demyelinating disease, or if there are any doubts after confrontation that the fields may be restricted. Perimetry can be performed using either a tangent screen, such as a Bjerrum screen (Fig. 1.3), or NEUROLOGICAL ASSESSMENT AND EXAMINATION 5 • total visual loss — optic nerve lesion • altitudinous hemianopia — partial lesion of the optic tract, radiation or calcarine cortex • bitemporal hemianopia — optic chiasm lesions such as pituitary tumour craniopharyngioma or suprasellar meningioma. Examiner Test object Patient Fig. 1.2 Visual field testing by confrontation. BJERRUM SCREEN 30 × 20 × 10 × Fixation point, e.g. 10/2000 Fig. 1.3 The Bjerrum screen. a Goldmann perimeter. The Bjerrum screen records the central field of vision. By enlarging the central area out to 30° it is easier to detect scotomas and to measure the blind spot and, provided a small enough target is used, the tangent screen provides an accurate representation of the peripheral fields. An automated perimetry machine will enable an accurate and reproducible field test that is particularly useful in cooperative patients. The pattern of visual field loss will depend on the anatomical site of the lesion in the visual pathways (Fig. 1.4): Fundal examination The fundus should be examined using the optic disc • vessels • retina. A pale optic disc is due to optic disc is due to optic disc be examined using the optic disc be examined using the optic disc • vessels • retina. A pale optic disc is due to optic disc be examined using the optic disc be examined using the optic disc • vessels • retina. A pale optic disc • vessels • retina. result of an optic nerve lesion caused by compression or demyelination, or consecutive, which follows severe swelling of the disc. Papilloedema is due to raised intracranial pressure and is evident by: • blurring of the disc margins • filling in of the optic cup • swelling and engorgement of retinal veins, with loss of normal pulsation of the veins haemorrhages around the disc margin (if severe). Third, fourth and sixth cranial nerves are all involved in innervation of the exercise they are usually examined together. This examination involves assessment of: • the position of the exercise they are usually examined together. paralysis of the levator palpebrae superioris as a result of a 3rd cranial nerve lesion or due to weakness of the tarsal muscle due to a sympathetic lesion (Horner's syndrome). The pupils An assessment should be made of the pupil size, shape and equality. The pupils' reaction to light should be tested by shining a beam into the eye and noting the reaction in that eye, as well as the 6 CHAPTER 1 Temporal field Nasal field Right eye Left eye asking the patient to fix on a distant object and then placing a pen approximately 12 cm in front of the bridge of the nose. A unilateral constricted pupil (miosis) often indicates a lesion in the sympathetic supply to the pupillary dilator muscle. Horner's syndrome, in its complete state, consists of miosis, ptosis, enophthalmos and dryness and warmth of half of the face. It is due to a lesion of the sympathetic supply such as results from an intracavernous carotid artery aneurysm, or a Pancoast's tumour of the parasympathetic fibres originating from the nucleus of Edinger–Westphal in the midbrain, and is therefore seen in a 3rd nerve palsy. The possible causes are an enlarging posterior communicating from intracranial pressure on these fibres in the 3rd cranial nerve (Chapter 9) and tentorial herniation resulting from intracranial pressure with the herniated uncus of the temporal lobe compressing the 3rd nerve (Chapter 5). The Argyll-Robertson pupil is a small, irregular pupil not reacting to accommodation but responding poorly to mydriatics; it is usually caused by syphilis. The myotonic pupil (Holmes-Adie) usually occurs in young women and presents as a unilateral dilatation of one pupil with failure to react to light. The pupil shows a slow constriction occurring on maintaining convergence for a prolonged period. In the complete syndrome the knee and ankle jerks are absent. Ocular movement The following are the general actions of the extraocular muscles. NEUROLOGICAL ASSESSMENT AND EXAMINATION • Lateral rectus (6th nerve) moves the eye horizontally outwards. • Inferior oblique (3rd nerve) elevates the eye when it is turned outwards. • Inferior rectus (3rd nerve) depresses the eye when it is turned inwards. outwards. • Superior oblique (4th nerve) depresses the eye when it is turned inwards. The patient should be tested for diplopia, which will indicate ocular muscle and cranial nerve are involved. • The displacement of the false image may be horizontal vertical or both. • The separation of images is greatest in the direction in which the weak muscle has its purest action. • The false image is displaced furthest in the direction in which the weak muscle should move the eye. Disorders of eye movement may be due to impaired conjugate ocular movement. The centre for the control of conjugate lateral gaze is situated in the posterior part of the frontal lobe, with input from the occipital region. The final common pathway for controlling conjugate movement is in the brainstem, particularly the median longitudinal bundle. A lesion of the frontal lobe causes contralateral paralysis of conjugate gaze (i.e. eyes deviated towards the side of the lesion) and a lesion of the brainstem causes ipsilateral paralysis of conjugate gaze (i.e. eyes deviated to side opposite to the lesion). Nystagmus should be tested by asking the patient to watch the tip of a pointer. This should be held first in the midline and then moved slowly to the right, to the left and then vertically upwards and downwards. Jerk nystagmus is the common type, consisting of slow drift in one direction and fast correcting movement in the other. Horizontal jerk nystagmus is produced by lesions in the vestibular system which may occur 7 peripherally in the labyrinth, centrally at the nuclei, in the vestibular system which may occur 7 peripherally in the labyrinth, centrally at the nuclei, in the vestibular system which may occur 7 peripherally in the labyrinth, centrally at the nuclei, in the vestibular system which may occur 7 peripherally in the labyrinth, centrally at the nuclei, in the vestibular system which may occur 7 peripherally in the vestibular system. amplitude is greater in the direction of the esion. By convention the quick phase is to the side of the lesion. By convention the quick phase is taken to indicate the direction of the nystagmus, so that if the slow phase is to the right and the quick phase to the left the patient is described as having nystagmus to the left. Vertical nystagmus is due to intrinsic brainstem lesions such as multiple sclerosis, brainstem tumours or phenytoin toxicity. The so-called 'downbeat' nystagmus, which is characterized by a vertical nystagmus exaggerated by a vertical nystagmus exagger syndrome, where the lower brainstem has been compressed by the descending cerebellar tonsils (Chapter 11). Trigeminal nerve; corneal sensation should be tested using a fine piece of cotton wool. The motor function of the 5th cranial nerve; corneal sensation should be tested using a fine piece of cotton wool. nerve can be tested by palpating the muscles while the patient clenches their jaw, testing the power of jaw opening and lateral deviation of the jaw (Fig. 1.5). Facial nerve is tested by assessing facial movement. In an upper motor neurone facial weakness the weakness of the lower part of the Greater occipital C. 2, 3 Lesser occipital C. 2 Greater auricular C. 2, 3 Dorsal rami of C. 3,4,5 Supraclavicular C. 3,4 Ophthalmic (V1) Maxillary (V2) Mandibular (V3) Transverse cutaneous nerves of neck C. 2,3 Fig. 1.5 Cutaneous nerves of neck C. 2,3 Fig. 1.5 Cutaneous nerves of neck C. 2,3 Fig. 1.5 Cutaneous nerves apply of the face, scalp and neck. 8 face is very much greater than the upper, with the strength of the orbicularis oculis being relatively preserved. This is due to a lesion between the cortex and the facial nucleus in the pons. Lower motor neurone weakness is evident by equal involvement of the upper and lower parts of the facial nerve nucleus in the pons. The chorda tympani carries taste sensation from the anterior two-thirds of the tongue and this should be examined using test flavours placed carefully on the anterior tongue. Vestibulocochlear nerve The 8th cranial nerve consists of: • the cochlear nerve Hearing can be examined at the bedside by moving a finger in the meatus on one side, to produce a masking noise, and repeating words at a standard volume and from a set distance in the other ear. Differentiation between conduction and sensorineural deafness can be aided using tests with a tuning fork. The Rinne's test involves holding a vibrating tuning fork in front of the external meatus and then on the mastoid process. In nerve deafness both air and bone conduction are reduced, but air conduction remains the better. In conductive deafness bone conduction will be better than air conducted to the abnormal ear. Formal audiometry should be performed if there are symptoms of impaired hearing. The vestibular nerve The simplest test of vestibular function is the caloric test, which is usually performed in patients suspected of having a cerebellopontine angle tumour or as a test of brainstem function in patients with severe brain injury. The test is described in Chapte 4, p. 44. CHAPTER 1 Glossopharyngeal and vagus nerves The glossopharyngeal and vagus nerves can be most easily assessed by testing palatal movement and sensation from the posterior one-third of the tongue (glossopharyngeal nerve) can be examined and taste from the posterior one-third of the tongue (glossopharyngeal nerve) can be most easily assessed by testing palatal movement and sensation from the posterior one-third of the tongue (glossopharyngeal nerve) can be examined and taste from the posterior one-third of the tongue (glossopharyngeal nerve) can be most easily assessed by testing palatal movement and sensation from the posterior one-third of the tongue (glossopharyngeal nerve) can be most easily assessed by testing palatal movement and senset (most easily assessed by testing palatal movement and senset) can be examined and taste from the posterior one-third of the tongue (glossopharyngeal nerve) can be most easily assessed by testing palatal movement and senset (most easily assessed by testing palatal movement and senset) can be examined and taste from the posterior one-third of the tongue (glossopharyngeal nerve) can be most easily assessed by testing palatal movement and senset (most easily assessed by testing palatal movement and senset) can be examined and taste from the posterior one-third of the tongue (glossopharyngeal nerve) can be examined and taste from the posterior one-third of the tongue (glossopharyngeal nerve) can be examined and taste from the posterior one-third of the tongue (glossopharyngeal nerve) can be examined and taste from the posterior one-third of the tongue (glossopharyngeal nerve) can be examined and taste from the posterior one-third of the tongue (glossopharyngeal nerve) can be examined and taste from the posterior one-third of the tongue (glossopharyngeal nerve) can be examined and taste from taste tested. Accessory nerve The accessory nerve supplies the motor power to the upper part of the trapezius and sternocleidomastoid. The latter muscle can be tested by turning the patient's head against resistance and watching and palpating the opposite sternomastoid muscle. The trapezius muscle is best tested by asking the patient to shrug the shoulders and attempting to depress the should be inspected to detect wasting and movements from side to side should be observed to detect wastenes. The tip of the protruded tongue will deviate toward the side of weakness. Examination of the periphery Posture and general inspection The patient's posture may indicate an underlying neurological disability, or an abnormal posture may result from pain. A patient with sciatica will often lie on the opposite side with the affected leg flexed at the hip and knee. should be inspected to compare size and shape and to detect deformity; longstanding neurological lesions may result in impaired growth or wasting. Lesions of lower motor neurone in infancy, such as a cute infantile hemiplegia and cerebral birth trauma, will also cause retardation in growth, but of a lesser degree, with a hemiplegic posture and exaggerated reflexes. NEUROLOGICAL ASSESSMENT AND EXAMINATION Wasting for specific posture and shoulder girdles should be inspected to detect wasting and fasciculation. As well as palpating for specific posture and shoulder girdles should be inspected to detect wasting and fasciculation. muscle wasting in each limb the circumference of the limbs should be measured at clearly identifiable positions, such as 8 cm above or below the tibial tuberosity. The pattern of wasting will be an important clue as to the underlying neurological disease. Wasting of the forearm and small muscles of the hand. This results from lower motor neurone lesions affecting particularly the C7, C8 and T1 levels and may be due to lesions of the: • spinal cord tumours • cervical disc prolapse • brachial plexus — trauma, cervical rib, axillary tumour • peripheral nerve — ulnar nerve compression at the elbow, carpal tunnel syndrome (median nerve). Wasting of the muscles of the lower leg. This will result from compression of the cauda equina or lumbosacral nerve myopathies and cause particular patterns of muscle wasting. • Facioscapulohumeral dystrophy involves the face and shoulder girdle. • Proximal limb girdles. • Dystrophia myotonica involves the face, sternomastoids and quadriceps femoris. Myotonia (the failure of muscle to relax after contraction) is present, particularly in the peripheral muscles and tongue. • Peroneal muscular atrophy, with predominant involvement of the lower limbs, causes the 'inverted bottle appearance' with similar but less striking changes in the upper limbs. appearance. Tone The tone in the upper limbs should be tested using a flexion-extension movement of the wrist, by holding the patient's terminal phalanges and by pronation-supination of the forearm. The tone in the lower limbs should be tested by flexion of the hip, knee and ankle. Decreased tone This is due to: • a lower motor neurone lesion involving the spinal roots or anterior horn cell of the spinal cord • lesions of the sensory roots of the reflex arc, e.g. tabes dorsalis • cerebellar lesion usually due to trauma). Increased tone This will be produced by any upper motor neurone lesion involving the corticospinal tracts above the level of the anterior horn cell in the spinal cord. There are three major types of hypertonicity, in which the resistance is most pronounced when the movement is first made. It is usually more marked in the flexor muscles of the lower limbs and is a sign of an upper motor neurone lesion. 2 'Lead pipe' rigidity, in which there is an alternating jerky resistance to movemen start from an upper motor neurone lesion. 3 'Cog wheel' rigidity, in which there is an alternating jerky resistance to movemen start from an upper motor neurone lesion. and which occurs in degenerative lesions of the extrapyramidal system, particularly Parkinson's disease. 'Clonus' is best demonstrated by firm rapid dorsiflexion of the foot and is indicative of marked increased tone. 10 Power The power should be tested in all limbs, comparing each side. A systematic evaluation will enable the recognition of a particular pattern of weakness that will be in keeping with either a cerebral, spinal cord, plexus or peripheral nerve weakness. The major nerve and main root supply of the muscles are shown in Table 1.1. The Medical Research Council classifies the degree of weakness by recording power, ranging from 0 to 5 (Table 1.2). It is apparent that there is a considerable range of power between grades 4 and 5 and some clinicians make their own further subclassification in this region. Weakness due to a corticospinal tract lesion is most marked in the abductors and extensors of the upper limbs and the flexors of the upper limbs. It is normally associated with increased tone and exaggerated reflexes Weakness due to lower motor neurone lesions is usually more severe than when the upper motor neurone is involved and is seen in the deltoid or calf muscles. It occurs classically in motor neurone disease but may also occur in lower motor neurone lesions, e.g. in the lower limbs following long-standing lumbar root compression. Reflexes The deep tendon reflex requires the stimulus, sensory pathway, motor neurone, contracting muscle and the synapses between the neurones in order to elicit a response. Reduced or absent tendon reflex arc: • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyneuritis • sensory root — tabes dorsalis • anterior horn cell — polyne of the pyramidal system, increased deep tendon reflex is retarded. Each deep tendon reflex is associated with a particular segmental innervation and peripheral nerve as listed in Table 1.3. The superficial abdominal reflex has a segmental innervation extending from T9 in the upper abdominal region to T12 in the lower area. The reflex may be absent in pyramidal lesions. However, the reflex may also be difficult to elicit when the abdominal muscles have been stretched or damaged by surgical operations, or in a large, pendulous, obese abdomen. Plantar reflex This should result in the great toe flexing the metatarsophalangeal joint, and usually at the interphalangeal joint, and indicates disturbance of the pyramidal tract. Sensation The modalities of sensation which should be tested are: • light touch • pinprick (pain) • temperature • position (proprioception) • vibration. All modalities of sensation travel by the peripheral nerve and sensory root to the spinal cord, or via the cranial nerves to the brainstem. The fibres for pain and temperature sensation enter the posterolateral aspect of the spinal cord, travel cranially for a few segments and then cross to the opposite anterolateral spinothalamic (trigeminothalamic) tract in the pons. The fibres end mostly in the ventrolateral nucleus of the thalamus and from here the NEUROLOGICAL ASSESSMENT AND EXAMINATION 11 Table 1.1 Nerve and major root supply of muscles. Spinal roots Upper limb Spinal accessory nerve Trapezius Brachial plexus Rhomboids Serratus anterior Pectoralis major root supply of muscles. Teres major C3, C4 C4, C5 C5, C6, C7, C8 C5, C6 Radial nerve Elexor carpi ulnaris Flexor digitorum enve Biceps Brachiolis Extensor carpi radialis longus Spinal roots Ulnar nerve Flexor carpi ulnaris Flexor digitorum profundus III and IV Hypothenar muscles Adductor pollicis Flexis pollicis brevis Palmar interossei Dorsal interossei Lumbricals III and IV C5, C6 C5, C6 Posterior interosseous nerve Supinator Extensor digitorum Abductor pollicis longus Extensor pollicis brevis Palmar interossei Lumbricals III and IV C5, C6 C5, C6 Posterior interosseous nerve Supinator Extensor digitorum Abductor pollicis brevis Palmar interossei Lumbricals III and IV C5, C6 C5, C6 Posterior interosseous nerve Supinator Extensor digitorum Abductor pollicis brevis Palmar interossei Lumbricals III and IV C5, C6 C5, C6 Posterior interosseous nerve Supinator Extensor digitorum Abductor pollicis brevis Palmar interosseous nerve Supinator Extensor digitorum Abductor pollicis brevis Palmar interosseous nerve Supinator Extensor digitorum Abductor pollicis III and IV C5, C6 C5, C6 Posterior interosseous nerve Supinator Extensor digitorum Abductor pollicis brevis Palmar interosseous nerve C8 C7, C8 C7, C8 C7, C8 Median nerve Pronator teres Flexor carpi radialis Flexor digitorum superficialis Abductor pollicis brevis Flexor pollicis brevis Flexor pollicis brevis\* Opponens pollicis Lumbricals I and II C6, C7 C7, C8, T1 L4 } L4, L5, S1 Inferior gluteal nerve Gluteus maximus L5, S1, S2 S1, peroneal nerves Tibialis anterior Extensor digitorum longus Extensor digitorum brevis longus Extensor digitorum brevis is often supplied wholly or partially by the ulnar nerve. 12 CHAPTER 1 sensory impulses pass through the posterior limb of the internal postcentral sensory cortex (see Chapter 19, Fig. 19.1). Fibres carrying light touch, proprioception and vibration sensation ascend mainly in the postation sensation ascend through the brainstem in the medial lemniscus, to synapse in the thalamus and then on to the sensory cortex. The sensory loss involving nocioceptive stimuli (pain and temperature) should conform to a particular pattern: • peripheral neuropathy • hemianalgesia — thalamic or upper brainstem Table 1.2 Medical Research Council classification of power. 0 Total paralysis 1 Flicker of contraction but no movement of limb 2 Muscle only able to make normal movement resistance 5 Normal power • loss of pain and temperature on one side of the body — lesion of the body — lesion of the body — lesion of the body. is best assessed using the 'finger-nose' test and in the lower limb using the 'heel-knee' test. It is important to determine whether abnormalities of coordination are due to defects in: • cerebellar function • proprioception • muscular weakness. Gait An essential part of the examination is to observe the patient's gait. This is best done not only as a formal part of the examination but also when the patient is not aware of observation. The type of gait is characteristic of the underlying neurological disturbance. A hemiparesis will cause the patient to drag the leg and, if severe, the leg will be thrown out from the hip, producing the movement called circumduction. A high stepping gait occurs with a foot drop (e.g. L5 root lesion due to disc prolapse, lateral popliteal nerve palsy, peroneal muscular atrophy). The patient raises the foot drop and the toe hits the ground first. In tabes dorsalis the high stepping gait is due to a profound loss of position sense but Table 1.3 Deep tendon reflexes, peripheral nerve and segmental innervation. Tendon reflex Major segmental innervation Peripheral nerve Biceps jerk Supinator jerk Knee jerk Supinator jerk Triceps jerk Knee similar gait, of lesser severity, will result from involvement of the posterior column of the spinal cord or severe sensory neuropathy which interferes with position sense. The gait is worse in the dark and the heel usually strikes the ground first. In Parkinson's disease or other extrapyramidal diseases the patient walks with a stooped, shuffling gait. The patient may have difficulty in starting walking and stopping. A slight push forward will cause rapid forward movement (protopulsion). In the ataxic gait, the patient is unstable due to cerebellar disturbance. A midline vermis tumour will result in the patient starting walking and stopping. fall to the ipsilateral side. A waddling gait is associated with congenital dislocation of the hips and muscular dystrophy. The hysterical gait is often bizarre and is diminished when the patient is unaware of any observation. Following the clinical assessment, a presumptive diagnosis is made and further investigations can be performed to confirm the diagnosis. These laboratory investigations and radiological procedures are described in the following chapter. Brain death The use of donor organs for transplantation and the advent of improved intensive care facilities have resulted in the necessity of medically accepted criteria of brain death. If there is irrecoverable brainstem damage and the tests described below show no evidence of brainstem function, then the patient is medically and legally dead. If artificial ventilation is continued the other organs may continue to function for some time. However, continued the other organs may continue to function for some time. distressing to the relatives, but is also wasteful of expensive medical resources that are often in short supply. The diagnosis of brain death tests. 13 The preconditions are that all reversible causes of brain death tests. hypothermia (temperature must be greater than 35°C) • neuromuscular blocking drugs • metabolic or endocrine disturbance as a cause of the patient's condition. Brain death testing must be delayed until these preconditions are absolutely satisfied. The tests for brainstem function are: • lack of pupil response to light • lack of corneal reflex to stimulation • lack of oculocephalic reflex • failure of response in the face or muscles supplied by the cranial nerves in response to painful stimulus • failure of respiratory movements when the patient is disconnected from a ventilator and the PaCO2 is allowed to rise to 50 mmHg. The tests should be repeated after an interval of 30 minutes and it is essential that they should be carried out by two doctors of adequate seniority and with expertise in the field. Further reading Conference of Medical Royal Colleges and Their Faculties in the UK (1979) Diagnosis of death. British Journal of Medicine 1, 322. Harrington D (1974) The Visual Fields, 4th edn. C V Mosby, St Louis. Jennett B (1981) Brain death. British Journal of Anaesthesia 53, 1111–1119. Medical Research Council (1976) Aids to the examination of the peripheral nervous system. Her Majesty's Stationery Office, London. Plum F (1980) Brain death. Lancet ii, 379. Plum F, Posner JB (1980) Diagnosis of Stupor and Coma, 3rd edn. F A Davis, Philadelphia. Walton J, ed. (1977) Brain. In: Diseases of the Nervous System. Oxford University Press, Oxford. CHAPTER 2 2 Neurosurgical investigations to determine the exact diagnosis are nearly always necessary following the clinical examination. The following is a list of the more common investigations that may need to be undertaken: • cerebrospinal fluid (CSF) studies • radiological investigations. Some of these investigations will be described in this chapter. The others will be dealt with in the chapters dealing with the relevant neurosurgical problems. Cerebrospinal fluid investigation The CSF is produced by the choroid plexus at a rate of approximately 0.4 ml per minute. The fluid circulates from the lateral ventricles through the cerebral aqueduct of Sylvius into the 4th ventricle, and into the subarachnoid space via the two laterally placed foramina of Luschka and a medial aperture in the roof of the 4th ventricle — the foramen of Magendie. The fluid circulates caudally into the spinal subarachnoid space, throughout the basal cisterns, up through the tentorial hiatus and then over the cerebral hemispheres. It is absorbed by the arachnoid villi of the dural sinuses, and especially by the superior sagittal sinus. Approximately 500 ml of CSF is produced each day. The total CSF volume is 140 ml; the lateral ventricles contain approximately 25 ml, the spinal cord 14 subarachnoid space 30 ml and the remainder of the fluid is found in the basal cisterns. Table 2.1 shows the normal constituents of CSF. The CSF glucose content is approximately 65% of the blood plasma level in the fasting state. There is a gradient for many of the constituents of CSF along the cerebrospinal axis (Table 2.2). The fluid is normally clear and colourless; it will appear turbid if it contains more than 400 white blood cells or 200 red blood cells per mm3. Yellow discolouration, xanthochromia, is due to the breakdown products of red blood cells; these follow haemorrhage into the CSF. CSF can be obtained by: • lumbar puncture • cannulation of the lateral ventricle. The fluid is usually obtained by: • lumbar puncture • cannulation of the lateral ventricle. puncture has failed due to technical difficulties, if there is local skin sepsis or, in some radiology investigations, where it is the preferred route of contrast administration for myelography. Ventricular puncture is usually only performed as an intraoperative procedure or for temporary reduction of intracranial pressure in an emergency. Lumbar puncture The most common indications for CSF examination by lumbar puncture are: • meningitis • subarachnoid haemorrhage • neurological diseases such as multiple sclerosis NEUROSURGICAL INVESTIGATIONS Table 2.1 CSF statistics (lumbar). Volume Rate of production Pressure (recumbent) Cells Protein Glucose IgG Chloride 140 ml 0.4 ml/min 10-15 cm of CSF Less than 3-4 white cells/mm3 0.15-0.45 q/l (15-45 mg/100 ml) 2.8-4.2 mmol/l (50-75 mg/100 ml) 10-12% of total protein 120-130 mmol/l The values are expressed in SI (Système Internationale) units and the corresponding traditional units are in parentheses. Table 2.2 CSF gradients along the cerebrospinal axis. Protein (q/l) Glucose (mmol/l) Ventricle Cisternal Lumbar 0.1 4.5 0.2 4.0 0.4 3.4 • cytological examination for neoplastic disease • radiological imaging (e.g. myelography) or radio-isotope investigations • measurement of intracranial pressure. The most important contraindication to lumbar puncture is clinical evidence of raised intracranial pressure. Papilloedema is an absolute contraindication and a lumbar puncture should never be performed in a patient in whom an intracranial space-occupying lesion is suspected. If there is any doubt a CT scan or MRI must be performed prior to lumbar puncture. patient should be positioned on the side, the back vertical on the edge of the bed and the knees flexed up to the chest. The iliac crest is palpated; 15 this lies at the L3/4 level. The lumbar puncture can be carried out at this space or at the spaces immediately above or below. The area is prepared with antiseptic solution and draped. The procedure must be performed under completely sterile conditions. The interspinous area is palpated and the skin injected with 1-2 ml of 1% lignocaine local anaesthetic. The lumbar puncture needle is inserted between the two spinous processes, pointing in a slightly cranial direction. If performed carefully it is usually possible to feel the needle pass through the interspinous ligament and then through the dura. The stilette of the lumbar puncture needle is withdrawn and a manometer attached to measure the pressure. The fluid is drained into sterile containers and sent for examination. Complications of lumbar puncture is well tolerated and complications should be minimal. However, there are several potential hazards and complications; these include: • progression of spinal cord compression compression compression compression compression compression compressio risk of lumbar puncture worsening brain herniation can be avoided if the procedure is not undertaken in patients with raised intracranial pressure. Neurological deterioration may follow lumbar puncture and myelography in patients with raised intracranial pressure. make the diagnosis, myelography should be avoided as magnetic resonance imaging is the investigation of choice for spinal tumours. Neurological deterioration requires prompt surgery; this is discussed in Chapter 15. Infection should be avoided by the use of scrupulous sterile techniques. If the procedure is performed at a level that is too high there is a risk of neural damage, particularly to the conus medullaris. Rarely, a nerve root may be in-16 jured by the improper placement of the needle. Injury to a spinal subdural or epidural haematoma; this risk is increased if the patient is taking anticoagulation therapy. The traumatic effects of the lumbar puncture are responsible for minor, transient low back discomfort. Very rarely, frank disc herniation has been reported due to damage of the annulus fibrosus of the disc. Headache The most common complication of lumbar puncture is headache. In most cases this is due to low CSF pressure that results from persistent leakage of the fluid through a hole in the arachnoid and dura. It is generally recommended that patients should remain flat for 12 hours following a lumbar puncture to minimize the risk of this complication. The use of a narrow-gauge needle (20 gauge or less) and avoiding multiple puncture holes in the meninges also decreases the chance of troublesome postlumbar puncture headache. If the headache develops following mobilization the patient should be instructed to lie flat for a further 24 hours and encouraged to drink large volumes of non-alcoholic fluids. Some clinicians advocate the use of 'blood patch' for the treatment of persistent postspinal headache. This technique uses the epidural injection of autologous blood at the site of dural puncture to form a thrombotic tamponade which seals the dural opening, but this is usually unnecessary. CSF examined in a centrifuge and examined for evidence of xanthochromia, this being indicative of haemorrhage into the CSF. Three major pigments derived from red cells may be detected in CSF: oxyhaemoglobin, bilirubin and methaemoglobin, bilirubin and methaemoglobin, bilirubin and methaemoglobin, bilirubin and methaemoglobin. subarachnoid space. It reaches a maximum in the first 36 hours and gradually disappears over the next 7-10 days. Bilirubin is yellow and is the iron-free derivative of haemoglobin produced in vivo following the haemolysis of red cells. to degrade haemoglobin. It is first detected about 10 hours after the onset of subarachnoid bleeding and reaches a maximum at 48 hours. It may persist for 2-4 weeks after extensive haemorrhage. Methaemoglobin is a reduction product of haemoglobin. It is a brown pigment that is dark yellow in dilution and it is characteristically found in encapsulated subdural haematomas. Although it may be detected by spectrophotometry of the spinal fluid in patients with large encapsulations of this sort, the pigment is not usually observed in other xanthochromic spinal fluids. Xanthochromic spinal fluids. disease of the newborn. The fluid should be sent for microbiological and biochemical examination and, if clinically indicated, cytological examination for malignant cells. The common abnormalities are shown in Table 2.3. Normal CSF contains no more than four lymphocytes or mononuclear cells per mm3. Polymorphonuclear cells are never found in normal CSF but an isolated granulocyte, presumably derived from blood at the time of lumbar puncture, may be seen if the CSF has been cytocentrifuged. A granulocytic phase also occurs at the onset of a viral meningitis, prior to the development of a purely mononuclear reaction. Eosinophils are not seen in normal CSF. The most common causes of prominent eosinophilic reaction are parasitic diseases, but eosinophilia may also occur in inflammatory diseases, as shown in Table 2.3. Examination of the CSF using the polymerase NEUROSURGICAL INVESTIGATIONS 17 Table 2.3 CSF abnormalities. CSF abnormality Disease suspected Polymorphonuclear pleocytosis Bacterial meningitis Mononuclear pleocytosis Viral meningitis Acute demyelination Eosinophils Parasitic infections Trichinella and Ascaris Toxoplasma Cysticercosis Inflammatory diseases Tuberculosis Syphilis Subacute sclerosing panencephalitis Fungal infections Other diseases Lymphoma Hodgkin's disease Multiple sclerosis Raised protein CNS infection Spinal neurofibromas Acoustic neuromas Guillain-Barré syndrome Low sugar Bacterial meningitis Low chloride (24 hours Early seizures 3 25 Post-traumatic amnesia > 24 hours Early seizures 25 Compound depressed fracture Dura torn Extradural haemorrhage Subdural haemorrhage 7 25 20 40 50 Post-traumatic amnesia > 24 hours Early seizures Compound depressed fracture 35 70 neurosurgical procedure, but the overall incidence is estimated to be about 18%. Almost half occur within the first postoperative month. Conditions such as cerebral abscess and tumour are frequently associated with seizures and how much of the risk relates to

the surgery and how much to the underlying pathology is difficult to assess. The incidence of postoperative seizures following aneurysm surgery is about 20%, with seizures being most common with middle cerebral artery aneurysms. A similar incidence is posterior for soaveryty meningiomas. Burr hole biopsy has a lower incidence of seizures of 9%. Seizures do not complicate posterior for soaveryty meningiomas. Burr hole biopsy has a lower incidence of postoperative seizures prophylaxis Most studies assessing the value of seizures of 9%. Seizures are complicated postoperative methy, and then continue on maintenance therapy. If seizures contrading dose of phenytoin is usually discontinue of after 6 months. If seizures occur despite adequate phenytoin plasma levels, carbamazepine should be introduced. Tumours and seizures Although the major concern when a patient presenting with a seizure. There is night are in patients presenting with partial seizures. Tumours are responsible for late-onset epilepsy (as defined by seizures. Or of a diloma and uncommon cause of childhood epilepsy. Seizures are a common presentation in patients present with a seizure. There incidence of seizures with meningiomas is approximately 50%, 40% with glioblastoma multiforme and 80% with anaplastic astrocytoma. Investigation of seizures of epilepsy is a contrading of epilepsy is a contrading of epilepsy. The EEG is non-invasive and epilepsy is constrained to event. A history of febrile convul- 273 sions of infancy in a patient presenting with complex partial seizures of the brain by the use of scale peletrode are upperied epilepsy. The EEG is non-invasive and an epileptic seizures of the brain by the use of scale peried and injury may suggest posttraumatic epileps and the nording of electral activity from the patient advance of seizures of the brain by the use of scale peletral activity form the patient s

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