16 - 52_Clinical_Examination

01 - 1. History Taking & Interview Skills

1. History Taking & Interview Skills

1. History Taking & Interview Skills The four tasks of a psychiatric interview are 1. Build a therapeutic alliance. 2. Obtain the demographic information required. 3. Interview for diagnosis. 4. Negotiate a treatment plan. Basic concepts on approaching threatening topics: 1.Use normalizing questions to decrease a patient's sense of embarrassment about a feeling or behaviour. 2. Use symptom expectation and reduction of guilt to defuse the admission of embarrassing behaviour. 3. Use symptom exaggeration to determine the actual frequency of a sensitive or shameful behaviour. 4.Use familiar language when asking about behaviours. Nondirective techniques Use Example Comments Open-ended Qs The opening stage of the interview, to allow free narration. Non-directive technique What brings you to the hospital? Preferable when highly suggestible; not very useful to focus if overtalkative or extremely poor historian. Usually starts with ‘tell me’, ‘describe’, etc. Repetition Repeating the exact words of the patient Pt: I was having bad dreams last night.

Dr: So, you were having bad dreams last night. Helps patient to feel that doctor is listening actively Restatement Similar to repetition but phrases rearranged Pt: I was having bad dreams last night.

Dr: So, you are getting disturbed by the dreams you have. Helps patient to feel that doctor is listening actively Summation Brief summarisation of what the patient has said up to a point in the interview

‘So from what you have told so far, you are worried for last 4 months and not sleeping well, and your job is at risk. Right?’ Helps patient to check if he has said what he intended to say. Helps the doctor to form an idea of the narration so far. Clarification Doctor tries to get details from patients about what the patient has already said.

‘You said you are feeling depressed ever since you can remember. When do you feel most depressed?’ Helps in avoiding misconceptions by the clinician. Also shows clinician’s interest in knowing more. Facilitation Helping patients continue the interview by providing both verbal and nonverbal encouragement.

Approval nods, leaning forward slightly to express interest, ‘Yes. And then?’, ‘yeah, go on…’ ‘Uh-huh’ etc. Helps patient to feel that doctor is listening actively. Encourages flow of information.

© SPMM Course Techniques when changing topics: 1. Use smooth transitions to hint at something the patient just said. 2. Use referred transitions to hint for something said earlier in the interview. 3. Use introduced transitions to pull a new topic from thin air.

1. Non-directive techniques: These techniques are employed without focussing on a particular answer.
2. Directive techniques: These are focussed on seeking a particular answer or driven by other motives of the doctor. Note that these are not necessarily detrimental but must be used judiciously. Directive techniques Use Example Comments Closed questions When, where, how many, which and what questions. Answers can only be ‘yes or no’, in most occasions. When clubbed with non-facilitative gestures, can be detrimental to interview process. Stating a presumption followed by tags can be very directive. Did you sleep well last night?

You have lost weight. Haven’t you? Better avoided in early parts of the interview as they can produce prescribed answers lacking in detail. Also avoid in highly suggestible patients. Good technique is to start with open; move to closed by the end of the interview. Useful to rule out less likely symptoms. Question rephrasing Persisting with a question to seek an answer; so, restating the question in different terms for a second time.

Often used when patient digresses from the topic of discussion. The motive is to collect the specific information. Redirection Gently reorienting patient towards the topic of discussion. Pt: ‘It is not good if one’s parents are divorced even before one goes to school.’ Doc: ‘I’d like to hear more about your parents, but first let me get a picture of what’s happening to you of late’. The motive is to keep the patient on track. Transition Moving from one to another topic – this is a special skill and preferably must be done as smoothly as possible to keep the patient interested. ‘You mentioned that your mother is a medical secretary. What about yourself? What job do you do?’ Smooth transitions – uses the cue off something the patient just said. Referred transitions – uses the cue off something said earlier in the interview. introduced transitions -uses a new topic to proceed.

© SPMM Course Limit setting Useful to manage time pressure, especially in garrulous patients. ‘I am going to interrupt you as there are few important things we need to cover today’. To be used cautiously, overuse may detach patient from the doctor. The motive is to use time effectively.

© SPMM Course Other methods to elicit information:

Technique Description Example Comments Confrontation Point out to a patient something to which the doctor thinks the patient is missing or denying.

‘You seem not to have gained any weight in last 6 months. Is it possible that your eating has been poor again?’ Must be done in a respectful way. The aim is to help patients face a difficult aspect rather than dismissing patients by showing a negative aspect. Interpretation Clarifying certain associations or relationships that the patient may not see.

You seem very anxious when talking about your job. Are you having any problems at workplace? Sophisticated technique and should generally be used only after the doctor has established some rapport. Should be stated as a hypothesis after sufficient collection of evidence from the interview. Self-revelation Limited, discreet selfdisclosure by physicians

‘Do you like Shakespeare? I was a mad fan when I was at school.’ Helps physician feel atease sometimes. Excessive self-revelation is a boundary violation. Silence Silence can be used either to facilitate discourse or to indicate disapproval or disinterest. Sometimes useful and allows free emotional expression.

Relieves patient’s pressure and he/she may fell relaxed that not every moment must be spent talking. Symptom expectation

Without a formal admission from the patient, asking about details of problem behaviour. Doctor assumes (rightly) that the patient is involved in the act. What sorts of drugs do you usually use when you're drinking? (Assuming that the patient uses drugs)

Defuse the admission of embarrassing behaviour. May help in reduction of guilt. But must be used with experience and according to the context. Symptom exaggeration When deception or minimisation is expected, overstating a guessed frequency in order to elicit a true answer. How many times have you taken overdoses since your last hospitalization? Four? Five? Also helpful in reducing guilt to certain extent as the patient feels that the doctor has expected a higher amount of problem that what she/he actually has brought.

© SPMM Course Supportive techniques – not aimed at eliciting information: Supportive technique Use Example Reassurance Used to instil positive hope and avoid or reduce despair. Must not be falsely reassuring. ‘The depression may be very difficult for you. I think it is very likely with the proper treatment you can get back to your job’.

Advice Many patients seek advice directly; it is acceptable to provide advice but based on sound understanding of the context. Premature advice can be obstructive than facilitative.

‘I think it is best for you to consider ECT at this time. If I am you, I will give this a serious thought.' Postponement Conscious and deliberate postponement of delicate issues; but must be opened at an appropriate time. ‘I can see that you are uneasy to tell me about your relationships. That’s OK, we can come back to this when you feel ready to discuss with me.’ Validation / normalisation Helps to decrease a patient's sense of embarrassment about a feeling or behaviour. Generally done by quoting how it is normal for people to have different emotions/ reactions/ behaviours, etc. ‘Sometimes when people are very depressed, they think of hurting themselves. Has this been true for you?’

Acknowledgement of affect Making a remark about patient’s affect can facilitate disclosure. I can see that you look anxious when talking about those voices.

Positive reinforcement Gently uplifting self-esteem by statements of praise (but at a realistic extent) ‘I've never been good at expressing my problems’. ‘Well, I think you've described the situation in a way that helped me understand what you have been going through’. Statement of respect Affirmative statements (must be genuine and appropriate) indicating respect and dignity along with positive reinforcement “You have been through a lot.” “I’m impressed at how you have hung in there.” “You must be a very strong person.” Partnering The interviewer encourages the patient to ask questions and to express any concerns, encouraging team working “I’m here to help.” “Let’s plan on working on this together.”

© SPMM Course Obstructive techniques that may hamper the progress of information sharing: Obstructive techniques Use Example Suggestive questions Answers are contained in the question itself. Misleads both the patient and the doctor. The patient is left with little choice. These voices are not from your head. Am I right? Why questions These questions ask the patient to discover their own problems, in a way. Not useful when used to elicit information from a distressed patient. Why do you keep waking up so early in the morning? Compound questions Adding two or more questions in a single statement. This confuses the patient and will lead to either a vague response or non-response. Do you take a vacation every year, and are you able to relax? Negative Nonverbal gestures Facial expression, body posture, and behaviour that indicate lack of interest or inattentiveness, The doctor is yawning or repeatedly checking his/her watch, other repetitive gestures like tapping the table, etc. Disapproval Expressing unhappiness with a topic that the patient wants to discuss; may lead to withdrawal and not revealing the important problem faced b y the patient. ‘Over the last month I have had trouble with sex’. ‘Dr: We are here to talk about your sleep.' Setting traps Tricking the patient using his own words. Often seen as doctor’s attempt to negate patient’s problems. You wanted to see me as nothing had gone well for you, but you just said that you have got a new job and keeping a good shape. Adapted from Kay J & Tasman A. Essentials of Psychiatry, 2nd edition, 2006. John Wiley & Sons, Ltd.

© SPMM Course Open-Ended vs. Closed-Ended Questions Open-Ended Questions Closed-Ended Questions Highly informative answers They produce spontaneous formulations. Low yield answers They lead the patient. Low reliability of answers. Non-reproducible at a later date, or by a different doctor. High reliability. Low precision – do not focus on target symptoms.

The intent of the question is clear, and so precise, focused answers elicited. Not very time efficient. My lead to circumstantial elaborations. High time efficiency.

Low diagnostic coverage as patient selects the content revealed. Good diagnostic coverage as doctor selects interested content. Adapted from Othmer E, Othmer SC. The Clinical Interview Using DSM-IV. Washington, DC: American Psychiatric Press; 1994. Techniques for a poor historian Use open-ended questions and commands to increase the flow of information. Use continuation techniques to keep the flow coming. The Shift to the neutral ground when necessary. Schedule a second interview when all else fails. Techniques for over-talkative garrulous historian Use closed-ended and multiple-choice questions to limit the flow. Perfect the art of the gentle interruption. Educate the patient about the need to move along in the interview. Ancillary

methods of gathering information: Behavioural observation methods: Observing and recording behavioural events, to study mental state or plan intervention. Often used when patients are in seclusion. Event sampling: e.g. every fifth or tenth event is coded in detail Time sampling: observations may be made only every 5 or 10 mins ‘Functional analysis' refers to attempts to explain and predict the functions of a phenomenon by examining any relationships to the outcome. It is a special variant of behavioural observation methods, where the sequence of antecedent environmental events, target behaviour and concurrent events and consequent outcomes are observed. This is also called ABC analysis. Often used in LD setting, dementia care, and challenging behaviour services.

 Using an interpreter: Explain the goals of the interview to the interpreter Explain structure and content of interview Explain the need for literal translation – not interpreted translation in the Mental Status Examination Ask for feedback when something is hard to translate Offer to debrief the interpreter to address any of their own emotional concerns following the interpretation Ask interpreter about the patient’s degree of openness or disclosure Preferably work with same interpreter/culture-broker for the same case whenever possible

02 - 2. Laboratory assessment of physical factors

2. Laboratory assessment of physical factors

© SPMM Course 2. Laboratory assessment of physical factors Depression Depression is a clinical diagnosis. A physical examination is always required to rule out several common medical disorders that can present with depression (especially endocrine disorders). Laboratory tests are required if medical causes are suspected and to assess baseline fitness before starting antidepressants. Several chronic medical disorders are associated with depression (e.g. Coronary artery disease, Diabetes mellitus, End-stage renal disease, HIV infection, various malignancies, degenerative neurological disorders and stroke) but these do not ‘present’ with depressive features and as such for a patient presenting with depression, there is no need to exclude all of these medical disorders before diagnosis depression. TEST WHY DO WE DO IT? Full blood count Rule out infectious and inflammatory pathology Thyroid-stimulating hormone (TSH) Rule out hypothyroidism Vitamin B-12 Deficiency can mimic depression HIV test & Syphilis (rapid plasma reagin) In suspected cases of sexually transmitted infections Electrolytes, including calcium, phosphate, and magnesium levels Deficiency can contribute to fatigue and mimic depression GFR and creatinine In preparation for antidepressant use and to rule out renal insufficiency contributing to depression Liver function tests (LFTs) In preparation for antidepressant use and to rule out alcohol-related liver damage in suspected cases Blood and urine toxicology screen In suspected cases of drug abuse 24-hour urinary free cortisol In suspected cases of Cushing disease; will require additional confirmation, as this can be positive in a large number of patients with depression. ACTH stimulation test Addison’s disease can also mimic depression

The following table displays some common abnormalities. Please note that none of the tests below are required routinely during a workup for depression. LAB ABNORMALITIES INTERPRETATION Dexamethasone Suppression Test DST nonsuppression (DST-positive result) is seen in many disorders associated with depression e.g. grief reactions (10%), dysthymic disorders (23%), major depressive disorder (44%), melancholia/somatic syndrome (50%), psychotic affective disorders

(69%), and in depression with serious suicidality (78%). DST-positive patients respond more favorably to biological interventions. DST nonsuppression is nonspecific; can be seen in chronic pain, patients with anorexia or bulimia, alcoholism, obsessive-compulsive disorder, or anxiety disorders. Corticotropin-Releasing Hormone Test HPA axis abnormality in major depression results in blunted ACTH response to CRH.

© SPMM Course Serum Thyroxine Concentrations 1% and 4% of depressed patients, esp. women show evidence of overt hypothyroidism; 4% to 40% have subclinical hypothyroidism, contributing to treatment failure. Serum T4 reductions may accompany treatment with antidepressants, lithium, sleep deprivation, or ECT, especially in responders. Thyrotropin-Releasing Hormone Test ~ 30% of depressed patients show blunted TSH response during depression

Anxiety and other neuroses A number of medical disorders can directly contribute to anxiety and panic attacks, but in practice, patients seeking clinical consultations seldom require specific investigations to diagnose these conditions. Diagnostic possibilities for panic attacks include paroxysmal atrial tachycardia, pulmonary embolus, seizure disorder, Meniere's disease, transient ischemic attack, carcinoid syndrome, Cushing's disease, hyperthyroidism, hypoglycemia, and pheochromocytoma. A physical examination is warranted for all first presentations; extensive medical evaluation for these disorders is indicated only when other features suggest physical disease. Lactate infusion: Nearly 72% patients with panic disorder have a panic attack when administered IV injections of sodium lactate. Therefore, lactate provocation is used to confirm a diagnosis of panic disorder. Hyperventilation and CO2 inhalation have been used. Panic attacks triggered by sodium lactate are not inhibited by peripherally acting beta-blockers but are inhibited by benzodiazepines and tricyclic drugs. Narcoanalysis: Interviews with amobarbital are very rarely used in current clinical practice for diagnostic and therapeutic indications. These are sometimes helpful in differentiating nonorganic and organic conditions, particularly in patients with symptoms of catatonia, stupor, and muteness. Organic conditions tend to worsen with infusions of amobarbital, but nonorganic or psychogenic conditions tend to get better because of disinhibition, decreased anxiety, or increased relaxation. Therapeutically, amobarbital interviews are useful in disorders of repression and dissociation such as amnesia and fugue. Benzodiazepines can be substituted for amobarbital. Psychosis Differential diagnoses to be considered in the history of presenting illness: head trauma (subdural haematoma), seizures, new-onset headaches, focal neurological deficits, abnormal body movements, memory loss, and tremor especially in older patients, recreational drug use, dietary history (deficiencies of vitamin B12, folate, thiamine, and niacin can all cause psychosis). Physical examination: vital signs, level of consciousness, evidence of malnutrition, signs of hypoor hyperthyroidism or cushingoid features, rashes associated with autoimmune disorders,

© SPMM Course dysmorphic facial features (genetic syndromes e.g velocardiofacial), focal neurological signs and examination for signs of raised intracranial pressure Initial tests TESTS WHY DO WE DO IT? Full blood count Rule out infectious and inflammatory pathology; the baseline for starting haemotoxic antipsychotics such as olanzapine, clozapine. Thyroid profile (TSH, free T4) Rule out hypo or hyperthyroidism. If abnormal carry out free T3 level and thyroid autoantibodies ELISA for anti-thyroid peroxidase Blood glucose, lipid profile, ECG Baseline for antipsychotic therapy; metabolic syndrome is common among patients with psychotic disorders Prolactin levels Baseline for antipsychotic therapy

In suspected cases of sexually transmitted infections Electrolytes, including calcium, phosphate, and magnesium levels May be abnormal if there is an underlying metabolic or endocrine disturbance GFR and creatinine In preparation for antidepressant use and to rule out renal insufficiency contributing to depression Liver function tests (LFTs) In preparation for antipsychotic use; to rule out chronic alcohol abuse and Wilson's disease in suspected cases Blood and urine toxicology screen Acute toxic drug effects the most common cause of psychosis; screen for amphetamines, cocaine, cannabis, and benzodiazepines

SUSPECTED CONDITION TESTS REQUIRED Delirium with psychosis Blood glucose, Blood alcohol, Urine microscopy and culture, Blood culture, Chest X-ray. Suspected STDs HIV test & Syphilis (rapid plasma reagin) Screen for autoimmune disorders in suspected cases Anti-Nuclear Antibodies, CRP and ESR Suspected encephalitis syndrome NMDA receptor (NMDAR) and voltage-gated potassium channel receptor (VGKC) auto-antibodies (IgG) Cushing's syndrome 24-hour urinary free cortisol test followed by DST and ACTH challenge, evening salivary cortisol, and the dexamethasone-corticotropin-releasing hormone test Porphyria Spot urine sample for porphobilinogen during acute attack, and 24-hour urine for porphyrins, porphobilinogen, and delta-aminolevulinic acid Hyperparathyroidism Serum calcium and serum parathyroid hormone test Wilson's disease Serum ceruloplasmin, 24-h copper excretion test Lysosomal storage diseases Skin biopsy, genetic tests, and the detection of serum alpha-galactosidase enzyme Homocystinuria Homocysteine in urine and blood and molecular genetic testing Metachromatic leukodystrophy Arylsulfatase A enzyme activity in WBCs or in cultured skin fibroblasts Malnourishment Serum homocysteine and foalte (folate deficiency) , vitamin B12, niacin, tryptophan, nicotinamide adenine dinucleotide (NAD) and NADP CNS lesions MRI or CT scan; EEG if TLE is suspected

Porphyrias: Acute intermittent porphyria (AIP) is one of the groups of disorders of haem

 metabolism, characterised by neurological and psychiatric manifestations without obvious cutaneous markers. AIP manifests itself by abdomen pain, neuropathies, and constipation, but, unlike most types of porphyria, patients with AIP do not have a rash. It is an autosomal dominant disorder with the presentation starting between ages 18 and 40. It is episodic in nature, and the episodes are often triggered by certain medications including estrogens, barbiturates and benzodiazepines. Diclofenac can precipitate an episode. Psychiatric manifestations include depression, anxiety, delirium and psychosis. Most important lab test is demonstrating increased urinary porphobilinogen during acute attacks. Treatment is aimed at reducing haem synthesis by administering haemin. Autoimmune encephalitis presenting as psychosis: Autoimmune disorders with antibodies produced against crucial neurotransmitter receptors can present with psychosis. Several anecdotal reports have pinpointed the following receptors as most vulnerable in this regard. Voltage Gated Potassium Channel complex (LGI1, CASPR2, contactin-2) N-Methyl-D-aspartate receptor (NMDA) AMPA receptor GABA-B Glycine receptor Some studies have estimated that 6.3% of first onset psychosis patients have pathogenic antibodies against brain receptors (Zandi et al., 2011). The most well known of these syndromes is the anti-NMDA receptor (NMDAR) encephalitis. Anti-NMDAR antibodies result in the titre-dependent destruction of synaptic NMDAR through crosslinking and internalisation. Around 4% of patients with anti-NMDAR present with isolated psychiatric symptoms. It is more common in females (80%) than males ~50% of women with anti-NMDAR have an underlying ovarian teratoma. 75% of patients first present to a psychiatrist with acute psychosis and/or mania. Psychosis associated

with anti-NMDAR encephalitis usually presents with a prodromal illness (fever, headaches, malaise). In suspected cases, the following investigations are appropriate o Serum NMDAR and VGKC antibodies o Test for ANA, CRP, ESR, FBC, U+E (low sodium is seen in those with anti-VGKC antibodies) o If there is a strong suspicion EEG (look for encephalopathy with disorganized

 delta/theta activity) and MRI brain (look for medial temporal hyperintensity, usually seen in T2 or FLAIR sequences in the hippocampi, frontobasal and insular regions and basal ganglia; normal in ~50%). o Confirmatory diagnosis requires CSF analysis: Lymphocytic pleocytosis, elevated protein and oligoclonal bands are seen in ~60% of cases; almost all have intrathecal anti-NMDAR antibodies. Note that patients who are cured of anti-NMDAR encephalitis may continue having detectable antibodies in serum and/or CSF. CSF antibodies rise during each relapse o Elevated creatine kinase is a non-specific feature of the anti-NMDAR illness. o In females with anti-NMDAR, ask for ultrasound or CT pelvis. Anti-NMDAR encephalitis usually responds to 3 days of methylprednisolone orally or intravenous followed by oral prednisolone, in association with 5 days of plasma exchange. The remission thus achieved can be maintained by either (1) steroids alone; (2) steroids with a steroid-sparing agent, such as azathioprine or mycophenolate mofetil or (3) rituximab. Regular benzodiazepines may be required. AVOID ANTIPSYCHOTICS as dystonic reactions and NMS-like syndrome with rigidity, hyperthermia, and autonomic instability might occur on the use of antipsychotics in patients with anti-NMDAR antibodies. Dementia Patients presenting with memory difficulties always require a thorough physical examination to look for signs of neurological disorders. In addition, several nutritional and metabolic factors can produce what is called ‘reversible’ dementia – cognitive impairment with no progressive, degenerative pathology. Laboratory investigations required for initial dementia workup are shown below. TEST WHY DO WE DO IT? Full blood count Rule out infectious and inflammatory pathology; the baseline for starting antidementia medications. Thyroid profile (TSH, free T4) Low T4 can cause cognitive impairment Blood glucose, lipid profile, ECG Baseline before starting antidementia medications; metabolic syndrome is common among patients with vascular dementia Thiamine, folate levels Thiamine deficiency can result in memory impairment esp. in alcoholics Tests for syphilis or HIV HIV is associated with cognitive impairment that can worsen with opportunistic infections. Electrolytes, including calcium, phosphate, and magnesium levels May be abnormal if there is an underlying metabolic or endocrine disturbance causing cognitive impairment GFR and creatinine In preparation for antidementia medications Liver function tests (LFTs) In preparation for antidementia medications CT or MRI brain This is becoming a routine practice though the diagnostic yield of routine imaging is low in senile dementia of Alzheimer’s type. Recommended when suspecting vascular dementia, subdural hematoma or tumours.

03 - Anorexia

Anorexia

© SPMM Course EEG No need for routine EEG. But rapid onset dementia may suggest CJD for which EEG and MRI are warranted.

Anorexia Several abnormalities are expected in physical investigation in anorexic subjects: (The list below is adapted from Fairburn & Harrison, 2003) Endocrine Low concentrations of luteinising hormone, follicle stimulating hormone, and oestradiol Low T3, T4 in low normal range, normal concentrations of thyroid stimulating hormone (low T3 syndrome) Mild increase in plasma cortisol Raised growth hormone concentration Severe hypoglycaemia (rare) Low leptin (but possibly higher than would be expected for bodyweight) Cardiovascular ECG abnormalities (especially in those with electrolyte disturbance): conduction defects, especially prolongation of the Q-T interval, of major concern Gastrointestinal Delayed gastric emptying Decreased colonic motility (secondary to chronic laxative misuse) Acute gastric dilatation (rare, secondary to binge eating or excessive re-feeding) Haematological Moderate normocytic normochromic anaemia Mild leucopenia with relative lymphocytosis Thrombocytopenia Other metabolic abnormalities Hypercholesterolaemia Raised serum carotene Hypophosphataemia (exaggerated during refeeding) Dehydration Electrolyte disturbance o Varied in form; present in those who frequently vomit or misuse large quantities of laxatives or diuretics o Vomiting results in metabolic alkalosis and hypokalaemia. o In repetitive vomiting, loss of hydrochloric acid from gastric juices leads to metabolic alkalosis (loss of acid – alkalosis). o Laxative misuse results in metabolic acidosis, hyponatraemia, hypokalemia

04 - Alcohol use disorders

Alcohol use disorders

© SPMM Course o During laxative induced diarrhoea, a large amount of bicarbonate may be lost in the stool. With normal kidneys, the lost bicarbonate is replaced effectively and a serious base deficit does not develop. When there is poor renal blood flow due to hypovolemia/starvation, base deficit and acidosis develop rapidly. o Acidosis also results from excessive production of lactic acid when patients have severe diarrhoea. Other abnormalities Osteopenia and osteoporosis (with heightened fracture risk) Enlarged cerebral ventricles and external cerebrospinal fluid spaces (pseudo atrophy)

Calculating BMI: BMI = Weight in kg / (height in meters)2. e.g. if weight = 50kg and height is 165cm, then BMI = 50 / (1.65) (1.65) = 50 / 2.7225 = 18.36. BMI categories: Underweight = <18.5; Normal weight = 18.5-24.9; Overweight = 25-29.9; Obesity = BMI of 30 or greater Alcohol use disorders No single lab test can dependably diagnose alcohol abuse. Of available tests such as GGT (a liver enzyme), Mean Corpuscular Volume, Breathalyzer and Carbohydrate Deficient Transferrin (CDT), the CDT is the single most specific and sensitive test for detecting heavy alcohol use over last 10 days. But due to a high degree of intersubject variability it is best to compare CDT levels with patient’s own baseline. In primary care, AUDIT is often considered to be the best screening tool. As alcohol abuse is associated with systemic complications, several other lab tests may be required when these complications are suspected. System involved Complications Neurological Seizures (intoxication or withdrawal), Wernicke's encephalopathy, Korsakoff's dementia, polyneuropathy, coma, amnesia, alcoholic dementia, cerebellar degeneration, damage to corpus callosum (Marchiafava syndrome) Gastrointestinal GI bleeds, peptic ulcer, malnutrition (esp. thiamine), Mallory-Weiss tears, esophageal strictures, fatty liver, hepatic cirrhosis, portal hypertension, pancreatitis and hypoglycaemia Cardiovascular Cardiomyopathy, hypertension, hyperlipidemia Hematologic Pancytopenia, folic acid and B12 deficiency resulting in MCV changes and anaemia, clotting disorders due to liver failure, immune compromise Respiratory Lung cancer, pneumonia due to aspiration under intoxication Musculoskeletal Muscle wasting, osteoporosis Renal Renal failure, hepatorenal syndrome, hyponatremia and other electrolyte imbalances Endocrine Testicular atrophy, sexual disorders, menstrual irregularities Pregnancy Low birth weight, foetal alcohol syndrome, developmental delays, neural tube

05 - Specific investigations

Specific investigations

 defects due to maternal malnourishment.

Specific investigations

ELECTROCARDIOGRAM: The major use of ECG in a psychiatric ward, apart from emergency needs, is to measure QT interval when treating patients using antipsychotics. Prolonged QT can predispose to fatal ventricular arrhythmias such as torsades de pointes (polymorphic ventricular tachycardia). QTc is QT corrected for heart rate. While valuable for classifying risk groups, it is not a precise predictor of torsade de pointes as it has low positive predictive value. There are different methods to arrive at QTc from QT – these give markedly different values. As a clinical measure, the risk is said to increase if QTc is beyond normal limits (440 ms for men; 470 ms for women) – anything more than 500 ms is clearly an increased risk. QT varies with gender, time of day, food intake, alcohol intake, menstrual cycle, ECG lead used. Risk factors for prolonged QTc include • Congenital long Q-T syndrome, • Underlying heart disease, bradycardia, heart failure, and ischemic disease • Female gender, • Extremes of age, • Presence of liver disease, • Electrolyte abnormalities (hypokalemia, hypocalcemia and hypomagnesemia), • Illicit drug use (principally stimulants), • Starvation or anorexia, • High physical exertion (agitation), • High dosages of the drug contributing to the lengthened Q-T interval, and • Rapid infusion of torsadogenic drugs.
URINALYSIS: Testing for drugs: This is one of the most frequently used lab investigations in psychiatry. When a patient repeatedly gives negative urine samples despite strong suspicions, a cheap and quick way of checking the sample is by testing specific gravity – this will reveal any adulteration of urine with tap water. The following table will help answering some recurrent questions on this theme.

 Substance Time present in urine Alcohol Up to 12 hrs Amphetamine Up to 48 hours Benzodiazepine 3 days (depending on t1/2) Cannabis Occasional use – up to 3 days. High daily use for long time – up to 4 weeks. Cocaine 6 – 8 hrs; metabolites up to 2 - 4 days Codeine 48 hours Heroin 1 to 3 days Methadone 3 days or more Morphine 2 to 3 days Phencyclidine (PCP) 8 days Data from Oxford Handbook of Psychiatry & Rudolph’s Paediatrics 21e. p 230 Renal disturbances in IV drug users: Renal disease in cocaine and heroin abusers has been associated with the nephrotic syndrome, acute glomerulonephritis, amyloidosis, interstitial nephritis, and rhabdomyolysis. In a heroin user with a puffy face, hypertension and weight gain – suspect heroin-related nephropathy. Infective endocarditis, HIV, and HBV and HBC infections are associated with renal pathologic patterns similar to those that can be caused by the drug itself. In Black patients, focal segmental glomerulosclerosis is often seen while in Whites mostly membranoproliferative glomerulonephritis is noted. SIADH: Urine analysis may be important with regard to SIADH

induced by antidepressants or antipsychotics/Psychogenic polydipsia where excessive water consumption occurs without obvious organic illness and Diabetes insipidus due to lithium (nephrogenic) or head injury (central). As a rough guide use the following tables. Plasma osmolality Urine osmolali Diagnosis High (>295mosm/kg) Low Diabetes Insipidus (Central / nephrogenic) Low (<280 mosm/Kg) Low Psychogenic polydipsia Low High SIADH - hyponatraemia Psychogenic / Primary polydipsia Diabetes Insipidus Gradual onset Acute or sudden Nocturia is rare Nocturia is common Plasma osmolality normal/low Elevated plasma osmolality Urine osmolality normal/low Low urine osmolality

 Plasma ADH levels normal compared to osmolalit Low in central type NOTE - polydipsia and polyuria are not features of SIADH or hyponatraemia per se. The clinical features of SIADH are attributed to water retention, hyponatraemia, and hypo-osmolality of the serum. Most hyponatraemic patients have no symptoms or signs until the serum sodium concentration falls below 125 mmol/L. Initially, the symptoms include lethargy, muscle cramps, anorexia, nausea, and vomiting. When hyponatraemia develops more rapidly or more profoundly, coma, convulsions, and death may occur. On longer term hyponatraemia can cause neurologic signs and symptoms such as altered levels of consciousness, headache, impaired memory and confusion. If the serum sodium concentration drops below 110-115 mmol/L, seizures and irreversible brain damage can occur.

06 - 3. Physical examination of a psychiatric pati

3. Physical examination of a psychiatric patient

07 - General examination

General examination

© SPMM Course 3. Physical examination of a psychiatric patient General examination SIGNS Relevant conditions Argyll-Robertson pupil Neurosyphilis; the more common cause is diabetes. Checker-board abdomen Multiple surgical scars in factitious disorder. Constricted pupils Opiate intoxication, Horner’s syndrome Dilated pupils Stimulant abuse or opiate withdrawal, anxiety states Kayser Fleischer ring Golden Brown pigment around cornea in Wilson’s disease Generalised lymph node enlargement HIV illness, Lymphomas. Goitre Thyroid disease, very small number related to lithium use Gynaecomastia Hyperprolactinaemia, cirrhosis, androgen or steroid abuse Jaundice Heavy alcohol use. Lanugo hair Anorexia nervosa Lemon stick appearance, central obesity Cushing’s syndrome Lid lag, lid retraction, exophthalmia, and proptosis Hyperthyroid state Mask like face Extrapyramidal affect is seen in Lewy body dementia, Parkinsonism, and in psychomotor retardation of depression Parotid swelling Bulimia, mumps Piloerection Opiate withdrawal Rapid/irregular pulse Anxiety, delirium states, Drug/alcohol withdrawal and Hyperthyroidism Russell's sign Bulimia nervosa – calluses at knuckles Sialorrhoea Clozapine treatment; parkinsonism; facial palsy of Bell’s, stroke involving cranial nerves Splinter haemorrhages, Osler’s nodes, and Janeway lesions Due to infectious endocarditis in IV drug users. Xanthelasma Lipid accumulation, related to Olanzapine or another antipsychotic treatment. Patients with acute hyperventilation (often in the context of panic attack in a psychiatric clinic) may present with agitation, increased breathing rate with shallow breaths (tachypnoea), chest pain, dizziness, palpitations, tetanic cramps (carpopedal spasm), paresthesias, generalized weakness, and syncope. Paresthesias are due to acid-base imbalance, and occur more commonly in the upper extremity and are usually bilateral. Unilateral paresthesias are left-sided in approximately 80% of cases. Perioral numbness is very common. Minor Physical Anomalies (MPAs) are often observed in a range of developmental disorders. MPAs are also more frequent in patients and siblings of patients with schizophrenia than in healthy controls, supporting neurodevelopmental aetiology. MPAs can be rated using Lane Scale

08 - Neurological examination in psychiatry

Neurological examination in psychiatry

© SPMM Course Neurological examination in psychiatry Cranial nerves examination: No. Name Main clinical examination technique I Olfactory Smell – each side separately II Optic Test visual acuity using Snellen’s charts (near and distance), colour using Ishihara charts, field by confrontation/perimetry and pupillary reflexes. III Oculomotor Eyelid elevation, eye elevation, adduction, depression in abduction, pupillary reflex for efferent fibres IV Trochlear Eye intorsion, depression in adduction V Trigeminal Facial and corneal sensation, muscles of mastication VI Abducens Eye abduction VII Facial Facial movement, taste fibres VIII Vestibular Balance – Romberg / Caloric test Minor Physical Anomalies in putative developmental disorders Preauricular tag Preauricular pits Lip pit Bifid uvula Supernumerary nipples: Partial syndactyly (generally involving toes 2–3) Pigmented naevi Cafe-au-lait spots Sacral haemangioma Prominent or flat occiput Prominent or flat forehead Primitive shape of ears Earlobe crease Fine electric hair Tongue with smooth and rough spots Double antihelix Simian crease [Instead of the two usual creases only a single uninterrupted palmar crease traverses the palm from the radial to the ulnar border. To be considered as an anomaly, the line should be uninterrupted]. Single flexion crease on 5th finger Sole crease Prominent heel Double posterior hair whorl Multiple buccal frenula Furrowed tongue Brushfield spots

© SPMM Course Cochlear Hearing – Rinne, Weber tests. IX Glossopharyngeal Sensation - soft palate, taste fibres X Vagus Cough, palatal and vocal cord movements XI Accessory Head turning, shoulder shrugging XII Hypoglossal Tongue movement Adapted from Kumar & Clark Textbook of clinical medicine 6th edition Pg 1179 The auditory function is tested using 512 Hz – Weber’s test and Rinne’s test; vibration sense is tested for peripheral neuropathy using a 128Hz fork. The Weber test involves holding a vibrating tuning fork against the forehead in the midline. The vibrations are normally perceived equally in both ears because bone conduction is equal. In conductive hearing loss, the sound is louder in the abnormal ear than in the normal ear. In sensorineural hearing loss, lateralization occurs to the normal ear. In the Rinne test, the vibrating tuning fork is placed over the mastoid region until the sound is no longer heard. It is then held at the opening of the ear canal on the same side. A patient with normal hearing should continue to hear the sound. In conductive hearing loss, the patient does not continue to hear the

sound since bone conduction, in that case, is better than air conduction. In sensorineural hearing loss, both air conduction and bone conduction are decreased to a similar extent. The vestibular portion transmits information about linear and angular accelerations of the head from the utricle, saccule, and semicircular canals of the membranous labyrinth to the vestibular nucleus. The Romberg test is performed to evaluate vestibular control of balance and movement. When standing with feet placed together, and eyes closed, the patient tends to fall toward the side of vestibular hypofunction. Results of the Romberg test may also be positive in patients with polyneuropathies, and diseases of the dorsal columns, but these individuals do not fall consistently to one side as do patients with vestibular dysfunction. Provocative tests include caloric testing. Normally on cold water testing, nystagmus is noted to the opposite side; warm water elicits nystagmus towards the same side. (COWS – Cold Opposite, Warm Same, can be used as a mnemonic) Neurological soft signs: Neurological signs are often referred to as either “hard” or “soft” signs. The ‘hard signs’ refer to impairments of the basic motor and sensory functions that are localisable to the pyramidal, extrapyramidal or cranial nerve systems.

© SPMM Course The soft signs are non-localisable neurological findings thought to reflect neurodevelopmental aberrations when seen in psychiatric disorders. These are seen in many psychiatric disorders including schizophrenia, autism, OCD and ADHD. However, this distinction between hard and soft signs is artificial, merely reflecting our inability to define the brain-behaviour relationship that underlies certain neurological abnormalities. There are three groups of symptoms collectively known as soft signs - abnormalities of motor coordination, sensory integration and signs of cortical disinhibition. In recent times, neuroimaging studies that parse finer details of the cortex have implicated several parts of the brain in ‘soft’ signs, further blurring their distinction from hard signs.

Cerebellar signs The cerebellum provides an important feedback loop for coordination of muscle activity. Midline cerebellar dysfunction results in ataxia of gait, difficulty in maintenance of upright posture, and truncal ataxia. The following cerebellar signs are noted in various degrees in psychiatric disorders. The lateral cerebellar hemispheres (the neocerebellum) controls the movement of the ipsilateral limbs. The midline vermis is involved in the control of truncal tone, speech and eye movements. The flocculonodular lobe (also called archicerebellum) is involved in vestibular functions. Cerebellar signs Ataxia Difficulties in coordinating truncal and limb movements, often seen in midline damage. Tested using tandem walking (heel-to-toe walk) test. Hypotonia Reduced muscle tone resulting in loss of ‘checking’ effect when passively manipulated (leg swinging test results in pendular swinging of legs until passive inertia sets in) Intention tremor An oscillating tremor that accelerates in pace on approaching the target Dyssynergia (incoordination) Results in loss of smoothness of execution of a motor activity. Dysmetria (past pointing) Overshooting or undershooting of a target while attempting to reach an object Common soft neurological signs in psychosis Choreoathetosis (predating psychosis esp. in children) Abnormal gait Grimacing Abnormal reflexes Changes in muscle tone Abnormal saccades Frequent blinking Dysdiadochokinesia Astereognosis Poor left-right discrimination Anosognosia Apraxia Gaze impersistence Frontal release

© SPMM Course Dysdiadochokinesis Inability to perform rapid alternating movements. Tested by asking the patient to tap 1 hand on the other repeatedly while simultaneously pronating and supinating the hand Dysrhythmia Inability to tap and keep a rhythm Dysarthria Staccato or

scanning speech with poor modulation of the volume and pitch of the speech. Dyssynergia, dysmetria and tremor can be elicited by finger nose or heel shin test. Dysarthria is usually a sign of diffuse involvement of the cerebellum. Meningeal signs: These signs can be elicited in the presence of meningeal inflammation or irritation due to haemorrhage/trauma. Nuchal rigidity or neck stiffness: The Brudzinski sign (Flexion of his knees and hips when you try to flex one's neck constitutes a positive Brudzinski's sign.) The Kernig sign (this is elicited by flexing one hip and knee and then extending the knee with the hip still flexed). Hamstring spasm may occur; if severe, opposite knee may flex during the test – positive Kernig's) The Lasègue or straight-leg raising (SLR) sign is elicited by passively flexing the hip with the knee straight while the patient is in the supine position. Limitation of flexion due to hamstring spasm and/or pain indicates local irritation of the lower lumbar nerve roots. Reverse SLR sign is elicited by passively hyperextending the hip with the knee straight while the patient is in the prone position. Limitation of extension due to spasm and/or pain in the anterior thigh muscles indicates local irritation of the upper lumbar-nerve roots. Cortical sensory signs: The cortical sensory system includes the somatosensory cortex and its central connections. Functions include kinaesthetic sensation, stereognosis, graphesthesia and tactile localization and tactile 2-point discrimination on both sides of the body. Position sensation is tested with the subject's eyes closed. The subject is then tested in the various directions of passively elicited distal joint movements. Movement abnormalities: Fibrillations are not visible to the naked eye except when the tongue is affected.

  Fasciculations may be seen under the skin as quivering of the muscle. Although fasciculation is usually benign (e.g. can occur with fatigue); if widespread it can be associated with neuromuscular disease, including amyotrophic lateral sclerosis (ALS). Asterixis can be elicited by having the patient extend both arms with the wrists dorsiflexed and palms facing forward, and eyes closed. Brief jerky downward movements of the wrist are considered a positive sign. Asterixis is commonly seen in metabolic encephalopathies. (Note pronator drift is elicited by having the patient extend both arms with the wrists supinated and palms facing upwards and eyes closed – slow unequal drift towards pronation indicates hemiparetic weakness) Myoclonus is a brief <0.25 seconds muscle jerk; generalized and sometimes asymmetric. These occur alone or in association with various primarily generalized epilepsies; associated with CJD and also with severe Alzheimer's. In athetosis, the spasms have a slow writhing character and occur along the long axis of the limbs or the body itself; the patient may assume different and often peculiar postures. The term chorea means dance. Quasi-purposeful (patient turn it to appear as if they are purposive) movements affect multiple joints with a distal preponderance. It is associated with caudate lesions. Hemiballismus is a violent flinging movement of half of the body. It is associated with lesions of the subthalamic nucleus. Reflexes Primitive reflexes: These include the glabellar tap, rooting, snout, sucking, and palmomental reflexes. These are generally absent in adults. When present in the adult, these signs signify diffuse cerebral damage, particularly of the frontal lobes (hence the name frontal-release signs). Superficial reflexes: These are segmental reflex responses that indicate the integrity of cutaneous innervation and the corresponding motor outflow. These include corneal, conjunctival, abdominal, cremasteric and plantar (Babinski) reflexes. Corneal and conjunctival reflexes – afferent is via 5th nerve while efferent is via 7th nerve. Abdominal reflex can be elicited by drawing a line away from the umbilicus along the diagonals of the 4 abdominal quadrants. A normal reflex draws the umbilicus toward the direction of the line that is drawn. The cremasteric reflex is elicited by scratching on the medial surface of the thigh to elicit scrotal contraction or lift in male subjects. A normal reflex results in elevation of the ipsilateral testis.

© SPMM Course The best known of this group of reflexes is the plantar reflex or Babinski reflex. The normal response is plantar flexion of the great toe. This normal response is considered an absent (negative) Babinski sign. Dorsiflexion of the great toe (Babinski sign present) suggests an upper motor neuron lesion and is referred to as a positive Babinski sign. Lack of either response may indicate the absence of cutaneous innervation in the S1 segment or loss of motor innervation in the L5 segment ipsilaterally. Deep tendon reflexes: Intact cutaneous innervation, motor supply, and cortical input to the corresponding spinal segment are required for normal deep tendon reflexes. Deep tendon reflexes include biceps, brachioradialis, triceps, patellar, and ankle jerks. These get exaggerated in UMN lesions and are absent in respective LMN lesions. Pseudobulbar palsy is a UMN lesion; exaggerated jaw jerk is noted in patients with this condition. Bulbar palsy is a result of LMN lesion and jaw jerk is absent in this case. Neurocutaneous system Frontal baldness: Myotonic dystrophy Dermatomal eruptions: Herpes Zoster Ash leaf macules: Tuberous sclerosis Ungual fibromas: Tuberous sclerosis Dimples and large moles along the spine: spina bifida occulta Cafe au lait spots: Neurofibromata, Tuberous sclerosis. Axillary freckling: Neurofibroma Speech abnormalities Dysarthria Types Description Spastic dysarthria Strained and hoarse voice, hypernasality and slow, imprecise articulation related to bilateral UMN lesions. Often accompanied by swallowing and drooling difficulties (Palmer 2005). Flaccid dysarthria Isolated areas of involvement are depending on which motor neurone is affected. LMN type lesion. The tongue is usually small due to loss of tone if XII nerve is involved and lies flaccidly on the floor of the mouth. Could be of nasal quality if IX and X nerves are involved. Ataxic dysarthria Excess loudness, tremor and irregular articulatory breakdowns (scanning speech). Intonation, pitch and volume and also be affected, as well as difficulty with alternate tongue movements. The cerebellum is often involved. Hypokinetic dysarthria A breathy monotone voice with reduced loudness and articulation tends to be accelerated and imprecise. Associated with motor control circuit Muscle Spinal Roots Biceps C5, 6 Brachioradialis C6 Triceps C7 Patellar L2-4 Achilles S4

© SPMM Course damage. Hyperkinetic dysarthria Features strained hoarseness and voice arrests. Associated with basal ganglia damage. Mixed dysarthria Similar symptoms to spastic dysarthria, and tends to be accompanied by a wet sounding voice with rapid tremor, poor laryngeal and tongue movements and poor control of lips (Damage to more than one motor system). Hysterical aphonia The examination is usually normal. Sudden loss of voice, but preserved vocal cord activity is notable.

Gait Gait is the motor attitude of a person in the upright position. Hemiparetic gait: Seen in patients with stroke affecting the pyramidal system. Typically, clenched hand with extended knee and plantarflexed ankle. This makes the paralyzed leg appear longer (pole-like) than the other. The patient resorts to circling it around resulting in repeated circumduction of the affected leg while walking. Ataxic gait: In mild cases this can only be elicited by or tandem walking tests. In severe cases, a staggering wide-based gait is seen. Unilateral (rather than midline) cerebellar lesions may result in the patient veering to the side of the lesion (resulting in sailor’s gait). Shuffling gait: This is often seen in Parkinsonian patients. The patient takes very short steps and appears to shuffle legs away or apart rather than propelling them forward. Progressively short steps result from a tendency of the patient to accelerate (festinating gait). Steppage gait: Here the patient takes high steps as if climbing a flight of stairs while walking on a level surface. Steppage gait is seen in chronic peripheral neuropathies e.g. drop foot and dorsal column disorders. Waddling

gait: It is seen in patients with proximal myopathy. Patients have a broad-based gait with a duck-like waddle resulting from the dropping of the pelvis to the side of the leg being raised. A compensatory forward curvature of the lumbar spine adds to the body swing. This is also be seen in patients with congenital hip dislocation and near term in pregnant women. Scissoring gait: This is seen in patients with spastic paraplegia. Marked rigidity and excessive adduction of the swinging leg together with plantar flexion of the ankle and flexion at the knee due to contractures of all spastic muscles leads to forced tip-toe walking with knees rubbing together and crossing like scissors.

09 - Other neurological signs

Other neurological signs

© SPMM Course Other neurological signs Absent ankle jerks, upgoing plantars: This is an odd combination - UMN lesion of corticospinal tracts is expected to cause exaggerated ankle reflex (i.e. clonus) with upgoing plantar normally. But in subacute combined degeneration cord, Syphilitic taboparesis and Friedrich's ataxia and MND we see absence of ankle jerk as spinal reflex pathway is affected (afferent) while UMN type damage still produces Babinski – upgoing plantar. Anisocoria: This refers to pupillary asymmetry, which may result from sympathetic or parasympathetic dysfunction. Sympathetic dysfunction results in Horner syndrome, in which the pupil is small but reacts to light. Parasympathetic dysfunction results in the tonic pupil. Argyll-Robertson pupil, seen in neurosyphilis, is irregular and small; it does not react to light, but does accommodate. Anosognosia refers to the denial of illness and typically is seen in patients with right frontoparietal lesions, resulting in left hemiplegia that the patient denies. Asterixis involves momentary loss of tone and flapping of the hand are seen when the patient extends his arms in front with the wrists dorsiflexed. This is seen in patients with metabolic encephalopathies GAIT Conditions Antalgic gait Trauma, Osteoarthritis Broad, unsteady gait (Drunken/sailor’s gait) Cerebellar lesions Festinating/shuffling gait Parkinson’s Gait apraxia (Magnetic gait or failed gait ignition) Hydrocephalus High stepping due to foot drop Neuropathic / polio / peripheral lesions in MS Lurching, chaotic gait Huntington’s disease Pigeon gait Torsional abnormalities seen in hip dysplasias Propulsive gait Carbon monoxide poisoning (stiff with head and neck bent) Stiff, scissoring gait UMN lesions, cerebral palsy, cortical lesions in MS or stroke Stomping gait Friedreich's ataxia Pernicious anaemia, Tabes Dorsalis (Syphilis) Trendelenburg gait Weakness of the abductor muscles of the lower limb, principally gluteus medius Waddling myopathic gait Pregnancy, proximal myopathy.

© SPMM Course Beevor sign is seen with bilateral lower abdominal paralysis that results in upward deviation of the umbilicus when the patient tries to raise his head and sit up from the supine, recumbent position. Brown Sequard syndrome is due to hemisection of the spinal cord; the full syndrome is rare. Clinical features are related to various tracts that are severed.

 Chvostek sign is seen in hypocalcemia. Tapping the cheek at the angle of the jaw precipitates tetanic facial contractions. Doll 's eye maneuver: This refers to turning the head passively with the patient awake and fixated or when the patient is in a coma. In the former, the eyes remain fixated at the original focus when all gaze pathways are normal; in the latter, the eyes deviate in the opposite direction when the brainstem is intact. Friedreich’s ataxia is an inherited neurological disease (trinucleotide repeat) with pes cavus, kyphoscoliosis, cerebellar signs, impaired joint position / vibration, cardiomyopathy, optic atrophy. Gower sign: This sign, seen in severe myopathies, occurs when the patient attempts to stand up from the floor. Patients first sit

up, then assume a quadruped position, and then climb up their own legs by using their arms to push themselves up. Holmes-Adie syndrome - A benign form of the tonic pupil is seen in Holmes-Adie syndrome, i.e., a tonic pupil with absent patellar and Achilles reflexes. Horner's syndrome: Remember PAMELA – Ptosis, Anhidrosis, Miosis, Enophtholmos and Loss of ciliospinAl reflex. This collection of signs indicates a lesion of the sympathetic pathway on the same side. Seen in cervical lesions –e.g. apical lung tumour affecting cervical sympathetic ganglion, carotid aneurysms. Kayser-Fleischer ring: This is a brownish ring around the limbus of the cornea. It is best demonstrated by an ophthalmologic slit lamp examination. Marcus-Gunn pupil: This sign requires a swinging flashlight test to assess. As the flashlight swings from 1 eye to the other, the abnormal pupil dilates as the light swings back from the normal side. No anisocoria is seen. The phenomenon is also called a paradoxical pupillary reflex and indicates an afferent (optic nerve) pupillary defect. Lateral corticospinal damage Lateral corticospinal damage •Ipsilateral spastic paralysis below the level of the lesion •Babinski sign ipsilateral to lesion •Abnormal reflexes (UMN type hyperreflexia) Posterior column damage Posterior column damage •Ipsilateral loss of tactile discrimination, vibratory, and position sensation below the level of the lesion Lateral spinothalamic damage Lateral spinothalamic damage •Contralateral loss of pain and temperature sensation. This usually occurs 2-3 segments below the level of the lesion.

  Mononeuritis multiplex: Painful asymmetric asynchronous sensory and motor peripheral neuropathy with isolated damage to at least 2 separate nerve areas. Causes: diabetes, vasculitis, amyloidosis, direct tumor involvement, autoimmune disorders paraneoplastic syndromes. Milkmaid's grip: This refers to the inability to maintain a sustained grip commonly seen in patients with chorea. Myerson sign: Patients with Parkinson disease, particularly those with bilateral frontal lobe dysfunction, continue to blink with repeated glabellar taps. Optic neuritis: The classic triad of optic neuritis consists of (1) loss of vision, (2) eye pain, and (3) dyschromatopsia. 70% unilateral. Usually recover spontaneously (Multiple sclerosis) within 2-3 weeks. Movement- or sound-induced phosphenes are seen. Reduction in vision may worsen in bright light, a symptom that seems paradoxical. The Uhthoff symptom, described as exercise- or heat-induced vision loss is seen in 50% of patients. Afferent pupillary defect is noted on testing (i.e. direct light reflex absent; consensual present) Subacute combined degeneration is due to vitamin B12 deficiency; causes peripheral neuropathy, posterior column signs with pyramidal signs below the waist. Trombone tongue: This is seen in patients with chorea. It refers to the unsteadiness of the tongue when the patient tries to protrude it outside the mouth.

Rigidity Hypertonia Exaggerated reflexes Mild atrophy (disuse) e.g. pseudobulbar palsy Atonia or hypotonia Loss of deep tendon reflexes Atrophic, wasted Fasciculatione.g. bulbar palsy UMN Lesion UMN Lesion LMN Lesion LMN Lesion

10 - Bedside cognitive examination tools

Bedside cognitive examination tools

© SPMM Course Bedside cognitive examination tools (This section is best read in conjunction with the section on neuropsychological tests in the Applied Neuroscience chapter and the chapter on Rating Scales) MMSE: The Mini-Mental State Examination (MMSE) is the standard screening instrument for dementia introduced by Folstein in 1976. It takes 5–10 minutes to administer and has a median positive Likelihood Ratio of 6.3 and a median Negative Likelihood Ratio of 0.19. Brief tool for grading cognitive impairment in elderly and screening form dementia. Not very sensitive to change, but used in anti-dementia drugs' clinical trials. ADAS-Cog may be better suited to detect change. Practice effect may occur with MMSE. It is a 30point scale With less than 9 years of formal education, the cut off for suspecting dementia must be 21/22 and not the usual 23/24. Insensitive to early decline. Doesn't pick up frontal executive defects Bulbar Palsy Bulbar Palsy •LMN weakness of 9-12 cranial nerves •Wasted, fasciculating tongue •Nasal speech •Lost jaw jerk and gag reflex •emotional lability not seen •MND, polio, botulism, myasthenia gravis, muscular dystrophies Pseudobulbar palsy Pseudobulbar palsy •bilateral supranuclear (UMN) lesions of lower cranial nerves •Stiff tongue; wasting seen only in later stages •Donald-duck speech •Exaggerated jaw jerk; preserved gag reflex •emotional lability (pathological emotionalism) •MND, multiple sclerosis, multiinfarch dementia and severe head injury.

© SPMM Course ITEMS in MMSE o Orientation (10) o Registration (3) and recall (3) tasks (6 points total) o Attention task (5) o Multistep command (3) o Naming (2) o Repetition language (1) o Reading comprehension (close your eyes, 1 point) o Writing (1) o Visual construction (copy interlocking polygons, 1 point) Clinical interview with carers and patients is the best diagnostic tool for any disorder including dementia; overreliance on MMSE scores can be counterproductive. The clock drawing test: Clock drawing test requires verbal understanding (comprehension), short-term working memory to process the instruction and spatially coded knowledge in addition to constructive skills and planning (executive function). (It does not test orientation to time!) Watson introduced a 7 scores screening method with a good degree of reliability. The placing of any three digits in a quadrant is considered to be correct. An error score of one is assigned to each of the first three quadrants containing any errors, and an error score of four is assigned for the fourth quadrant if it contains an error. Thus, a maximum error score of seven can be obtained. The

normal range for the score is 0-3. A score of 4 or greater in this scoring system has a sensitivity of 87%, a specificity of 82% and a kappa value of 0.70 for identifying dementia (according to the NINCDS-ADRDA criteria for probable dementia). The test has a high correlation with the MMSE and other tests of cognitive dysfunction. It can also be used in diagnosing unilateral neglect and inattention. Subjects of low education, advanced age and depression perform more poorly. There are many methods of administering and scoring. Normal clock-drawing ability reasonably excludes cognitive impairment

Addenbrooke's cognitive examination (ACE-Revised): ACE-R evaluates six cognitive domains (orientation, attention, memory, verbal fluency, language and visuospatial ability). It is useful for detecting dementia and mild cognitive impairment.

  Frontal tests such as verbal fluency are also included in the ACE, making it more sensitive to frontal types of dementia than MMSE. (Hodges R et al., 2000). It is also effective for differentiating the subtypes of dementia, such as Alzheimer's disease, frontotemporal dementia, progressive supranuclear palsy, and other forms of dementia associated with parkinsonism (Rittman et al., 2013). The normative data provided with ACE-R (revised version) states that there are two defined cut-offs (<88: sensitivity=0.94, specificity=0.89; <82: sensitivity=0.84, specificity=1.0). The likelihood ratio for a positive test of dementia at a cut-off of 82 is 100:1. Language domain receives the major share of the scoring in ACE.

11 - 4. Imaging of the nervous system

4. Imaging of the nervous system

© SPMM Course 4. Imaging of the nervous system Computed Tomography – CT The most widely available scan in clinical practice CT scanners effectively take a series of head X-ray pictures from 360 degrees around a patient's head. The CT image contrast is determined by the degree to which tissues absorb X-rays. Structures close to bone may appear obscured in a CT image e.g. brainstem The difference in the attenuation between gray matter and white matter is not very high. CT is limited to one plane of rotation – often axial. Appreciation of tumours and areas of inflammation is possible by intravenous infusion of iodine-containing contrast agents. Iodinated compounds in the vascular compartment absorb much more irradiation than the brain tissue and so appear bright. One feature that is better visualized on CT scanning is calcification, which may be invisible in MRI. CT scans and MRI are the most common neuroimaging tools used in psychiatry. The CT is widely available with shorter scan duration at a low cost, but exposure to radiation is a disadvantage. CT has poor sensitivity to early ischemia and has poor visualization capacity for posterior fossa lesions. Magnetic Resonance Imaging – MRI MRI does not rely on the absorption of X-rays but is based on nuclear magnetic resonance (NMR) principle. MRI magnets are rated in Tesla (T) units of magnetic field strength. The nuclei of all atoms spin about an axis that is randomly oriented in space. When atoms are placed in a magnetic field, the axes of all odd-numbered nuclei (H1 in particular) align with the magnetic field. This axis deviates away from the magnetic field when exposed to a pulse of radiofrequency electromagnetic radiation oriented at 90 or 180 degrees to the magnetic field. When the pulse terminates, the axis of the spinning nucleus realigns itself with the magnetic field, and during this realignment, it emits its own radiofrequency signal. MRI scanners collect these signal emissions. The images can be in the axial, coronal, or sagittal planes. The rate of the realignment of the H1 axis is determined by its immediate environment and the degree of water content. Hydrogen nuclei within fat realign rapidly, and hydrogen nuclei within water realign slowly. Routine MRI studies use 2 different radiofrequency (RF) pulse sequences: T1 and T2. T1 images:

© SPMM Course The RF pulses are brief, and data collection is brief Hydrophobic environments are emphasized i.e., fat is bright on T1, and CSF is dark. The T1 image most closely resembles that of CT scans and is most useful for assessing overall brain structure. T1 is

also the only sequence that allows contrast enhancement with the contrast agent gadolinium-diethylenetriamine pentaacetic acid (gadolinium-DTPA). On T1 images, gadolinium-enhanced structures appear white. T2 images This RF pulse lasts four times as long as T1 pulses, and the collection times are also extended. Emphasizes signal from hydrophilic areas i.e. brain tissue is dark, and CSF is white on T2 images. Areas of the brain tissue that have abnormally high water content, such as tumors, inflammation, or strokes, appear brighter on T2 images. T2 images reveal brain pathology most clearly. The proton density sequence A short radio pulse is followed by a prolonged period of data collection, Useful to see periventricular structures Fluid attenuated inversion recovery (FLAIR) The T1 image is inverted and added to the T2 image to double the contrast between gray matter and white matter. Very useful for detecting sclerosis of the hippocampus caused by temporal lobe epilepsy and for localizing areas of abnormal metabolism in degenerative neurological disorders. MRI scans are contraindicated in patients with pacemakers or implants of ferromagnetic metals. Claustrophobia is a relative contraindication. MRI is less useful in emergencies due to limited availability and longer scan duration, in addition to higher costs. But it involves no radiation and can use water soluble Gadolinium for contrast studies. It has good sensitivity for early ischemia with better posterior fossa visualization. Structures / pathology CT scan T1 image T2 image Infarct Dark Dark Bright Bleed (haemorrhage) Bright Bright (unless too old / too fresh) Bright (unless too old / too fresh) Tumour Dark Dark Bright MS plaque Dark Dark Bright

12 - Functional Magnetic Resonance Imaging (fMRI)

Functional Magnetic Resonance Imaging (fMRI)

© SPMM Course Magnetic Resonance Spectroscopy –MRS MR spectroscopy can detect several biologically important nuclei with an odd number of protons and neutrons. H-1 proton spectroscopy can be used to quantify Nacetyl aspartate (NAA), creatine, and cholinecontaining molecules. GABA and glutamate can be detected using MRS but not dopamine as it is available in a very low concentration Phosphorus-31 MRS can be used to determine the pH of brain regions and the concentrations of phosphorus-containing compounds (e.g., adenosine triphosphate [ATP] and guanosine triphosphate [GTP]) that are important in the metabolic activity of the brain. Additional indications include the use of MRS to measure concentrations of psychotherapeutic drugs such as lithium in the brain. Some compounds, such as fluoxetine and trifluoperazine (Stelazine), contain fluorine-19, which can also be detected in the brain and measured by MRS. Functional Magnetic Resonance Imaging (fMRI) Neuronal activity within the brain causes a local increase in oxygen consumption. Consequently the local concentration of deoxyhaemoglobin increases, relative to oxyhaemoglobin. While oxyhaemoglobin is diamagnetic (weak magnetic contrast), deoxyhemoglobin is paramagnetic, producing an MR signal that can be detected with the T2 (demyelinated) CSF Dark Dark Bright Bone Bright Bright Dark Air Dark Dark Dark Fat Dark Bright Bright Tissue Shades of grey Grey matter – grey White matter - white Shades of grey MR molecule Potential clinical uses 1H Magnetic resonance imaging (MRI), Analysis of metabolism – NAA, creatine and choline. 19F Measurement of pO2, Analysis of glucose metabolism Measurement of pH, Pharmacokinetics 7Li Pharmacokinetics 31P Analysis of bioenergetics Measurement of pH 14N Measurement of glutamate, urea, ammonia 13C Analysis of metabolite turnover rate Pharmacokinetics of labelled drugs 17O Measurement of metabolic rate

13 - Single Photon Emission Computed Tomography SP

Single Photon Emission Computed Tomography - SPECT

© SPMM Course sequence. This is called Blood Oxygen Level Dependent (BOLD) technique. This process is the basis for functional MRI. fMRI is a proxy measure of tissue activity that depends on relative changes in perfusion; it does not measure the actual neuronal metabolism. No radioactive isotopes are administered in fMRI; this is a significant advantage over PET and SPECT. A subject can perform a variety of tasks, both experimental and control, in the same imaging session. In resting fMRI, the brain regions that have high levels of activity during rest are studied. These regions include the precuneus, lateral parietal regions and medial prefrontal cortex. A network of these regions showing higher baseline activity at rest is called default mode network or DMN. Single Photon Emission Computed Tomography - SPECT SPECT uses radioactive compounds to study regional differences in cerebral blood flow within the brain. This records the pattern of photon emission from the bloodstream which varies according to the level of perfusion in different regions of the brain. Similar to fMRI it does not measure neuronal metabolism directly. SPECT uses compounds labeled with single photon-emitting isotopes: iodine-123, technetium-99m, and xenon-133. Xenon-133 quickly enters the blood and is distributed to areas of the brain as a function of regional blood flow. Xenon-SPECT is thus referred to as the regional cerebral blood flow (rCBF) technique. Xenon-SPECT can measure blood flow only on the surface of the brain, which is an important limitation. Assessment of blood flow to the whole brain with SPECT requires the injectable tracers such as technetium-99m-d,l-hexamethyl propylene amine oxime (HMPAO). This is attached to highly lipophilic molecules that rapidly cross the blood-brain barrier to enter brain cells. Once inside the cell, the ligands are enzymatically converted to charged ions, which remain trapped in the cell. Thus, over time, the tracers are concentrated in areas of relatively higher blood flow. This is the ligand most commonly used in detecting perfusion changes in dementia. In addition to studying perfusion, Iodine-123 (123I)-labeled ligands for the muscarinic, dopaminergic, and serotonergic receptors can be used to study the occupancy and distribution of these receptors. Iodobenzamide is used for D1/D2 receptors; iomazenil is used for GABA-A

receptors; nor-β- CIT for dopamine and serotonin transporters; epidepride for D2/D3 receptors.

© SPMM Course Positron Emission Tomography – PET PET can be used to study blood flow, receptor distribution and metabolic activity of brain tissue. A key difference between SPECT and PET is that in SPECT a single particle is emitted, whereas in PET two particles are emitted; the latter reaction gives a more precise location for the event and better resolution of the image. The isotopes used in PET decay by emitting positrons, with the resolution closer to its theoretical minimum of 3 mm. Relatively few PET scanners are available because they require an on-site cyclotron to make the isotopes. The most commonly used isotopes in PET are fluorine-18, nitrogen-13, and oxygen-15. These isotopes are usually linked to another molecule, except in the case of oxygen-15 (15O). The most commonly employed ligand is [18F]fluorodeoxyglucose (FDG). FDG gives direct information about neuronal metabolism. Other molecules are listed in the table below. Diffusion tensor imaging – DTI DTI combines the principles of nuclear magnetic resonance and molecular diffusion. Diffusion refers to the random translational motion of molecules, also called Brownian motion, that result from the energy carried by these molecules. During their random, diffusion-driven displacements, molecules probe tissue structure at a microscopic scale well beyond the usual image resolution: the predominant direction of the molecular movement can help determine the integrity and trace white matter tracts. In traditional diffusion weighted images only 3 gradient directions are applied; DTI – diffusion tensor allows multiple (e.g. 16) gradients . From DTI, mathematical measures such as the Fractional Anisotropy (FA) can be calculated. This is an index of the integrity of white matter. The principal direction of the diffusion tensor can be used in tractography to infer the whitematter connectivity of the brain.

Purpose PET ligand Blood flow C15/H215O Glucose metabolism F18 deoxyglucose Dopamine D2 receptors 11C raclopride Dopamine neuron density 18F dopa; 18F metatyrosine GABA-A receptors 11C flumazenil 5HT2 receptors 18F altanserin; setoperone Striatal D2, cortical 5HT2 11C methylspiperone Serotonin synthesis rate 11C methyltryptophan Muscarinic receptors 11C scopolamine

© SPMM Course Neuroimaging findings in psychiatry: Neuroimaging findings in depression Periventricular and deep WM hyperintensities Subcortical – thalamic and striatal hyperintensities Decreased frontal and basal ganglia volumes Decreased metabolism in prefrontal cortex, Anterior cingulate & amygdale Higher prefrontal metabolism (esp. anterior cingulate) predict better treatment response Higher 5HT2A receptor density – higher dysfunctional negative thoughts Increased MAO-A activity (especially women) Elevated D2 binding in untreated depression – psychomotor retardation Therapeutic dose of SSRIs- 80% 5HT transporters occupied Neuroimaging findings in schizophrenia Ventricular enlargement Loss of grey matter – especially insular cortex, anterior cingulate (medial prefrontal cortex) and medial temporal lobe Progressive loss of brain volume in first few years of diagnosis fMRI reveals poor DLPFC activation in executive tasks Decreased NAA (N-Acetyl aspartate) in PFC (neuronal loss) in MRS Widespread reduction in DTI (diffusion tensor) – fractional anisotropy: frontal and corpus callosum – more in chronic treated patients Neuroimaging findings in Alzheimer’s

Ventricular enlargement Loss of temporal lobe volume – especially hippocampus Decreased parieto-temporal fMRI activation and SPECT blood flow Neuroimaging findings in OCD

Both reduced and increased volumes of caudate nuclei reported. Higher caudate blood flow due to increased metabolism. This reduces after effective treatment of the OCD. (Adapted from Murray, R, et al. (ed) Essential Psychiatry, Cambridge Press) Neuroimaging findings in Childhood-Onset Schizophrenia: Summary of key grey matter structural changes reported from Childhood-Onset Schizophrenia samples (Rapoport & Gogtay, 2011). In addition to what is shown, a ventricular enlargement at baseline and slower growth rates of (especially right hemispheric) white matter are also noted. From Hollis & Palaniyappan, Rutter's Child and Adolescent Psychiatry, Ed: Thapar et al...6e. Wiley & Sons.

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