# APEx Handouts - Candidates

Contents

[APEx Handouts - Candidates 1](#_Toc54781578)

[SESSION 1\_ABCD, AEIO, ABCD-AEIOU 2](#_Toc54781579)

[Session 1\_Michal’s case 3](#_Toc54781580)

[APEx proforma – primary assessment 3](#_Toc54781581)

[Session 2: Michal’s case 2 4](#_Toc54781584)

[Session 2\_Declan’s case 5](#_Toc54781585)

[De-escalation flowchart 5](#_Toc54781586)

[Session 2\_Mental state exam 6](#_Toc54781587)

[Session 2\_SBAR and Maudsley 7](#_Toc54781588)

[SBAR 7](#_Toc54781589)

[Maudsley structure 7](#_Toc54781590)

[Session 3\_David’s case 8](#_Toc54781591)

[Key points, comorbidities and mimics of alcohol intoxication 10](#_Toc54781592)

[Session 3\_Harriet’s case 11](#_Toc54781593)

[APEx proforma – definitive care and disposal 12](#_Toc54781594)

[Session 3\_Declan’s case 13](#_Toc54781595)

[Session 3\_Differential diagnosis 14](#_Toc54781596)

[Insidious onset 14](#_Toc54781597)

[Acute onset 15](#_Toc54781598)

[Strange behaviour … 16](#_Toc54781599)

[… Associated with Drug and Alcohol Misuse 16](#_Toc54781600)

[Illicit substance use 17](#_Toc54781601)

[Common psychiatric presentations 17](#_Toc54781602)

# SESSION 1\_ABCD, AEIO, ABCD-AEIOU





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# Session 1\_Michal’s case

|  |  |
| --- | --- |
|  | A 42 year old Polish man jumped 40 feet from scaffolding, resulting in a fracture to his right ankle. He has been treated medically and is medically fit to leave the ED. The man had worked as a labourer but had been unable to find work for several months prior to the episode, and was about to be evicted from his flat. He was estranged from his wife who was preventing him from having access to his 9 year old daughter, whom he had been unable to see for several months. He had been low in mood for several weeks prior to the self harm episode. He intended to die.  |

APEx proforma – primary assessment

|  |  |  |
| --- | --- | --- |
|  | Physical health | Mental health |
| PRIMARY | Physical assessment looking for organic cause | A |  | Primary AEIOassessment | **A**gitation/arousal |  |
| B |  | **E**nvironment |  |
| C |  | **I**ntent |  |
| D |  | **O**bjects |  |
| Risk to self?Risk to others?Flight risk? |  |
|  |  |
| Unified assessment. Immediate Treatment: Measures to minimise psychiatric or physical risk to patient or others |  |

# Session 2: Michal’s case 2

|  |
| --- |
| **Risk assessment** |
| SLIPA |  |
| Demographic and historical factors:  |  |
| Co-morbid mental illness:  |  |
| Overall risk profile. |  |

De-escalation flowchart



#

# Session 2\_Mental state exam

|  |
| --- |
| You are completing a Mental state examination for a patient. The person who is playing the role of the patient will respond to you using information provided to them. To elicit a mental state, you will need to take enough of a history of presenting complaint to be able to put some information into each of the boxes below. Appearance, behaviour and speech will be gleaned from the progress of the interview, as will your observations on their mood. You will need to get information on their thoughts about their mood, their thoughts, perceptions and insight from asking the patient questions |

|  |
| --- |
| **Mental state examination** |
| A -  |  |
| B -  |  |
| C -  |  |
| S -  |  |
| M -  |  |
| I – |  |
| T- |  |
| H- |  |

# Session 2\_SBAR and Maudsley

* Using a standardised communication structure minimised clinical error and improves communication
* For general clinical information we use SBAR
* For mental health mental state, it’s the Maudsley structure

SBAR

|  |  |
| --- | --- |
| **S**ituation |  |
| **B**ackground |  |
| **A**ssessment |  |
| **R**ecommendation |  |

Maudsley structure

|  |  |
| --- | --- |
| Appearance and Behaviour |  |
| Speech |  |
| Mood |  |
| Thoughts |  |
| Perceptions |  |
| Delusions |  |
| Cognition |  |
| Insight |  |

# Session 3\_David’s case

|  |  |
| --- | --- |
| Case 1: David is 42 years old. He has been brought into the ED by paramedics and police. He appeared drowsy and confused at the scene and vomited twice. Witness accounts mention an altercation outside the bar where he was found. Medical and mental health history is unknown at this stage. He is currently aggressive and refuses to be assessed. There are concerns about alcohol withdrawal – he appears sweaty and tremulous. |  |
| **Primary assessment** | ABCD | Maintaining own airway, RR 24. Refusing all other parts of ABCD examination. 5mg Olanzapine is given with good response. B - Oxygen saturations 98%, chest clear, good expansion bilaterally. C - clammy peripheries, HR 102bpm, BP 162/90, cap refill 2 secs, HS I+II+0D – GCS 12/15, dilated pupils, no focal neurology, Apyrexial, fingerprick glucose 2.8mmol/LE – haematoma, bruising, focal tenderness behind right ear. Two small fragments of glass embedded in scalp in same region.  | AEIO | * Significant agitation
* Currently managed in a cubicle with security and nursing support
* No immediate concerns about presentation being linked to self harm but cannot rule out overdose at this stage.
* Risk of harm to others – currently aggressive (verbally aggressive in ED and possible recent assaultative behaviour)
* Risk from others – retaliative behaviour from others if provoked
* No objects found on person that could pose risk to self or others

Risks centre on aggression towards others, risk of retaliative behaviour towards him, refusal of care interventions and absconsion if level of alertness improves |

|  |  |
| --- | --- |
| **Immediate treatment** | Priority is to complete ABCD – hypoglycaemia detected and treated promptly. Head wound is cleaned and dressed. CT head scan requested. Mental health – consider capacity, observation level, contingency plan for treatment of acute agitation: sedating antipsychotic such as Olanzapine 5mg/Haloperidol 2.5-5mg can be used until more is known. Both – Agree that parallel assessment is required and that patient currently lacks capacity to make informed decisions on investigations and treatment |
| **Secondary assessment:** |
| Focused physical history and secondary examination plus investigations | Currently complains of mild headache and some dizziness. Patient confirms diagnosis of type 1 diabetes with frequent episodes of hypoglycaemia. Remainder of systems examination is unremarkable.Breathylyser: 0.07% (moderate intoxication)Routine blood tests confirm hypoglycaemia. Urinalysis including drug screen – glucose and protein. Toxicology screen – unremarkable. ECG – normal. CT brain – no intracranial injuries or fractures. |
| Secondary mental health assessment | GCS improves to14/15 after glucoseNo history of cognitive impairment, major affective or psychotic disorder.David admits to getting into a fight with three men and has a history of convictions for assaultative behaviour when intoxicated. Mental state examination – No current concerns regarding mood. No psychotic symptoms. Some cognitive slowing – residual effect of intoxication, possible concussion.Describes a longstanding history of hazardous drinking. Patient confirms he has not taken any illicit substances or overdosed on prescribed medications. Willing to remain in hospital if required for treatment.  |

Key points, comorbidities and mimics of alcohol intoxication

Key points – approach (chapter 5)

* Establish timelines and evolution of presentation
* Medical, psychiatric, substance misuse history?
* Current risks?
* ABCD / AEIO U (establish priorities)
* Mental state exam
* Self harm, confusional state, psychosis?
* Does anyone else need to be involved – if so, at what stage?

|  |  |
| --- | --- |
| Comorbidities not to missHead injury, including concussionOther traumatic injuries (falls, altercations, deliberate self harm)SeizuresWernicke’s encephalopathyHallucinosisSuicidalityAspiration and respiratory tract infections CardiomyopathyGastritisPancreatitisHepatitis / cirrhosisCoagulopathyIngestion of illicit drugs | Mimics of alcohol intoxicationMeningitis / EncephalitisPost ictal statesCerebellar or brain stem infarct, TIA, bleed, inflammation (cerebellitis/MS)Encephalopathy (Wernicke’s, hypoxic-ischaemic, hepatic, uraemic)Traumatic brain injury – parenchymal damage, subdural or extradural haematoma, raised ICP, concussionHypoglycaemia/Diabetic ketoacidosisHypo/hypernatraemiaMyocardial infarctionDrug overdose – Benzodiazepines, Phenytoin, Carbamazepine, Gabapentin, Phenobarbital, Lithium, Amiodarone, Ciclosporin  |

# Session 3\_Harriet’s case

|  |  |
| --- | --- |
| Case 1: A 66 year old woman has been brought into the ED by paramedics. She was found semi clothed in her garden and appeared confused and distressed. She is a heavy smoker and has a history of type 2 diabetes, chronic kidney disease stage 3 and previous myocardial infarction. There is also a previous history of depression.  |  |

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Primary assessment** | ABCD | Maintaining own airway, RR 29, Oxygen saturations 88% room air, crepitations right lower and middle lung zones. HR 105bpm. Normotensive. Pyrexial 38C. Fingerprick glucose 9mmol/L. No focal neurology. No obvious trauma or signs suggestive of self harm | AEIOU | Moderate levels of agitation secondary to confusion, distress and physical discomfortRequires ongoing observation in current environmentNo concerns currently regarding active plans to self harm or harm others. Risk of self neglectRisk from others – vulnerabilityRisks centred on wandering, refusal of care interventions and vulnerability to others |
| **Immediate treatment** | * Physical health – Sepsis 6: Oxygen, IV fluids, antibiotics, VBG, blood cultures, fluid balance
* Mental health – consider capacity, observation level, contingency plan for treatment of acute agitation
* Both – Agree on preliminary working diagnosis – delirium secondary to respiratory tract infection
 |
| **Secondary assessment:** |
| Focused physical history and secondary examination plus investigations | 7 day history of lethargy, increasing shortness of breath, right sided pleuritic chest pain and cough with green sputumRoutine bloods and blood cultures sent. CXR – right side consolidation lower and mid zones. Urine dip – protein ++. Allergic to penicillin – Clarithromycin given instead. Consider CT head |
| Secondary mental health assessment | GCS 14/15, disoriented and confused, anxious and restless. There are no obvious symptoms or signs suggestive of hallucinations or paranoia at this time. Currently lacks capacity to make an informed decision regarding investigations, treatment and remaining in hospital for these |

APEx proforma – definitive care and disposal

|  |  |  |
| --- | --- | --- |
| DEFINITIVE CARE AND DISPOSAL | Disposal |  |
| Reassess risk |  |
| Handover to:

|  |
| --- |
|  |

including on-going care plan | **S**ituation |  |
| **B**ackground |  |
| **A**ssessment |  |
| **R**ecommendation |  |

# Session 3\_Declan’s case

|  |  |
| --- | --- |
| Declan has been brought into the department by police after being found wandering around a local housing estate shouting at strangers. He accused passers-by of trying to control him.Declan required rapid tranquilisation and now that he is calmer a secondary assessment has been completed and further details are given below:Declan appears slightly drowsy and sedated, he is complaining of a headache and despite the sedation is still quite irritable. . Declan is orientated to person and place but is unsure of the time. He doesn’t smell of alcohol and denies any recent alcohol use. He states that he occasionally uses cannabis and ‘a bit of this and that’ unclear what he has used recently. Declan is not complaining of any physical health problems other than a headache but does not wish to give any further details about his physical health or any past mental health problems. Declan states that there is a local conspiracy against him, he believes that locals have been spying on him and trying to control his mind and behaviour, people are commenting on what he is doing and communicating with him through the TV. Today he finally had enough and went out to try and confront the people he thought were doing this to him. He did not have a weapon and after confronting people he had no further plan. | O:\02_Courses\01_Specific\APEX\11_Development\1st edition\03_Teaching materials\Images\Declan.jpg |
| **Secondary assessment:** |
| Focused physical history and secondary examination plus investigations |  |
| Secondary mental health assessment |  |

# Session 3\_Differential diagnosis

Insidious onset

**Acute Confusional State and Dementia -** See Chapter 7.

**Acromegaly** – depression, irritability, apathy or labile mood.

**Cushing’s syndrome** – depression, mania and psychosis.

**Addison’s disease –** Depression, anxiety and irritability.

**Hyperprolactinaemia –** Severe depression.

**Hypopituitarism –** Depression often with irritability, features of dementia.

**Hyperparathyroidism –**depression

**Hypoparathyroidism –** may rarely present with psychoses

**Huntington’s chorea** – may present with depression, aggression, psychosis, obsessive compulsive disorder.

**Wilson’s disease** – personality change, mood disturbance, psychosis and cognitive impairment.

**Cerebral Tumours** – depression, emotional lability. Symptoms may depend on the location of the tumour - Occipital may cause visual hallucinations, temporal lobe may cause visual and auditory hallucinations, parietal tumours are associated with tactile and kinaesthetic hallucinations. Frontal tumours may cause disinhibition and a variety of hallucinations. Diencephalic tumours may present as Korsakoff’s.

**Head Injury –** organic schizophrenia and affective psychoses. Personality change.

**Neuroacanthocytosis** – depression, anxiety, OCD and personality change.

**Spinocerebellar ataxia** – personality change, labile mood, aggression, dysexecutive syndrome

**Multiple Sclerosis** – depression, mania and psychosis

**Systemic Lupus Erythematosus** – psychotic symptoms

**Inherited leucodystrophies** (metachromatic leucodystrophy) – Psychotic symptoms

**Variant Creutzfeldt-Jakob disease** – depression or anxiety

**Sporadic Creutzfeldt-Jakob disease** – rapidly progressive dementia.

**Charles Bonnet Syndrome** – ocular pathology is related to visual hallucinations which are often complex and vivid and tend to occur at times of ow light. The patient often has full insight and is undistressed.

**Peduncular hallucinosis** – damage to the midbrain/thalamus causes vivid visual hallucinations often in the evenings.

**Parkinson’s disease** – depression

**Hypothyroidism** – severe depression with or without psychotic symptoms, may present as early dementia.

**Hyperthyroidism** – hypomania/mania, psychosis and anxiety

**HIV –** dementia

**Syphilis –** may present in a variety of ways including dementia, depression, and elation or with schizophrenic features.

**Cerebrovascular Disorders** – delirium, organic psychoses, mood disorders and personality change.

**Polyarteritis nodosa -** delirium, mania and paranoia

**Pellagra –** delirium, depression and psychoses.

**Phaeochromocytoma** – chronic anxiety and panic disorder.

**Paraneoplastic syndrome** – psychoses and affective symptoms.

**Motor Neurone Disease –** emotional lability

Acute onset

**Acute Intermittent Porphyria** – psychotic symptoms

**Encephalitis** including anti-NMDA receptor and anti-voltage gated potassium channel antibody associated limbic encephalitis – psychotic symptoms, delirium.

**Head injury** – may cause aggressive behaviour, agitation, amnesia and confusional states.

**Epilepsy**

* Temporal Lobe Epilepsy – hallucinations in all modalities, may appear to have thought blocking, deja vu and jamais vu, affective symptoms.
* Per-ictal, Inter-ictal, post ictal psychosis
* Complex partial seizure – may lead to epileptic automatism (the patient may perform often simple but at times complex activities while suffering from reduced consciousness).
* Fugue – patients may appear drowsy or intoxicated often undertake complex behaviours and wander large distances waking in an unknown area with no idea how they arrived there. These states may last from hours to rarely weeks at a time.
* Parietal and occipital lesions may cause visual phenomena.
* Transient Global Amnesia

**Migraine –** depression, anxiety, irritability and complex auditory and visual hallucinations.

**Carbon Monoxide Poisoning –** may present as Korsakoff’s.

**Hepatic encephalopathy –** delirium

Strange behaviour …

… Associated with Drug and Alcohol Misuse

Alcohol Intoxication

The symptoms vary according to the level of intoxication, in moderate intoxication symptoms include unstable gait, slurred speech, and disinhibited behaviour including aggressive behaviour.

Alcohol Related

* **Delirium Tremens –** occurs in withdrawal from alcohol in patients who have been alcohol dependent for some time and is a medical emergency. It presents with clouding of consciousness including disorientation, recent memory impairment, tremulous hands, hyperhidrosis, severe agitation and insomnia. Patients describe or can be seen interacting with vivid and at times bizarre hallucinations. Often hallucinations are visual or somatic in nature, patients may report seeing or feeling spiders, snakes or even small figures (Lilliputian hallucinations) running around. Patients may also have occupational hallucinations and undertake actions that they would in their day to day work.There may also be paranoid delusions

Physically the patients display autonomic dysfunction and dehydration with electrolyte disturbance especially low potassium and magnesium levels. Symptoms as with delirium tend to worsen at night, Delirium Tremens typically develops 24 hours after the last drink and lasts for up to six days.

* **Wernicke’s encephalopathy –** is due to thiamine deficiency and occurs most commonly in alcoholics but may also occur in other patients with severe nutritional deficiencies. There is an acute onset of acute confusional state, opthalmoplegia, nystagmus and ataxia. This is a medical emergency and without treatment has a mortality rate of ~15% and 84% will go on to develop Korsakoff psychosis.
* **Korsakoff Psychosis –** is predominantly an inability to lay down new memories with co-existing retrograde amnesia. Patients confabulate (invent memories to cover the periods of amnesia, the patient believes these memories to be true) for the episodes of amnesia.
* **Alcoholic Dementia and cerebral atrophy**

Psychiatric effects of commonly prescribed medication

A variety of medication may give rise to psychotic symptoms and in acute onset be aware that this may be the case and take into consideration.

Illicit substance use

Often intoxication with illicit substances leads to the development of psychiatric symptoms particularly psychotic symptoms. Some of the more commonly encountered substances and effects are listed below, however this is not an exhaustive list and there are frequently newly manufactured drugs and so called ‘legal highs’. Many of the newer drugs elicit both mood lability, impulsivity and psychotic symptoms refer to Toxbase for further details*.*

**Diazepam –** generally sedative effects but there may be paradoxical excitement and aggression

**Opiates, Buprenorphine, methadone –** sedative effects, depression. Psychotic symptoms have been reported with Buprenorphine and methadone.

**Cannabis –** both acute intoxication andchronic use may be associated with paranoia, psychotic symptoms and mood fluctuations.

**Amphetamines (inc methyl amphetamine) –** may present as mania or psychosis. Psychotic symptoms often include delusions of insects being present under the skin with associated tactile hallucinations. Amphetamine related psychosis can be expected to have significantly improved within a week of cessation of amphetamine use.

**Cocaine/Crack Cocaine –** Initially euphoria followed by a period of dysphoria leading to hallucinosis and psychosis. Similar to amphetamine psychosis there may be delusions of insects under the skin and associated tactile hallucinations. The psychotic symptoms generally resolve within 24-48 hours.

**Ketamine –** derealisation and depersonalisation, paranoia and psychotic symptoms.

**Lysergic Acid Diethylamide (LSD) –** labile mood, synaesthesia, vivid hallucinations and illusions which tend to be visual. Altered sense of time and space may be accompanied by distortions of body image. The effects are short lived lasting up to 16 hours however LSD induced psychosis may last several weeks and in some reports years.

**Magic Mushrooms (psylocybin, psilocin) –** predominantly hallucinations.

**Phenyclidine (PCP) –** psychotic symptoms and can be associated with violent behaviour.

**Ecstasy –** generallyfeelings of euphoria however psychotic symptoms have been reported with heavy use.

**Mephedrone –** depression with suicidal thoughts and psychosis.

**Solvent Abuse –** euphoria, disinhibition and hallucinations

**Anabolic Steroids –** Mood instability, aggression, depression and paranoia.

**Khat –** elation and psychotic symptoms when heavily used.

Common psychiatric presentations

* Schizophrenia
* Drug Induced Psychosis, Psychotic Episode, Delusional Disorders
* Catatonia
* Bipolar Affective disorder
* Depression
* Intoxication and Withdrawal
* Delirium and Dementia
* Anxiety
* Dissociative disorders and PTSD
* Personality disorder
* Munchausen’s Syndrome, Munchausen’s by proxy, Malingering