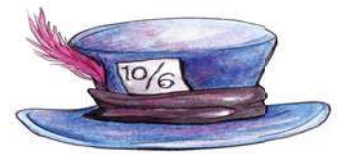


The Ultimate Alphabet GP Guide to Unusual Psychiatric Disorders



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1. Alcoholic Hallucinosis

Alcoholic Hallucinosis can occur in patients with chronic alcohol abuse; both during heavy alcohol consumption and on stopping alcohol suddenly. The ICD-10 classifies alcoholic hallucinosis as a psychotic disorder due to the use of alcohol^{1, 2} of rapid onset intense hallucinations, predominantly auditory in nature, which cannot be solely explained by acute intoxication or by delirium tremens. Patients may hear voices or music. Delusion and mood change can occur but this is not the prominent feature. Patients with alcoholic hallucinosis usually have insight into the condition and fluctuation in consciousness does not usually occur.³



It was first named in 1919 by the famous Swiss psychiatrist Eugen Bleuler,⁴ following descriptions of cases from as early as 1847, where a syndrome named 'folie d'ivrogne', meaning 'drunken madness' was noted by the French author C.N.S Marcel.⁵ Since the discovery, relatively little research has been conducted into alcoholic hallucinosis and the evidence base is recognised as poor.^{6, 7} This disorder occurs worldwide; with incidences of 0.4-12.36% in alcohol dependent patients, depending on the study quoted,⁸⁻¹⁰ although it is thought that true incidence is probably around 0.4%, due to issues with the design of the studies quoting higher values.⁷ The aetiology of alcoholic hallucinosis is unknown; evidence currently available has found no conclusive evidence of a genetic link to schizophrenia; and imaging studies have been unable to identify a cause for this disorder.⁷ One study discovered increased levels of the excitatory neurotransmitters glutamate and aspartate, and reduced levels of inhibitory GABA and glycine in these patients.¹¹ Several studies have examined the efficacy of different antipsychotics in managing alcoholic hallucinosis but conclusive results have yet to be drawn.

Currently, abstinence from alcohol and antipsychotic medication is the mainstay of treatment but more extensive evidence needs to be carried out in this area.⁷ One Indian study quoted that in a study of 52 patients with alcoholic hallucinosis, 13.5% continued to hallucinate even though patients had remained abstinent, and 21% of patients did not have relapse, even though they continued to consume alcohol.² This reinforces the view that further study of alcoholic hallucinosis is required to be able to understand and manage this disorder effectively.

Contributed by George Brahman

2. Alice in Wonderland Syndrome

"I can't explain myself sir, because I'm not myself you see"¹ - Lewis Carroll, Alice in Wonderland.¹



Alice in Wonderland Syndrome describes a rare neurological disorder, first described in 1955 as a perceptual disorder characterized by distortions of visual perception

(metamorphopsias), the body schema, and the experience of time.² Dr John Todd, a British psychiatrist, gave the disorder its name, noting that the misperceptions resemble Lewis Carroll's descriptions of what happened to Alice.²

The main symptom is of altered body image; there is a distortion in the sufferers' perception of the size of various objects, much like Alice.^{2, 4} During an episode, some objects appear to get smaller (micropsia), making the sufferer feel larger, whilst for others, objects get larger (macropsia) making the sufferer feel smaller.⁴ Objects can also get further away or get closer.

Occurring in children more than adults, symptoms usually occur at night. The hallmark feature of this syndrome is a migraine, which patients usually experience prior to the abnormal perceptions. Looking at the causes of Alice in Wonderland syndrome, there are several theories. It is thought to be caused by a change in the way the brain processes perceptions of the environment, other causes include temporal lobe epilepsy, psychoactive drugs, brain tumours and Epstein Barr Virus encephalitis.² Patients are referred for investigation (bloods, EEG, MRI brain) but a large number will have normal investigations.

Alice in Wonderland syndrome is a distortion of perception,⁴ not an optical problem, a hallucination nor a physiological change to the body's systems. The diagnosis can be presumed if all other causes of neurological disease have been ruled out and there is an association between symptoms and the onset of migraines.⁴

There is no known treatment of this condition, with patient reassurance being key. It is important to emphasise that these perceptions are not harmful and do not require treatment; the perceptions usually disappear with time.^{2, 5} Abnormal perceptions can recur several times a day, and may only become less frequent as a child increases in age, and can be frightening and alarming for patients to experience.⁵

Contributed by Nasreen Desai

3. Anti-NMDA receptor encephalitis



Anti-N-methyl-D-aspartate receptor (anti-NMDAR) encephalitis consists of acute psychiatric symptoms including hallucinations, delusions, agitation and aggression or catatonia. There may also be speech dysfunction, seizures, memory deficits, dyskinesias, autonomic instability, central hypoventilation, and reduced levels of consciousness.^{1, 2} These receptors are ion channels found in the nervous system and involve the chemicals glycine and glutamate. They have been implicated in many neurological diseases but in anti-NMDA receptor encephalitis there is an autoimmune reaction resulting in severe encephalitis with mental deterioration of the patient. The first description was in 2005 in four women with ovarian teratomas and was originally named 'Teratoma-associated encephalitis'.³ The antibodies against the NMDAR, which are responsible for the condition, were isolated in 2007 following more reported cases and the syndrome was named by Josep Dalmau and colleagues.⁴

One large study identifying it as is responsible for 4%

of confirmed cases of encephalitis in the UK⁵, and another showing that in 1% of all ITU admissions with encephalitis, NMDAR antibodies were discovered. Almost all of these patients present with psychiatric symptoms within the first month.² A multi-institutional cohort study⁶ revealed that anti-NMDAR encephalitis is more common in women (81%) and is prevalent across all age groups but predominantly in patients under age 45 (95%) with a median age of 21 years. Tumours were identified in 38% of the patients and 94% of these tumours were ovarian teratomas, with the majority occurring between the ages of 12-45. As expected therefore there is a female increased incidence of 4: 1 females to males. The remaining tumours identified included lung, breast, testicular, ovarian carcinoma, thymic carcinoma, and pancreatic cancer with no more than two cases of either. This study and others⁷ identified that although there is an association between tumour presence and the syndrome; in the majority a tumour is not identified and no cause is found for the presence of antibodies against the NMDAR.

Definitive diagnosis is by detection of NMDAR antibodies, which should be present in cerebrospinal fluid.⁹ If present, these antibodies are very specific for anti-NMDAR encephalitis.^{10, 11} The characteristic course of the illness¹² emphasises the rapid mental deterioration and complications. Treatment involves immunotherapy first-line and removal of any tumours present, with second-line immunotherapy if there is a poor response. With regards to prognosis within 24 months: the mortality rate is 6%, probability of making a good recovery is 81% and the relapse rate is approximately 12%. Like any encephalitis some patients are left with intellectual or other mental long term sequelae.

Contributed by George Brahman

4. Binswanger Disease

Described by Otto Binswanger in 1894, Binswanger's disease (BD), is a type of dementia caused by widespread, microscopic areas of damage to the deep layers of white matter in the brain.¹ The damage is caused by atherosclerosis, thromboembolism and other diseases that obstruct blood vessels that supply the deep structures of the brain² leading to disruption of the subneural cortical circuits controlling executive cognitive functioning.¹ This results in symptoms of deterioration in memory, organisation, mood, attention, decision making and behaviour¹. Binswanger Disease usually affects males and females equally and presents late in the fourth decade and severity increases with age.

The most characteristic feature of Binswanger Disease is psychomotor slowness - an increase in the length of time it takes, for example, for the fingers to turn the thought of a letter into the shape of a letter on a piece of paper.¹ Other symptoms include forgetfulness, speech changes, changes in personality or mood (apathy, irritability, and depression), and urinary symptoms in the absence of urological disease.¹ CT or MRI scan reveals Binswanger Disease damaged brain tissue and is essential for a positive diagnosis.¹

Risk factors for Binswanger's disease include³

- Hypertension
- Smoking
- Hypercholesterolemia
- Coronary artery disease



- Diabetes mellitus

There is no known treatment for Binswanger Disease, and as such the long term outlook is poor. Due to the vascular aetiology, the symptoms and signs may suddenly worsen due to small strokes, stabilise and then improve for a short time, but the overall condition continues to advance as the blood vessels become increasingly obstructed.¹ The aim of treatment is to reduce the rate of progression of symptoms. This is done by control of diet and increasing exercise, using anti-hypertensives to control blood pressure, statins for atherosclerosis, anti-platelet agents to reduce the risk of thromboembolism and optimising diabetes control.² Additional medications can be given to control individual symptoms such as depression.

Contributed by Nasreen Desai

5. Bouffée délirante

Bouffée délirante is a well-known diagnosis in French psychiatry, described in the 1880s by the French psychiatrist Valentin Magnan,¹ although information on this condition outside of France is minimal. It literally translates as “delusional flush”.² It is a cultural disorder, seen mainly in younger males in West Africa and Haiti. The condition is an acute remitting psychosis, described as sudden, short-lived outbursts of aggression and agitation³ with accompanying “clouding of consciousness” in connection with emotional instability.

Delusions with ever-changing themes are usually present and hallucinations may be co-existent. An episode may be triggered by a period of stress, although this is not essential for diagnosis. Patients often do not have any previous psychiatric history. Bouffée délirante influenced the ICD-10 label of “acute and transient psychotic disorders”, which are acute-onset psychotic disorders which resolve in less than 3 months. This condition generally has a good prognosis, as after an episode the patient will quickly return to their pre-morbid state, although relapses can occur after symptom-free periods.¹

Contributed by Sharna Bennett

6. Briquet's syndrome

Briquet's syndrome, named after the 19th century physician, Pierre Briquet, is now more commonly known as somatisation disorder.¹ In the ICD-10 diagnostic criteria, it is described as a fluctuating and chronic disorder with a long history, involving frequent contact with primary and secondary care. Patients have several physical symptoms, for at least two years, which are recurrent and often changing in nature and can be related to any part of the body. Somatisation disorder can have a severe impact on the patient's social functioning.² Symptoms are widespread and pain may be felt in any area, but common locations are the chest, abdomen and joints. Gastrointestinal symptoms are usually present; these can include bloating, nausea and diarrhoea. Neurological symptoms such as difficulty swallowing and sexual symptoms, for example heavy menstrual bleeding are also common.

Somatisation disorder is more common in females, with a prevalence of 0.2-2%, in males it is likely to be less than 0.2%.¹ The DSM-5 classification differs to ICD-10 as it



has now placed somatisation disorder under the umbrella term of “somatic symptom disorder”, which includes other diagnoses such as hypochondriasis and pain disorder.³

Contributed by Sharna Bennett

7. Capgras Delusion

Capgras is a delusion or of misidentification, therefore a belief that someone emotionally close to them such as a partner or child is an identical looking imposter. First described in the original case as an ‘illusion of look-alikes’ by Joseph Capgras and Reboul-Lachax in 1923. The delusion is characteristically associated with underlying psychiatric disorders such as paranoid schizophrenia, however it has also been seen in neurodegenerative disorders such as Alzheimer’s disease and Lewy body dementia.¹



There has also been identification of the delusion in association with more systemic organic and metabolic aetiologies; notable examples include cases associated with diabetes mellitus and hypothyroidism. Hence a thorough investigation of the presenting patient is important to exclude secondary causes.^{2, 3} It is important to note that the delusion is not simply misidentification, but the patient recognises that the imposter has the identical physical appearance of the true person e.g. a wife.¹

Contributed by Lucy Button

8. Charles Bonnet Syndrome

Charles Bonnet Syndrome (CBS) was first described in 1769 when he described hallucinations in his bright elderly father after cataract surgery. It is a condition characterised by the presence of complex visual hallucinations in patients suffering from acquired visual impairment¹ and occurs in up to 50% of people with age related macular degeneration (ARMD). Hallucinations typically develop hours to days after the loss of vision and may be of an insidious or sudden onset¹. Patients typically maintain intellectual functioning and full or partial insight into the fact that the hallucinations are not real.²



The hallucinations are typically pleasant, although they may sometimes cause the patient distress.¹ The hallucinations are said to be extremely vivid and patients may experience a sharp contrast between the clarity of hallucinations and reality. Although there are no stereotyped hallucinations specific to CBS, the most common hallucinations are those of faces, animals and geometric shapes. Interestingly, patients may experience visions of themselves, sometimes at earlier stages in their lives, a phenomenon known as *heautoscopia* or *autoscopia*.¹

There is often no identifiable trigger and hallucinations can last for anywhere from seconds to hours. The exact pathophysiological mechanism surrounding CBS is not currently known but there are a number of hypotheses which include sensory deprivation (e.g. “phantom vision” comparable to phantom limb pain) or perceptual release (i.e. the release of subconscious perceptions into the consciousness secondary to reduced afferent sensory input due to vision loss).¹

Contributed by Lucy Button

9. Cotard Delusion/ Syndrome

NIHILISM

Cotard delusion has become the eponym for a nihilistic delusion.¹ The word ‘nihil’ is Latin for ‘nothing’, therefore a nihilistic delusion is a belief in nothingness; either in the context of the patient’s own existence or the future or the world. A Cotard delusion is characterised by a patient’s belief that the self, part of the self or their body, such as their internal organs, are diseased, deceased or are non-existent. Within this description, there is an element of delusional thinking that is hypochondriacal or focuses on immortality.

The delusion has been described as a delirium of negation and is associated with self-loathing and severe depression². Jules Cotard in 1880 described this condition the ‘*délire des négations*’ as a new form of depression with associated anxiety and an agitated melancholia.³ However, this concept was later disputed and it was suggested that Cotard had observed these symptoms as part of a syndrome consisting of nihilistic, or hypochondriacal delusions with affective symptoms. Cotard’s syndrome is therefore the combination of this delusion with varying degrees of depression and it has been identified in patients with neurological and more usually with psychiatric disorders.^{1,2,4}

Cotard delusion may manifest or present itself in a patient with a pre-existing mental or neurological health disorder with significant affective symptoms and possibly suicidal ideation with an explanation of their non-existence.⁵ In some cases, the patient describes that certain body parts or their internal organs are diseased or no longer exist.¹

Contributed by Ben Rodgers

10. Couvade Syndrome

Couvade syndrome originates from the Breton/French verb “*couver*” which means “to brood or incubate”¹ and is applied to rituals practiced by many races and cultures around the world from as early as 60BC whereby expectant fathers are expected to adhere to strict diet and sexual restrictions or even mimic labour (some of whom have claimed to have felt contractions) whilst their partners are pregnant.¹



In Western culture Couvade syndrome, rather than Couvade ritual, describes the wide range of psychosomatic symptoms, usually these are weight gain, loss of appetite, nausea and vomiting and toothache.^{1, 2} Typically, the symptoms experienced resolve after the birth of the child.² Couvade Syndrome is more common in patients with a previous psychiatric diagnosis of depression or anxiety² and appears to be an expression of anxiety and occasionally parturition envy.¹

Contributed by Ben Rodgers

11. de Clerambaults syndrome

Known also as Erotomania, de Clerambaults syndrome is a recognised delusional disorder which describes the false belief which belongs to a deluded individual that a person who is socially and financially attractive, is madly in love with them¹. The syndrome is classically described in women but cases of affected males and even an equal prevalence has been reported.^{1,2} The subjects of



the delusional beliefs are often those with apparent wealth, authority and high social standing. All these attributes are romantically desirable to the delusional person. The delusional individual on the other hand is often a relatively isolated, introverted person, which may encourage the delusions to flourish and grow².

A number of triggers include high emotions, hormonal disturbance (for example around pregnancy), alcohol and certain medications have been described. However a strong family history of psychiatric disorders has been reported.¹ ² As time progresses, the delusional person sees signs and messages from their lover which only reaffirms their false beliefs. A song on the radio, a headline in a newspaper and even an ambiguous facial expression can all contain new meaning for the deluded person and demonstrate the mistaken individual's perpetual love.²

Contributed by George Reid

12. Delirium Tremens

Delirium Tremens (DT) is an uncommon but severe complication of acute alcohol withdrawal occurring in approximately 5% of withdrawals.^{1, 2} ICD-10 defines the syndrome of alcohol withdrawal as any three of the following: Tremor, sweating, nausea/vomiting, tachycardia, psychomotor retardation, headache, insomnia, malaise/weakness, transient hallucinations, grand mal seizures.³ DT is a medical emergency that can occur anywhere between 1-8 days after the patient's last ingestion of alcohol, but there is usually a peak around 48hrs.⁴ Alongside the symptoms of an uncomplicated withdrawal syndrome, DT presents with an acute global confusional state (hence 'delirium') with many psychological symptoms (clouded consciousness, fluctuating disorientation, delusions/illusions/hallucinations-often auditory, tactile or 'liputian').⁵ Loss of insight and amnesia are common and differentiates DT from alcoholic hallucinosis.^{1, 5} There are also somatic symptoms of DT such as marked tachycardia, hyperthermia and cardiac arrhythmias which may prove fatal!

The first descriptions of DT hail back to Hippocrates, in which he described a man by the name of Chaerion; who was grasped by fever, rigors and incomprehensible speech on day four of abstinence after a 'drinking-bout'.⁶ It took Chaerion 20 days to become completely symptom free. The pathophysiology of DT is thought to be due to the chronic effects of ethanol on the CNS i.e.: Increased inhibitory effect (via GABA-receptor stimulation), decreased stimulatory effect (via Glutamate-receptor inhibition). As a tolerance develops the patient requires more alcohol to achieve the same euphoric effects that a non-dependent person would otherwise experience. An abrupt abstinence from this 'tolerance state' reveals that the previously 'inhibited' Glutamate-receptors have been upregulated and GABA-receptors downregulated, therefore the excitatory tone of the patient is now increased.^{4, 5} It's somewhat unsurprising that risk factors for DT relate to a prolonged history of drinking, age, previous history of DT.⁴ The rationale behind treatment is to allow the discordance between the regulation of GABA and Glutamate receptors to resolve themselves slowly over time. Benzodiazepines such as oral Lorazepam or parenteral lorazepam/Haloperidol (which act on GABA-receptors) play a role by allowing a slow alcohol



detoxification without any acute side effects.^{5, 7} Other forms of treatment are Thiamine (vitamin B1) & glucose, propranolol for nutritional and somatic symptomatic relief respectively.⁵

Contributed by Sean Testrow

13. Dhat Syndrome

Dhat syndrome describes the preoccupation and anxiety associated with perceived excess semen loss. It is a culture-bound syndrome, almost exclusively described by young males who are often single and come from a rural, uneducated upbringing.¹ It is largely found in the Indian subcontinent, with cases commonly found in India and Sri Lanka.²



The cause of the semen loss has been attributed to a variety of different causes, some biological such as loss through urination, urinary tract infections and masturbation. Other causes are more psychosocial in nature, including monetary concerns, poor company and disturbed sleep.¹ In cultures where Dhat syndrome is prevalent, semen, alongside blood and bone marrow is described as 'Dhatu', which approximately translates to 'a fluid which is of fundamental importance to body'.

Imbalance in the 'Dhatu' is believed to be detrimental to the person's health, leading to vague generalised symptoms associated with Dhat syndrome.^{1, 2} Tiredness, anxiety, depression, weakness and sexual dysfunction are regularly reported and some sufferers even believe Dhat syndrome will lead to a reduced life expectancy and cause teratogenic effects in their unborn children.²

Contributed by George Reid

14. Diogenes syndrome

Diogenes, 4th century Greek philosopher, whose beliefs were: 'life according to nature', 'self-sufficiency', 'freedom from emotion', 'lack of shame', 'outspokenness', and 'contempt for social organisation' is the reference for the nomenclature of Diogenes syndrome.^{1, 2} Diogenes syndrome - is a reference to the isolation and rejection of the outside world and its material possessions practised by the philosopher, Diogenes. However, the syndrome differs from the philosopher as it incorporates refusal or rejection of help from the world as its foundation; rather than rejecting the world and its material possessions like the philosopher.¹



This syndrome creates irrational relationships with

1. Society - refusal or rejection of help
2. Body - extreme self-neglect
3. Objects - hoarding (sylligomania).

These unreasoned relationships eventually lead to social isolation, physical illness and hoarding an excessive amount of possessions which are valueless to others.^{1, 2, 3}

The main 'symptom' is the refusal or rejection of help. This makes it distinct from hoarding which is often used interchangeably with Diogenes.

It is associated with social isolation, not wanting/seeking help or refusing help, psychiatric illnesses like chronic psychotic illness, OCD, alcohol abuse, dementia with

frontotemporal and frontal lobe dysfunction. There may be varied pre-morbid personality traits; being unfriendly, aloof, stubborn, aggressive, compulsivity, paranoia etc.^{1,3} Over the years, Diogenes syndrome has been referred to as ‘senile breakdown’, ‘social breakdown’, or ‘senile squalor syndrome’.^{1,2} However, the much-debated terminology Diogenes is still used more frequently over others.

Contributed by Shanju Rai

15. Ekbom Syndrome

Ekbom Syndrome, named after the Swedish neurologist Karl-Axel Ekbom,¹ is also known as Delusional parasitosis or delusional infestation.² It commonly affects middle aged females and people who have cognitive, psychiatric disorders.¹ Some neuroimaging studies have identified that the putamen maybe involved.³



It is a psychiatric disorder where the person has a fixed false belief that he/she is infested with small living organisms in the skin such as bugs, parasites or insects.¹⁻⁴ The person has tactile hallucinations and can also have visual hallucinations of the organisms.^{2,4} Differentials might be:

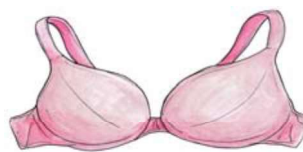
- a) actual parasitosis where the person is infested with an organism e.g. scabies or demodex mites etc. and can be proven with lab tests
- b) insect phobias where the patient has an irrational fear of insect bites or infestation occurring; the patient with Ekbom’s believes that they are already infested.¹

It’s a delusion because the belief that they are infested with these organisms is unshakeable and cannot be altered, even with evidence². Due to this patients often seek help from various specialties such as physicians, entomologists, dermatologists, psychiatrists etc.² The delusional nature of this syndrome requires neuroleptic medications, such as those used in schizophrenia.²

Contributed by Shanju Rai

16. Fetishism

Fetishism is an example one of the 8 paraphilia’s listed in the DSM-5. Paraphilia is defined as abnormal sexual desire. In order for a diagnosis to be given the patient must exhibit recurrent, intense sexually arousing fantasies, sexual urges and behaviours which involve either the use of non-living objects and/or a non-genital body part for at least a period of 6 months.¹ Sexual interests in fetishist can lead them to perform criminal activities. An example of this might be when an underwear fetishist breaks into a female hall of residence to steal underwear.²



Contributed by Naa Quayee

17. Fregoli delusion or the delusion of doubles

Fregoli syndrome is a subtype of delusional misidentification.¹ In this condition, a person believes that a familiar person, usually someone he



believes to be his persecutor, is impersonating multiple familiar people.¹ It is usually associated with schizophrenia.²

Fregoli syndrome is named after a famous Italian actor, Leopold Fregoli, who had a remarkable skill in changing appearance during his stage act.³ In 1927 a report written by P. Courbon and G. Fail first reported the syndrome.⁴ According to the report, a young woman had a strong belief that two actresses whom she often went to see at the theatre were persecuting her. The woman believed that these two actresses were disguising themselves as people she knew.

The causes of Fregoli delusion include traumatic brain injury, lesions on the brain and long term treatment with levodopa.³ The syndrome can be treated with antipsychotics, anticonvulsants or antidepressants.³

Contributed by Ying Yeo

18. Frotteurism

Frotteurism is described as recurrent, intense sexually arousing fantasies, sexual urges or behaviours which involve touching and rubbing against a non-consenting person and in which the condition lasts over a period of at least 6 months.¹ This type of paraphilia tends to occur in crowded public places such as on buses or trains and it is sometimes difficult to identify the perpetrator because of the crowded circumstances.²



Contributed by Naa Quayee

19. Fugue State

Fugue state is a rare dissociative disorder.¹ The fugue is a condition in which involves a sudden loss of all autobiographical memories and knowledge of personal identity.² This is associated with unexpected, purposeful wanders away from home or the person’s place of work.²



In order to diagnose a person with fugue, the following criteria should be fulfilled:³

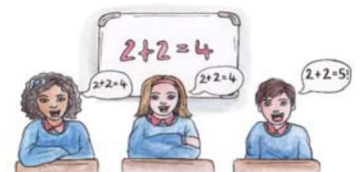
- a) Features of dissociative amnesia
- b) Purposeful travel beyond the usual everyday range
- c) Maintenance of basic self-care and simple social interaction with strangers

The fugue state may last from hours to months with a subsequent amnesia gap on recovery and usually precipitated by stressful events.^{1,4}

Contributed by Ying Yeo

20. Ganser’s Syndrome

Ganser’s syndrome, also known as “prison psychosis”, was first described by a German psychiatrist, Sigbert Ganser, in 1898 whilst working inside a prison.¹ He discovered three inmates, who when asked simple questions, would give approximate answers in response. He studied their behaviour and discovered the



syndrome was characterised by four aspects:¹

1. Giving approximate answers to questions
2. Visual/auditory hallucinations
3. Developing somatic symptoms
4. “Clouding of consciousness” which often fluctuated.

The most striking feature of this syndrome is giving approximate answers to questions. Patients may give answers which are nearly, but not quite correct e.g. saying there are five legs on a horse or recalling the month as September instead of August. They can comprehend the question but they give incorrect responses. An example was a South African man¹ who when asked to recall the words “honesty, window and lace”, gave the response of “modesty, house and shoe”.

The aetiology behind Ganser’s syndrome was initially suggested to be a form of malingering¹ in prisoners. This has evolved over time and is now classified as a dissociative disorder in ICD-10.³ It is thought to be due to a severe stress reaction rather than a factitious disorder, with some suggesting it there could be underlying brain injury.² Due to its rarity, there is little literature on the syndrome, with Ganser himself stating “I must openly admit that after observing many details there is a great deal that is obscure to me”.¹

Contributed by Chloe Desmond

21. Huntington’s Chorea

From the Ancient Greek word “chorea” meaning dance, chorea is described as “involuntary, purposeless, and rapid distal movements of the limbs”¹ and is a characteristic feature of Huntington’s disease.² Chorea was first accurately described by Thomas Sydenham, a British physician in the 17th Century.¹ However, it’s history reaches far back into the Middle Ages, when it was initially called “dancing mania”, at a similar time to the appearance of the black death.¹

It has also been described as “Saint Vitus’s dance”, due to reports that patients with the condition were supposedly cured following touching Saint Vitus’s relics.

From 1850, chorea was mostly described in association with rheumatic heart disease.¹ It was not until 1872 that George Huntington first described Huntington’s chorea.³ At this period, the full extent of the disease was unknown, only the most prominent motor feature. In the 1980’s more non-motor symptoms of the disease were established. Therefore, the name was changed to Huntington’s disease to encompass the wide variety of clinical features.

Huntington’s disease is a progressive neurodegenerative disorder which follows an autosomal dominant inheritance pattern. A CAG trinucleotide repeat in a protein called Huntingtin drives the disease, which causes chorea, cognitive decline, dystonia² and behavioural disturbances.³ Mean onset is usually around 30-50 years of age and there is currently no cure.³ However, Huntington’s chorea can be treated using dopamine antagonist agents, with tetrabenazine found to make the most significant improvement.³

Contributed by Chloe Desmond

22. Jerusalem Syndrome

Jerusalem syndrome is a behavioural phenomenon, by which visitors to the city of Jerusalem, suffer psychotic decompensation.¹ The main symptom of this syndrome is religious delusions in which the patient identifies with a biblical character, and their behaviour subsequently mimics that of the selected character.¹



There are three distinct classifications of the illness:

- Type 1: Patients who are already diagnosed with a mental disorder characterised by psychotic illness, which has influenced their decision to visit Jerusalem, based on their pre-existing mental illness. This may involve delusions of a higher purpose to their visit.^{1,2}
- Type 2: Patients with a mental disorder that is characterised by idiosyncratic ideation, that is not categorised as psychotic or delusional.¹ The patient experiences strange thoughts related to politics or religion in Jerusalem.¹
- Type 3: Occasionally described as ‘pure’ form of Jerusalem Syndrome, this describes patients who suffer a acute psychotic reaction while in Jerusalem, and who have no previous mental illness.¹ Recovery is rapid and uncomplicated on leaving, and the patient resumes normal state of mind.¹

In Israel there is a dedicated facility, Kfar Shaul Mental Health Centre, who deal with these cases of tourists suffering from acute psychosis, triggered by and related to Jerusalem.³ On average 100 tourists are seen annually at the facility, with 50 being admitted for treatment.^{1,3}

Contributed by Sasha Wilson

23. Kleptomania

Kleptomania is the repetitive impulse to steal items as a means of relieving intrusive thoughts to steal.¹ To distinguish this from ordinary theft, in kleptomania there is no monetary incentive to steal and the items that have been stolen are usually of trivial monetary value, and serve no personal use to the person e.g. only stealing one shoe.²



Kleptomania is generally accepted to be categorised as an impulse control disorder.¹ It is thought to have strong links with other psychiatric disorders such as drug and alcohol abuse, eating disorders and other impulse control disorders such as trichotillomania (the impulse to pull out hair).³

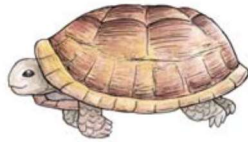
The stereotypical patient is female, although males can be affected also.⁴ Kleptomania is thought to begin in adolescence, usually in the form of shop-lifting.⁴ One of the most common reasons for patients to seek help is due to the legal consequences of kleptomania, if apprehended by law enforcement; this usually occurs in middle age.⁴

Historically, the mainstay of medical treatment has been using SSRI’s. However there is emerging evidence that anti-epileptics and lithium may have a future role to play in the treatment of kleptomania.¹

Contributed by Sasha Wilson

24. Koro

Koro is a culture-bound syndrome illustrated by the belief that the genitalia are retracted into the abdomen, in the absence of any true pathology of the genitals, or breasts into the chest. The name could be derived from a river and local tribe which resided in Sulawesi, Indonesia, hence the first Western reference of the term in B.F. Matthes' *Dictionary of Buginese Language* (1874) of South Sulawesi, Indonesia.¹ In Makassarese language koro means "to shrink". The fundamental symptoms of the syndrome are:



- delusional belief of retraction of the penis into the abdomen
- signs of anxiety and associated intense panic
- practice of mechanical means to avert penile retraction.²
- associated belief of death following the disappearance of the organ has also been described.
- However, a belief in breast shrinkage is also reported

The highest prevalence has been in South East Asian countries, often appearing in the form of epidemics, such as in Singapore³ and North Bengal.⁴ In an outbreak of Koro on a tea plantation in West Bengal, India, females complained of retraction of their breasts, with fear of impending death.⁵

Following several reports of similar epidemics, researchers have hypothesised that this is a 'mass psychogenic illness' and 'mass hysteria' in which a group of people, usually from low socioeconomic and literacy backgrounds, start exhibiting symptoms following a triggering factor.⁶

Several case reports have outlined the link between cannabis use and the development of Koro. In these cases, several factors are thought to act synergistically, such as cannabis lowering the threshold for a panic reaction, previous knowledge of cannabis-induced Koro and poor body image or body dysmorphia.

Benzodiazepines, selective serotonin reuptake inhibitors and tricyclic antidepressants have all been used in successfully. Group psychotherapy, mass education programmes and awareness campaigns can be effective in the cases of mass epidemics.⁷

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25. Korsakoff's Psychosis

In a series of three articles from 1887 to 1889, Russian neuropsychiatrist, Sergei Sergeievich Korsakoff, described a persistent amnesic confabulatory state that subsequently became known as Korsakoff's psychosis or Korsakoff's syndrome. This is a part of the Wernicke-Korsakoff syndrome or encephalopathy.¹



The key features that he noted on at least 46 patients, of which two thirds were alcoholics, were:

- Anterograde amnesia. This is an inability to form new memories after the brain insult
- Retrograde amnesia, exemplified by deficits in remembering past events.

He emphasised the manifestation of these though the patient had clear attention and consciousness.

- Confabulation (fabrication of memories)

De Wardener and Lennox made the important observation that thiamine (vitamin B1) depletion is the key mechanism resulting in the acute Wernicke episode, followed by Korsakoff's syndrome in malnourished prisoners-of-war.²

It is most commonly associated with chronic alcohol abuse, but it can also result from inadequate dietary intake, reduced absorption or a reduction in the rate of conversion to the active metabolite.³

Neuronal loss, micro-haemorrhages and gliosis in the paraventricular and periaqueductal grey matter represent the core neuropathology.¹ Debate arises as to which specific brain lesion is critical for the manifestation of the prolonged and severe memory disorder rather than the Wernicke classical clinical triad of confusion, cerebellar dysfunction and ocular signs.⁴ The medial dorsal nucleus of the thalamus and the anterior principal thalamic nuclei are reported to be the key differences between patients who suffered a persistent Korsakoff syndrome and those who experienced only a transient Wernicke episode.^{1, 5} As the chronic memory loss often follows an episode of Wernicke encephalopathy, the chronic disorder is known as Wernicke-Korsakoff syndrome.

However, Korsakoff's syndrome can also develop in individuals without a definite prior episode of Wernicke encephalopathy.

Korsakoff syndrome is a clinical diagnosis, differential diagnoses include head trauma, anterior communicating aneurysm rupture and herpes encephalitis.

In order to prevent the development of a severe chronic Korsakoff state, it is recommended that 200 mg thiamine should be administered three times daily, preferably intravenously before any carbohydrate, should a diagnosis be suspected.⁴ While complete recovery is perceived as unlikely, improvement is noted to occur over the course of years in 75% of patients who remain abstinent from alcohol. However, 25% show no change, even post treatment.¹ This can cause ethical dilemmas for GPs when assessing patient's capacity.

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REFERENCES on request from Nick Mullin

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