Classification in Psychiatry

Classification is difficult in psychiatry because:
- A number of different diagnoses must be considered for most patients.
- Patients rarely have typical symptoms.
- Co-morbidity – often coexist.
- Can take many years to diagnose condition with certainty.

Most diagnoses are defined by combinations of symptoms, some by aetiology or pathology. Classification helps determine treatment required and prognosis.

Hierarchy – some diagnoses carry more weight – i.e. if they are diagnosed it is presumed that co-existing disorders are secondary to this. Normally an organic disorder takes precedence over other conditions. This means that it is important to look for a medical or substance-related cause of psychological symptoms first.

There are 2 main classification systems:
- ICD10
- DSM IV – axis system. I = all disorders except those in II. II = personality disorders, mental retardation. III = concurrent medical condition. IV = contributing social or environmental conditions. V = score of global assessment of functioning.

Both are categorical – divided by description.

<table>
<thead>
<tr>
<th>Category</th>
<th>Examples</th>
<th>Basic characteristics</th>
<th>Presentations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Organic disorder</td>
<td>Dementia, delirium</td>
<td>Impaired memory</td>
<td>Forgetfulness, confusion</td>
</tr>
<tr>
<td>Psychosis</td>
<td>Schizophrenia, bipolar disorder</td>
<td>'Loss of touch with reality'</td>
<td>Bizarre ideas or behaviour</td>
</tr>
<tr>
<td></td>
<td>Anxiety disorders, somatisation</td>
<td>Emotional disturbance</td>
<td>Worried, tired, physical complaints</td>
</tr>
<tr>
<td>Mood disorder</td>
<td>Depression</td>
<td>Low mood, anhedonia. Can have associated psychosis.</td>
<td>Tearful, fed up</td>
</tr>
<tr>
<td>Substance misuse</td>
<td>Alcohol, opiates</td>
<td>Effects of substance</td>
<td>Addiction, withdrawal</td>
</tr>
<tr>
<td>Personality disorder</td>
<td>Dissocial, paranoid</td>
<td>Dysfunctional personality</td>
<td>Exacerbated when stressed</td>
</tr>
<tr>
<td>Mental retardation</td>
<td>Downs</td>
<td>Congenital; low IQ</td>
<td>Developmental delay</td>
</tr>
</tbody>
</table>

The Mental Health Act 1983

This is an act of parliament that applies in England and Wales to allow assessment and treatment of people with psychiatric disorders against their will. Patients on a section can only be compulsorily treated for their psychiatric disorder, not for physical problems. Common law allows a doctor to treat a patient without consent where immediate action is required to save life.

This is needed as patients are at risk of self-harm, self-neglect, exploitation and harm to others. This is particularly a problem when patients lose insight and refuse treatment.

For the act to be used, the patient must have refused voluntary treatment and need detention for their own health and safety or that of other people. The patient’s behaviour must be due to a known or suspected psychiatric illness. ‘Mental illness’ is not defined but is left as a ‘matter for clinical judgment’.
The major sections are:

- **Section 2** – 28 days for assessment (as part of which medication may be given), needs 2 doctors and ASW. This is used when there is no clear diagnosis.
- **Section 3** – 3 months for treatment, needs 2 doctors and ASW. This is used to give treatment when there is a clear diagnosis and can be extended, for 6 months at a time.
- **Section 4** – 72 hours for emergency admission from community, needs doctor and ASW.
- **Section 5 (2)** – 72 hours to detain inpatient in emergency, 1 doctor. Cannot treat patient, transfer patient or repeat.
- **Section 5 (4)** – 6 hours to detain patient in emergency by nurse.
- **Section 136** – police officer to place of safety.
- **Section 135** – power of entry and removal to place of safety by magistrate.

Patients can be discharged by doctors, hospital manager or nearest relative (with notice, can be stopped by psychiatrist). A section 117 meeting is held to discuss future management.

Patients who have not been detained on a section by agree to voluntary admission are known as informal.

**Psychiatric History Taking and Mental State Examination**

Components of a psychiatric history:

- History
- Mental State Examination – at a given time, may fluctuate, try to do during course of interview.
- Differential diagnosis
- Risk assessment – to self, to others, to dependents.
- Management plan – investigations and treatment (biological, psychological, social).

Differs from medical history in that:

- Longer
- Can be therapeutic
- More interested in background etc.

Basic interview techniques:

- Private environment with chairs at same level and not directly opposing.
- Establish rapport immediately.
- Before starting explain need to take notes and time constraints.
- Closed questions can be used at the beginning of the interview for a nervous patient or to establish specific facts.
- Open questions are best the rest of the time.

**History**

Demographics – name, age, marital status, occupation.
Reason for referral and legal status i.e. under section

**HPC:**

- Duration, development, mode of onset, course,
- Severity
- Associated symptoms
- Precipitating factors.
• Should also screen for other common symptoms – low mood, elevated mood, delusions, hallucinations, anxiety, obsessions, compulsions, substance abuse.
• For eating disorders – weight control mechanisms, typical days intake, binges (frequency, amount), menstrual history, weight history, current height and weight, comorbidity.

Psychiatric history:
• Previous illnesses
• Previous treatments
• Previous sectioning
• Current treatment

Personal history:
• Birth and early developmental problems.
• Adversity, separation from parents, abuse.
• Relationships.
• Academic performance, friends, bullying.
• Current relationships, social support, employment and concerns.
• Forensic history.

Premorbid personality:
• ‘How would you have described yourself before you were ill?’
• ‘How would others have described you?’

Drugs and alcohol:
• CAGE questionnaire (>2 is positive) – cut down, annoyed at others, guilty, eye opener.
• For alcohol – type, daily unit intake, time of first drink, place of drinking. 14 units for women and 21 units for men is top recommended limit.
• For drugs – name, route of administration, years, frequency, dependence.

Family history:
• Family composition
• Family medical history
• Family psychiatric history
• Relationships
• Dependants

Past medical history – especially head injuries or surgery, neurological condition especially epilepsy, endocrine abnormalities, signs of substance abuse or self-harm.
**Mental State examination**

Appearance and behaviour:
- Clothes and accessories – appropriateness, cleanliness.
- Facial expressions, movements (tics, tremors, due to EPSE of anti-psychotics), social appropriateness, psychomotor function (retardation or agitation).
- Rapport – interaction, eye contact.

Speech:
- Rate, volume, quantity (pressure/poverty), fluency.
- Dysarthria (neurological disorder), dysphasia (focal neurologic disorder, thought disorder).
- Understandable? – flight of ideas (tenuous links – mania), formal thought disorder (no connection between topics – schizophrenia), circumstantiality (goes round in circles including unnecessary details but does get there – mania).

Mood and affect:
- Objective impression (affect).
- Subjective description (mood).
- Congruence, lability, reactivity, range.
- Anhedonia.
- Thoughts of suicide or self-harm.

Thought form:
- Formal thought disorder
- Tangential thinking
- Loosening of association - Knight’s move thinking, derailment

Thought content:
- Delusions – ‘an unshakeable (not by definition false) belief, without logical basis, that is held with conviction and out of keeping with cultural norms.’ Can be fleeting or sustained and systematised (affecting every bit of life) or encapsulated. Primary delusions arise spontaneously fully-formed and are suggestive of schizophrenia. Secondary delusions are associated with affective disorders.
  - Self-referential
  - Persecutory – ‘Have you ever felt that people are against you?’
  - Grandiose – ‘Do you have any special powers?’
  - Nihilistic
  - Passivity – ‘Have you ever felt that you were being directly interfered with by an outside force?’
- Obsessions – ‘a recurrent intrusive unwanted thought, which the patient recognises as their own.’ ‘Do you have any particular things on your mind at the moment?’
- Overvalued ideas – ‘a belief which is understandable and reasonable but which has come to dominate a person’s life.’

Perceptual abnormalities:
- Hallucinations – perception with no stimulus. ‘Have you ever seen/heard things that other people couldn’t?’ ‘Have you ever heard voices when there was nobody there?’
- Illusions – misinterpretation of stimulus.
Cognitive function:
- Usually MMSE.
- Alert, orientated, attention, concentration, memory.
- Cognitive impairment is typical of organic disorders.
- Various specific tests.

Insight:
- ‘Do you think you are ill?’
- ‘Do you think you need treatment?’
- ‘Are you happy to stay in hospital?’

**Risk assessment**
Risk to self (self-harm, suicide, self-neglect, exploitation), risk to others.

Features in the history associated with increased risk of harm:
- Previous self-harm or harm. Suicide attempts are more serious if planned, precautions taken to avoid discovery, on help sought after, method considered dangerous and note left.
- Recent actions e.g. making will.
- Recent major stresses.
- Depressive disorder, psychosis, substance misuse, personality disorder.

Features in mental state:
- Suicidal or violent thoughts.
- Significant mood disturbance.
- Psychotic symptoms especially passivity.

Features in context:
- Single men.
- Substance abuse.
- Social restlessness.
- Easy access to weapons.

Risk formulation:
- What are risks?
- How probable?
- How immediate and long lasting?
- What can be done to reduce risk, what may affect probability?

Risk management:
- Reduce risk, maintain at low level and review.
- Immediate – admission and MHA, sedation, make people aware, police.
- Longer term – treat disorder, remove stresses, teach coping strategies.
Formulation

This information can be presented in a formulation:

- Description of patient
- Differential diagnosis
- Aetiology – why this patient, with this problem, at this time: predisposing, precipitating, perpetuating; and biological, psychological and social.
- Management – biological, social and psychological. Short-term and long-term.
- Prognosis based on natural course of condition and patient factors.

Psychopathology

24% of the population has some sort of mental disorder.
About 5% have depression.
About 1% have schizophrenia.
About 1% have bipolar affective disorder.

About 1/3 of all patients who go to their G.P.s have primarily emotional problems.
About one in 20 is referred to a psychiatrist.

Risk factors for psychological problems:

- Predisposing – personality type, development, genetics, early experiences, environment.
- Precipitating – developmental crisis, life events, physical illness, contextual threat, natural history.
- Perpetuating – wrong diagnosis or treatment, chronic stress, substance abuse, non compliance.

Psychopathology is the systematic study of abnormal experience cognition and behaviour. It is used to describe and classify mental experiences and subsequent behaviour.

- Descriptive psychopathology - avoids theoretical explanations and describes as accurately as possible the patient's experiences.
- Dynamic psychopathology - attempts to explain psychic events in terms of psychodynamic theories of aetiology e.g. Freudian or Jungian developmental theory.

Psychiatric diseases can be:

- Normal behaviour in the wrong amount (neuroses) e.g. anxiety, obsessions.
- Abnormal behaviour (psychosis).
- Change in mood linked with both.
Neurotransmitters

Anxiety:
- Low levels of GABA – inhibition, calming, sedation.
- Low serotonin – impulsivity and ability to cope with stress.
- High adrenaline and noradrenaline.

Schizophrenia:
- Overactive dopamine – psychosis.
- Underactive glutamate – poor cognition and attention.

Depression:
- Underactive serotonin and noradrenaline.

Alzheimer’s:
- Decreased A Ch.

Parkinson’s:
- Decreased dopamine.
Anti-Psychotic Drugs

Mainstay of treatment for schizophrenia and other psychoses - reduce hallucinations, delusions and thought disorders. Also have a tranquilising effect without impairing consciousness. Tolerance can develop to sedative effect but not anti-psychotic. These are not addictive.

Classification

Classical:
- Older conventional drugs.
- Cause side effects especially motor disturbance (extra-pyramidal) and endocrine disorders.
- 3 types:
  - Phenothiazines e.g. chlorpromazine (group 1), thioridazine (group 2), trifluoperazine (group 3).
  - Thioxanthines e.g. flupenthixol, clopenthixol.
  - Butyrothenones – haloperidol, pimozide.
  - Alkaloids – reserpine.

Atypical:
- Becoming more popular.
- Less EPSE.
- Better for negative symptoms.
- Clozapine, olanzapine, sertindole (limbic selective), quetiapine, risperidone, amisulpiride.
- Clozapine is also more potent than classical drugs and can treat resistance cases.

Mode of action

Block transmission of various neurotransmitters. Common action is blockade of transmission through dopaminergic synapses.
- Dopamine receptor blockers – D2 receptors, most drugs
- Dopamine depleters – reserpine, also depletes NA and 5HT, so can cause depression.

The drugs have an effect in areas where there are dopamine receptors. This can be therapeutic or cause side effects:
- Limbic system and neocortex – have antipsychotic effects, but may worsen negative and cognitive symptoms of schizophrenia.
- Striatum – cause parkinsonian side effects.
- Hypothalamus to infundibular region – cause hyperprolactinaemia (as D is tonically inhibitory).
- Brain stem chemoreceptor trigger zone – antiemetic.

Atypical antipsychotics:
- Mechanism unsure.
- Multi-receptor interactions e.g. block 5HT2 receptors, prevent activation of mesolimbic dopaminergic pathways but not others.
- Limbic selectivity
**Indications:**
Acute calming of disturbed patient regardless of underlying psychopathology – short acting potent drug as injection. Chlorpromazine is good as sedating.

Treatment of acute psychotic state, typically haloperidol generally or atypical antipsychotic for acute schizophrenia:
- e.g. organic psychoses, acute schizophrenia, mania, psychotic depression.
- Cause calming and anti-psychotic effect (reduce hallucinations, delusions and thought disorder).
- 75% effective. Clozapine is effective 30% of those who are resistant to typical anti-psychotics. This is only used when patients who are unresponsive to or intolerant of 2 other drugs after 6 weeks and will be compliant with tablets and blood tests.
- Need about 2 weeks to work.

Maintenance treatment – especially for chronic schizophrenia. Reduces rate of relapse. Often given as depot injections:
- Modify drug structure to ester (decanoate).
- Advantages – ensure compliance, avoid first pass metabolism, steady plasma levels.
- Disadvantages – hard to deal with sudden side-effects, higher levels of EPS.

Withdrawal should be gradual and closely monitored.

**Side effects**
Be extra careful in post-partum, elderly, obese, hepatic and renal impairment, Parkinson’s (made worse), angle closure glaucoma, epilepsy (lowers seizure threshold).

Different drugs have different profiles. In general:
- Group 1 phenothiazines e.g. chlorpromazine – pronounced sedative effect, moderate antimuscarinic and EPSE.
- Group 2 phenothiazines e.g. thioridazine – pronounced antimuscarinic, moderate sedative, and fewer EPSE.
- Group 3 and other classes e.g. haloperidol, trifluoperazine – pronounced EPSE, fewer sedative and antimuscarinic effects.

Neurological:
- Due to central dopamine receptor blockade.
- EPSE:
  - Parkinsonism - most common, rigidity, tremor, bradykinesia
  - Akathisia - experience of inner restlessness after large initial dose
  - Acute dystonia - sudden spasm of muscles especially neck and eyes – torticollis and oculogyric crisis, after a few doses.
  - Tardive dyskinesia - later in treatment affecting about 20% after 2 years, choreoathetoid involuntary movements, mainly face and tongue, may be irreversible on withdrawing therapy.
  - Neuroleptic malignant syndrome - fever, coma, muscle rigidity, high mortality.
- Butyrophenones (haloperidol, pimozide) and group 3 piperazine phenothiazines (trifluoperazine, prochlorperazine) are worst.
- Anticholinergic drugs e.g. procyclidine may be used to counteract EPSE except tardive dyskinesia, which may be worsened.
- Lowered seizure threshold.
Autonomic:
- Difficulty regulating temp – hypo and hyper thermia
- Fall in blood pressure – hypotension. Chlorpromazine and thioridazine are worst.
- Muscarinic receptor blockade – blurred vision (no accommodation), dry mouth, tachycardia, constipation.

Psychiatric - apathy, confusion and depression (especially reserpine)

Hyperprolactinaemia – breast enlargement, lactation, impotence (males), amenorrhoea (females), infertility.

Hypersensitivity reactions:
- Liver – cholestatic jaundice, especially chlorpromazine.
- Bone marrow – agranulocytosis or aplastic anaemia
- Skin – rashes and dermatitis, photosensitivity.
- Cardiotoxicity

Clozapine:
- Bone marrow suppression and agranulocytosis – blood cell count monitoring.
- Sedation – diminished through treatment.
- Hypersalivation – long term especially at night
- Reduction of seizure threshold
- Orthostatic hypotension
- Weight gain

Olanzapine - generally better tolerated, weight gain, increased risk of stroke in elderly patients with dementia, hyperglycaemia.

Risperidone - EPSE and hyperprolactinaemia especially at higher doses, increased risk of stroke in elderly patients with dementia.

Quetiapine – sedative, can develop tolerance.
Anti-Depressants

Lower levels of mono-amines (NA, DA, 5HT) are implicated in depression. Anti-depressants work in various ways to increase levels of these. This may lead to activation of brain-derived neurotrophic factor. They are generally non-addictive, though SSRIs may be associated with a withdrawal syndrome.

In general, drugs take about 2-4 weeks to work. However, in about 25%, they may take as long as 6 weeks to have an effect. The dose should be build up over about a week, continued at full dose for 6 months after recovery, then stopped gradually. If patients do not respond then increase dose or switch classes, then add lithium or liothyronine.

The dose depends on diagnosis – panic < anxiety < depression, OCD (clomipramine) < bulimia nervosa (fluoxetine).
Anti-depressants should not be used in depression associated with mania.

Tri-cyclic antidepressants
These are fairly non-selective noradrenaline reuptake inhibitors.
They are typically given daily at night.
About 80% of patients respond, less in very mild or severe cases.

These are divided into:
- Sedating – amitriptyline, dothiepin, trazodone, doxepin, good for anxious agitated patients.
- Activating – imipramine, lofepramine, good for withdrawn patients.

The big problems with TCAs are side effects and toxicity in overdose.
Side effects are due to lack of specificity:
- Anti-cholinergic – dry mouth, blurred vision, urinary retention, confusion in elderly. Generally develop some tolerance.
- Alpha1 adrenergic – postural hypotension, dizziness.
- H1 blockade – sedation, weight gain.
- Arrhythmia – at therapeutic dose get tachycardia, flat T waves, prolonged QT.
- Weight gain, seizures, SIADH.

In overdose, TCAs cause treatment resistant arrhythmias, convulsions, coma and death.

TCAs are also used for nocturnal enuresis, cataplexy (sudden loss of muscle tone with emotion) and chronic pain.

Amitriptyline – panic disorder (low dose), anxiety (medium dose), depression (higher dose).
Lofepramine – modified tricyclic, well tolerated, less sedating and safer in OD.

Serotonin selective reuptake inhibitors
These block reuptake of 5HT.
They are relatively safe in overdose and less sedating

SSRIs have a similar efficacy to TCA (70% response rate) and are particularly effective for depression associated with:
- Premenstrual worsening.
- Obsessional symptoms.
- Multi-impulsive symptoms.
They should probably also be chosen over TCAs when there is a serious suicide risk, intolerance of TCA side effects and in patients with diabetes or heart disease but avoided in epilepsy.
As these are more specific, they have different and generally fewer side effects than TCAs. However, they can cause nausea (tends to resolve), agitation, insomnia, altered sexual function, weight loss, headache and hyponatraemia due to SIADH.

Fluoxetine - 20mg per day, long half-life (>week, due to metabolite). Good for panic, anxiety, depression, OCD, bulimia nervosa.
Paroxetine – EPSE, less metabolism by liver, withdrawal reactions.
Sertraline.
Citalopram – safe with alcohol, fewest interactions.
Trazodone – good for some SSRI side effects, sedative.

Newer drugs:
- SNRI (serotonin and nor-adrenaline reuptake inhibitors) – like TCA but more specific and maybe more effective e.g. venlafaxine is associated with less sedation and postural hypotension.
- NaSSA (noradrenergic and specific serotonergic antidepressant) – sedative effects e.g. mirtazapine.
- NRI (noradrenaline reuptake inhibitor) – reboxetine.

**Monoamine oxidase inhibitors**
Second line antidepressants for resistant depression.
First line for atypical depression – increased sleep and appetite or with phobic anxiety.

Inhibit neuronal MAO so amines accumulate:
- MAO inhibitors – irreversible e.g. phenelzine.
- Reversible MAOa inhibitors – selective for central MAO, less ‘cheese reaction’ e.g. moclobemide.

Problems:
- Interactions (cheese reaction) – tyramine is pressor to smooth muscle in arterioles to increase BP. When gut MAO is inhibited tyramine in food e.g. broad beans, cheese, marmite is not metabolised causing a hypertensive crisis. This is treated by alpha1 adrenergic blockade.
- Interact with other drugs e.g. ephedrine, lithium, other antidepressants (serotonin syndrome) – must leave a 2 week gap (longer for fluoxetine) between drugs.
- Side effects – hypotension, insomnia, dry mouth, agitation, headache.
Mood Stabilisers

These are used to prevent mood swings in bipolar disorder as treatment and prophylaxis (episodes less frequent and severe). They are effective to treat acute mania, though slower acting than antipsychotics (can be given together with atypical antipsychotic initially until lithium becomes effective, needs caution as antipsychotics increased lithium-induced neurotoxicity). They are also used alongside antidepressants in resistant depression.

**Lithium**

Efficacy is related to serum levels:
- Very narrow therapeutic range so need monitoring of serum drug levels.
- Prophylaxis - low level, 0.5-0.8 mmol
- Acute mania – 1-1.4mmol
- Serious toxicity >2.0 – generally treat by giving sodium and rehydrating to increase urine output.

Blood tests:
- Before starting therapy – FBC, renal function, U&Es, TFT, pregnancy test, ECG.
- Drug levels – weekly until stable then every 3 months.
- TFT and U&Es are monitored every 6 months.

Sodium depletion leads to toxicity:
- Patients must be educated about the risk of thiazide diuretics, hot weather, dehydration, vomiting.
- Contra-indications are renal impairment and sodium imbalance – these should be checked before starting the drug. Lithium is excreted almost entirely by the kidneys.

Mechanism is unclear – thought to be related to neuro-transmitter induced 2nd messenger systems.

Side effects:
- General – metallic taste, fatigue, tremor, polyuria, weight gain, oedema, GI disturbances.
- Thyroid – goitre (5%), hypothyroidism (20%, rarer in men).
- Renal – nephrogenic diabetes insipidus, renal failure.
- Toxicity (dose related) – polyuria, ataxia, confusion, seizures, coma, death.
- Teratogenic – cardiac malformations about 5%.

**Valproic acid**

Useful in patients unresponsive to lithium.

**Carbamazapine**

This can be used if lithium is contraindicated or not tolerated.
It is more effective in rapid cyclers.
Side effects – rash, leucopenia, nausea, dizziness.
This is a potent enzyme inducer so interacts with other drugs.
Anxiolytics and Sedatives

These are GABA A receptor transmission enhancers. They include – benzodiazepines, barbiturates, anaesthetics, alcohol.

Benzodiazepines
These are good drugs as they are effective and have few side effects. The lethal dose is very high, there is no loss of consciousness or respiratory depression, less effect on sleep pattern and they do not induce enzymes. These have a specific binding site on the GABA A receptor, which facilitates GABA binding.

The main problem is dependency:
  Habituation to effect in 2 weeks.
  50% dependency at 6 months.
  Withdrawal syndrome – withdraw gradually, changing to a long acting drug first e.g. diazepam. For this reason they should only be used as short-term management (2-4 weeks) and other ways of managing anxiety should be considered first. Anti-depressants are now used to treat long-term anxiety disorders.

Indications:
• Acute psychosis alongside anti-psychotics e.g. lorazepam 1-2 mg (only drug absorbed reliably IM).
• Short term relief of severe anxiety e.g. diazepam (long acting), lorazepam (short acting).
• Alcohol withdrawal.
• Insomnia e.g. temazepam

Zopiclone – binds to specific subunit (partial agonist). Lower abuse potential and withdrawal effects.

Other drugs
Busiprone – serotonin receptors, low dependence and abuse potential, takes 2 weeks to respond to treatment. Used for generalised anxiety, not hypnotic.
Beta-blockers – to reduce autonomic symptoms of panic.
Barbiturates – severe insomnia (only if already taking barbiturates), avoid where possible due to dependence.
ECT

ECT is used for:
- Treatment resistant depression.
- Psychotic depression and depressive stupor.
- Catatonic schizophrenia.
- Post-partum psychosis.
- Acute mania.

It is especially good in pregnancy (no drugs) and the elderly (side effects).
There are no absolute contra-indications.

It is effective in about 70% of cases. Typically, an improvement is seen after a few treatments.

Modified ECT involves using a GA with muscle relaxant then inducing a generalised seizure by electric shock. The treatment is given once or twice a week for 6-12 weeks.
Treatment is either:
- Bilateral – slightly more effective but more short-term memory impairment.
- Unilateral – given to the non-dominant hemisphere.

The mechanism is unclear but it has been proved that the seizure is the effective component. A seizure causes release of monoamines, giving very high levels in the brain.

Adverse effects include:
- Those related to any general anaesthetic.
- Brief retrograde and anterograde amnesia.
- Headache.
- Rarely, confusion, delayed seizure, prolonged seizure (especially if on antidepressants or antipsychotics).

The other physical treatment is psychosurgery. This is now very rarely used (only for intractable OCD and depression).
**Psychotherapies**

These are defined as:
the range of psychological therapies that use the application of psychological models to promote understanding and change.
Interactions between a therapist and client that leads to beneficial changes in the client’s thoughts, feelings or behaviours.

They basically involve:
- observing, listening, speaking.
- understanding what the patient says and does.
- applying a particular theoretical framework.

<table>
<thead>
<tr>
<th></th>
<th>Psychotherapy</th>
<th>Counselling</th>
</tr>
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<tbody>
<tr>
<td><strong>Duration</strong></td>
<td>longer</td>
<td>shorter</td>
</tr>
<tr>
<td><strong>Problem</strong></td>
<td>complex, global, pervasive</td>
<td>less complex, often previously healthy</td>
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<tr>
<td><strong>Overall situation of patient</strong></td>
<td>relatively stable</td>
<td>in crisis</td>
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**Cognitive behavioural therapy**

This is based on:
- Behavioural model: situation -> emotion.
- Cognitive model: situation -> meaning (thinking) -> emotion

These emotions can be exaggerated:
- Fear (danger) -> anxiety disorders
- sadness (loss) -> depression
- happiness (increase in personal space) -> mania
- anger (intrusion to personal space) -> psychopathy

Cases can be conceptualised as:
- Cross sectional (‘hot cross bun’) – cognition, behaviour, emotion, physiological responses all interact with each other in positive feedback cycles.
- Longitudinal (how the situation arose) – beliefs and assumptions lead to aberrant thoughts (activation - due to a critical event).

**Indications for CBT -**

Stand alone treatment for:
- anxiety and panic disorders
- unipolar depression (but usual to continue antidepressants) – in mild depression is as effective as anti-depressants.
- bulimia nervosa

Adjunct in:
- psychoses
- addictions
- anorexia nervosa
- personality disorder

Exposure treatments involve exposing the patient to the situation they have come to avoid. As the person becomes accustomed to the situation panic subsides and on re-exposure is less. It also involves challenging ways of thinking that maintain the problem.
Work outside the session is very important e.g. behavioural experiments to test hypotheses (false beliefs that perpetuate problem).

**Psychodynamic psychotherapy**
This is about exploring the personal meaning of problems.
As such the key intervention is interpretation of unconscious process with relation to past, present or therapist interactions.
This involves:
- an opportunity to explore problems with a therapist regularly, over time in a non-judgmental and confidential setting.
- using past experiences to make sense of current problems.
- seeking to understand the personal meaning of symptoms and patterns of behaviour in order to help to change them.

Conditions include:
- relationship difficulties
- childhood trauma affecting present ability
- personality problems
- unhappiness/depression
- anxiety states
- eating disorders

Psychoanalytic principles:
- Transference – the feelings and attitudes the patients develop towards the therapist – these represent emotions transferred inappropriately to the therapist from the person they were originally attached to.
- Countertransference – the feelings the patient produces in the therapist.
- Unconscious process – processes we are unaware of. The therapist helps patients become more aware of these.
- Interpretation – how the therapist helps the patient become more aware of unconscious processes.
- Defence mechanisms – individuals employ this when anxiety-producing aspects of the self that are unconscious threaten to become conscious. This includes repression and projection. Analysis of these is important.

**Family therapy**
For children.
For families of schizophrenics to reduce high expressed emotion.
Social treatments

Social factors:
- Are important in aetiology and perpetuation of psychiatric disorders.
- Affect the success of treatment and quality of life.
- Changing social factors can be therapeutic in itself.

Acute interventions include psychiatric admission and crisis intervention e.g. finding temporary accommodation.

Long term treatments involve:
- Helping with accommodation and financial problems.
- Supporting and educating family.
- Addressing isolation.
- Rehabilitation to help social skills and self-confidence.
Organic Psychiatry

Organic psychiatric means:
- Psychiatric disorders arising from demonstrable structural disease of the brain e.g. brain tumours, injuries, degeneration.
- Psychiatric disorders arising from brain dysfunction caused by disease outside the brain e.g. myxoedema.
- Generally, these involve altered state of consciousness or memory impairment.

Syndromes can be subdivided based on:
- Generalised or specific impairment of psychological functioning.
- Acute or chronic.
- Generalised or focal underlying brain dysfunction.

Classification

ICD10
Organic, including symptomatic, mental disorders:
- Dementia in Alzheimer's disease
- Vascular dementia
- Dementia in other diseases classified elsewhere, e.g. Parkinson's disease, HIV
- Organic amnesic syndrome, not induced by alcohol or other psychoactive substance
- Delirium, not induced by alcohol or other psychoactive substance

Other mental disorders due to brain damage and dysfunction and to physical disease:
- Organic hallucinosis e.g. due to alcohol.
- Organic delusional (schizophrenia-like) disorder - especially due to epilepsy.
- Organic mood (affective) disorders – e.g. secondary to cancer, infections, thyroid disease, SLE, drugs (steroids, L-Dopa, anti-convulsants, oral contraceptives, Ca channel blockers)
- Organic anxiety disorder – hyperthyroidism, phaeochromocytoma, hypoglycaemia, drug withdrawal.
- Organic personality disorders – after head injury.

Dementia and delirium are defined by their characteristic clinical features.
Other organic disorders have the same symptoms as functional disorders but are differentiated by an identifiable medical disorder.
Psychological reactions to illness e.g. becoming depressed after diagnosis are not organic disorders.

Delirium

This involves:
- Acute generalised psychological impairment
- Generalised underlying brain dysfunction
- Impairment of consciousness (characteristic)

It is common and is estimated to affect 5 to 15% general medical/surgical patients.
Typically it develops usually over hours to days and most cases recover quickly (within 4 weeks or less) or die.

There are many causes. The majority are outside the brain and reversible.
- Drugs e.g. anticholinergics, anxiolytics, or withdrawal of drugs e.g. alcohol, anxiolytics.
- Metabolic failures e.g. liver, cardiac, respiratory, renal.
- Electrolyte balance disorders.
Endocrine disorders e.g. myxoedema, hypoglycaemia.
- Systemic infection e.g. sepsis, pneumonia.
- Intracranial infection e.g. encephalitis, meningitis, HIV, cerebral malaria.
- Other intracranial causes e.g. space occupying lesion, raised pressure, head injury
- Nutritional deficiency e.g. thiamine, vitamin B12.
- Epileptic e.g. status, post-ictal.

Clinical features:
- Clouding of consciousness.
- Global impairment of cognitive processes e.g. thinking, attending, memory.
- Reduced awareness of self and environment.
- Disorientation in time and place.
- Intensity fluctuating through day, typically worse at night.
- Slowness or drowsiness.

Other possible features include:
- Overactivity, restless, oversensitive to stimuli, irritable, psychotic.
- Inactivity, lethargic, quiet, slowness, reduced speech, perseveration.
- Hallucinations – typically visual.

Management is mainly directed towards treating the underlying cause.

It is also important to:
- Orientate patient – consistent approach, repeated explanations, use relatives.
- Keep patient calm – quiet single room.
- Drugs – give as few as possible, calming by day if at risk (haloperidol) and to promote sleep (short acting benzodiazepines).

**Dementia**

This is a chronic generalised psychological impairment of intellect, memory and personality, with no impairment of consciousness. Symptoms should be present for 6 months of sufficient severity to impair functioning.

The underlying brain dysfunction is generalised and the primary cause is usually within the brain e.g. Alzheimers. This is acquired, and generally irreversible and progressive.

Differential diagnosis – mild dementia may be confused with depression (poor concentration and memory, low mood comes first), also delirium, deafness, dysphasia, amnesic syndrome, late onset schizophrenia.

**Clinical features:**
- Memory impairment – difficulty with new learning, may confabulate, recent memory is affected more.
- Behavioural changes – may become disorganised, inappropriate, distractible and restless.
- Thinking – slows, poverty of content, perseveration, impaired judgement.
- Speech – syntactical errors, nominal dysphasia, later may become meaningless.
- Mood – anxiety, irritability, depression, later may be blunted or labile.
- Psychotic symptoms – visual hallucinations, persecutory delusions, lack of insight.
Aetiology:
- Affects <5% of 65 yr olds but 20% of 80 yr olds. Dementia in under 65s is called presenile dementia and tends to have different causes e.g. trauma, genetic, infectious.
- Alzheimer's (60%).
- Vascular multi-infarct dementia (20%).
- Lewy body dementia (15%).
- Degenerative e.g., Pick's disease, normal pressure hydrocephalus (treatable), prion disease.
- Metabolic e.g. sustained uraemia, liver failure, anoxia, alcohol dementia, B12 deficiency.
- Intracranial SOL e.g. tumour, subdural haematoma.
- Trauma
- Infection e.g. encephalitis, neurosyphilis, HIV.

Alzheimer's disease:
Pathology - neurofibrillary tangles and beta-amyloid plaques, loss of Ach, can be genetic (APP, presenilin, APO E4). Prominent memory loss, persecutory beliefs. Progressive.

Vascular dementia
Personality change, labile mood, preserved insight.
Signs of vascular disease.
Stepwise progression.

Lewy body dementia:
Fluctuating, parkinsonism, visual hallucinations.
Don't give antipsychotics.

Pick's disease:
Frontal lobe dysfunction, personality change, memory preserved.
Commoner in women.
Normal pressure hydrocephalus:
Frontal lobe dysfunction, urinary incontinence, gait apraxia.

Management involves firstly treating the cause if possible. Investigations to find a treatable cause (especially in younger people). However, most of the causes are irreversible so treat holistically.

Need to assess the degree of disability and social circumstances and improve functional ability where possible. This can be achieved by:
- Behavioural methods - positive reinforcement, shaping, desensitization.
- Prompts for memory deficit.

Drugs may be needed to relieve distressing symptoms. Initially, small doses should be given as patients are likely to be old and more sensitive to medication.
- Anxiety and agitation – haloperidol, benzodiazepine, phenothiazine.
- Night sedation – benzodiazepines, sedating phenothiazine.
- Delusions or hallucinations – phenothiazine.
- Depression – antidepressants.

Alzheimer's can be treated with drugs to inhibit ACh breakdown e.g. donepezil.

It is also important to consider practical provisions for patient, long term care, legal arrangements for financial affairs and support for the family.
Subcortical dementia
This is a syndrome of cognitive slowing, difficulty with complex intellectual tasks and affective disturbance without impairment of language, calculation or learning. The distinction from cortical dementia (e.g. Alzheimer's) remains controversial.

Possible examples include:
Huntington's disease
Parkinson's disease
Progressive Supranuclear Palsy
Hydrocephalus
AIDS dementia complex

Amnesic syndrome (Wernicke-Korsakov syndrome)
In this, the disorder of psychological functioning is specific. There is a:
• Prominent disorder of recent memory – digit span recall good first few seconds, then impaired few minutes/hours later.
• Grossly defective new learning, confabulation is common and they are often suggestible.
• Disordered time sense and associated disorientation in time.
• Absence of generalised intellectual impairment, otherwise cognitively intact.
• Some emotional blunting and lack of volition.

This usually results from lesions in the posterior hypothalamus but rarely can also be due to bilateral hippocampal lesions.

Aetiology:
• Thiamine deficiency – due to alcohol abuse (commonest cause), gastric carcinoma, severe dietary deficiency.
• Haemorrhagic lesions – especially mamillary bodies and medial dorsal nucleus of thalamus.
• Focal brain damage - vascular lesions, CO poisoning, encephalitis, tumours in 3rd ventricle
• Surgery - bilateral hippocampal damage, tend to be less likely to confabulate or lose insight.

Prognosis:
• About 20% died acutely.
• Of the rest - 25% complete recovery, 25% partial, 50% no recovery

Frontal lobe syndrome
This affects personality rather than cognition.
• Lack of initiative and spontaneity
• Decrease in activity - sluggish, unfinished tasks, need for prompting. Occasionally become restless and hyperactive but this is not goal directed.
• Mood – inappropriately mildly euphoric alternating with apathy and inertia, irritable, petulant ("witzelsucht").
• Social awareness and behaviour – tactless, sexual disinhibition, poor judgement.

Psychometry generally shows normal intellect, but specific tests may reveal loss of abstraction, incapacity to shift between frames of reference and inattention.
Assessment
Detailed history from patient and other informants.
Examination - intellectual function, neurological symptoms, physical examination.
Standardised mental state schedules e.g. MMSE at different times of day.
Investigations - FBC, ESR, MSSU, urine drug screen, LFT's, ca, phosphate, TFT's, serum B12, red cell folate, HIV, syphilis.
Imaging - CXR, lateral SXR, CT, MRI.
EEG
Psychometric testing e.g. Wechsler Adult Intelligence Scale, Wisconsin Card Sorting Test.

Organic mood disorders
Mania – high dose steroids, cerebral pathology
Depression – steroids, antihypertensives, Cushings, hypothyroidism, CVA.
Schizophrenia

Schizophrenia is a psychiatric disorder with characteristic symptoms that leads to a deterioration in function. Incidence is about 1% with an equal sex ratio (though men tend to develop it earlier and have worse prognosis). Incidence is higher in Afro-Carribeans and lower social classes.

Clinical presentation
Generally presents in late adolescence or early adulthood.

Positive symptoms (presence of abnormal mental activity):
- Thought disorder.
- Reality distortion – delusions and hallucinations.

Negative symptoms (diminution of normal mental activity):
- Social withdrawal.
- Decreased emotional response.
- Cognitive dysfunction and dementia.
- Tend to present later in illness and be harder to treat.

Schneider’s first rank symptoms (if these are present the diagnosis is more likely to be schizophrenia):
- Thought insertion, withdrawal and broadcast (delusions) and thought echo (1st person auditory hallucination).
- Delusions of control (made will, made acts, made affect) and somatic passivity and somatic hallucinations.
- Delusional perception.
- 3rd person auditory hallucinations e.g. commentary, discussing person.

Can also get:
- Other delusions especially paranoid.
- Other hallucinations.
- Formal thought disorders – derailment, knight’s move thinking, tangentiality, neologisms, word salad.
- Inappropriate affect or behaviour.

Diagnosis
ICD 10 diagnostic criteria requires:
- 1 or more strongly characteristic symptom e.g. 1st rank or persistent bizarre delusion.
- 2 or more less characteristic symptoms e.g. formal thought disorder, catatonia, negative symptoms.
- Duration of >1 month (6 months for DSM IV including prodrome).
- Exclusion of organic cause, affective psychosis, drug toxicity.

Differential diagnosis:
- organic disease (about 5% of cases)
- temporal lobe epilepsy
- neoplasm of the cerebral cortex
- substance abuse – hard to differentiate as many people with schizophrenia abuse alcohol or drugs.
- acute and transient psychotic disorder
- delusional disorder
- schizoaffective disorder – symptoms of both schizophrenia and affective disorder.
- mood disorder with psychotic features
Types of schizophrenia:
- Paranoid – predominant persecutory systematised delusions and auditory hallucinations. This is the commonest form and personality is usually preserved.
- Hebephrenic (disorganised) – predominant thought disorder and odd behaviour. Early onset with poor prognosis.
- Catatonic – motor signs, now rare.
- Undifferentiated.

**Natural history**
Acute schizophrenia – positive symptoms.
Residual schizophrenia – transition state.
Chronic schizophrenia – negative symptoms, poor functioning.

40% - undulating course, some persistent deficits.
25% - chronic schizophrenia, persistent functional disability.
25% - few episodes with full recovery.
10% suicide, especially after acute episode.

Prognosis is worse with younger onset, insidious onset, male, isolation, premorbid personality disorder, substance misuse, disorganised subtype, negative symptoms.
Relapse rate is increased by high expressed emotion, long duration of previous episode. 2/3s wills relapse if anti-psychotic is discontinued within 5 years.

**Aetiology**
Thought to be developmental rather than progressive.

Predisposing factors:
- Genetic component - twin concordance (45% in monozygotic twins), in adoption studies risk is determined by biological parent.
- Early lesion hypothesis – intra-uterine insult e.g. maternal viral infection, maternal starvation, maternal stress or birth complications increase risk.

Precipitating factors:
- Stress
- Drug abuse e.g. amphetamines, cocaine, marihuana.

Pathology:
- Overactivity of dopamine – positive symptoms.
- Reduction of 5HT – negative symptoms.
- Decreased glutamate – dopamine inhibits glutamate function.
- Enlarged ventricles and reduced temporal lobe volume.
- Changes in blood flow.
Management
Investigations:
- Physical - drug screen, look for organic cause, bloods, EEG, CT
- Psychosocial – collateral history, home environment.

Treatment goals:
- Alleviate symptoms and impairments.
- Prevent relapse.
- Minimize side effects
- Enhance quality of life

Use care programme approach to co-ordinate service delivery.

Treatment:
- Generally needs admission in acute phase.
- Biological - antipsychotic medication for positive symptoms and to prevent relapse, continue for 1-2 years after single episode. Benzodiazepines for distress.
- Psychological – CBT (coping with stress, compliance, improving insight), adequate challenge and autonomy in chronic phase, family therapy to decrease expressed emotion.
- Social – relationships, occupation, accommodation, social skills training.

Associated conditions
Schizophreniform disorder – features of schizophrenia but present for <1 month.
Paraphrenia – schizophrenia beginning after the age of 45.
Schizotypal disorder – cold suspicious manner, eccentric behaviour, avoidance of social contact, vague speech, tendency to odd ideas (like mild schizophrenia, not quite psychotic).
Schizoaffective disorder – features of both conditions in the same episode.
Neuroses – Anxiety and Stress related disorders

<table>
<thead>
<tr>
<th>Anxiety</th>
<th>Depression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Worry, apprehension or fear</td>
<td>Loss of enjoyment, energy or hope</td>
</tr>
<tr>
<td>Future directed</td>
<td>Past directed</td>
</tr>
<tr>
<td>Onset insomnia with vivid dreams</td>
<td>Early morning wakefulness</td>
</tr>
<tr>
<td>Autonomic symptoms: tachycardia and increased BP, palpitations, sweating</td>
<td>Suicidal thoughts, loss of libido, changes in appetite, poor concentration</td>
</tr>
</tbody>
</table>

At extremes, anxiety and depression are different, however, symptoms overlap in milder forms.

Anxiety disorders involve a maladapted fight or flight response. The person perceives danger and experiences fear, where most people would not. They are the most common type of psychiatric disorder. Memory, fear and stress interact to produce anxiety.

Symptoms of anxiety are:
- Behavioural – escape behaviour, agitation, sleeplessness, nausea, anorexia.
- Autonomic – tachycardia, palpitation, raised bp, sweating, tremor.
- Cognitive - inability to concentrate, perpetuating thoughts, anticipation.
- Emotional – fear

Patients may present due to anxiety, cognitions, behaviour or physical symptoms. Avoidance tend to perpetuate the problem as it reinforces the anxiety.

Classification

Basic types of anxiety disorders include:
- panic disorder
- agoraphobia
- specific phobias
- social phobia
- generalised anxiety disorder
- anxiety due to a general medical condition e.g. thyrotoxicosis, phaeochromocytoma, hypoglycaemia, drugs.

Phobic avoidance is prominent in specific phobias, social phobia and agoraphobia. Panic attacks are prominent in panic disorder.
### Specific phobias
- **Features**: Fear of a discrete object or situation e.g. spiders.
- **Epidemiology**: Common, about 7%. Early onset.
- **Secondary morbidity**: Avoidance.

### Social phobia
- **Features**: Fear of being ridiculed in public e.g. eating, blushing, public toilets. Comfortable when alone (cf agoraphobia).
- **Epidemiology**: Prevalence about 4%. Insidious onset. More common in males and substance abusers.
- **Secondary morbidity**: Isolation. Avoidance.

### Agoraphobia
- **Features**: Characteristic unfocussed pattern of fears and avoidance of crowds, open spaces etc. Uncomfortable alone.
- **Epidemiology**: Prevalence 3%. Generally sudden onset in early adulthood. Typically women with children.

### Panic disorder
- **Features**: Panic attacks. May be associated agoraphobia. Sympathetic arousal. Escape and safety behaviour.
- **Epidemiology**: Prevalence 1%. Sudden onset. More common in females.
- **Secondary morbidity**: Avoidance leads to agoraphobia. Medication abuse. Parasuicide. Dependency.

### Anxiety disorder
- **Features**: Excessive anxiety or worry for >6months, which becomes a concern in its own right.
- **Epidemiology**: Prevalence 4%. Gradual onset.
- **Secondary morbidity**: As above.

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**Panic disorder**

Panic is defined as:

- A sudden intense rush of anxiety that occurs unexpectedly and is associated with intense awareness of bodily sensations e.g. palpitations, numbness, breathlessness, dizziness, nausea.

There must be no physical explanation for this.

There is a perceived threat, which is triggered by a stimulus e.g. breathlessness, this then causes anxiety induced bodily sensations which are interpreted in a catastrophic fashion. This reinforces the perceived threat.

Panic disorder is thought to be due to a tendency to interpret autonomic events in a ‘catastrophic fashion’.

This can be demonstrated experimentally by the modified Stroop test (the colour of words such as death, heart attack etc. is recognised more slowly) or contextual priming (patients are quicker to complete sentences with words that have a catastrophic implication).

Safety behaviours are what the patient does when they feel panicked e.g. feeling dizzy and lying down to prevent collapsing. This allows the continuation of the patient’s belief that a catastrophe will happen despite the fact that it repeatedly does not i.e. the safety behaviour stopped it.
Management
Drug treatment:
- Drug treatment is adjunctive to proper psychological management. Placebo and symbolic effects of tablets are powerful and can become safety behaviours.
- Benzodiazepines – safe, specific, fast acting, few unwanted effects, agonists to specific regulatory site on GABA receptor. Use ones with longer half-life. These cause dependence so would not be used long term. They reduce anxiety and aggression, sedate helping insomnia, relax muscles and suppress convulsions.
- Beta-blockers - treatment of autonomic symptoms to prevent tremor, palpitations, sweating. These have no CNS effect.
- Long term treatment – SSRIs. These are slower onset but effectively treat OCD, panic and PTSD.

CBT:
- Probably best treatment for all anxiety related conditions.
- Identify stereotypy by analysis of fears, beliefs and behaviours when anxious. A stereotypy is a recurring constellation of fears, beliefs and behaviours that makes sense to the patient. The behavioural element serves to prevent disconfirmation of the belief.
- Enable patient to understand stereotypy.
- Allow dismantling by encouraging risk taking – graded exposure leads to desensitisation.

Obsessive-compulsive disorder
This is also an anxiety disorder.
- Emotion – anxiety about obsession.
- Cognition – preoccupation with obsession.
- Behaviour – compulsions.
- Bodily symptoms – tension, especially if prevented from compulsive act.

Perpetuated by avoidance and performing compulsions.
Prevalence is equal in men and women.

This is treated by CBT and clomipramine.
Depression

Depression is common with a prevalence of about 5% - about 10% of these are seen by a psychiatrist. Depression is not all or nothing – there is a continuum from mood lowering, to abnormal mood lowering, to abnormal mood lowering with loss of function.

Diagnosis

Core features are depressed mood (typically worse in morning), anhedonia, self-reproach and reduced energy and activity. However, there is much individual variation. Atypical presentations include anxiety, irritability and motor agitation.

Diagnosis requires 2 core features and an additional symptom. Examples of other possible symptoms:

- Biological symptoms – diminished appetite, disturbed sleep especially early morning waking, loss of libido. Patients with these symptoms are more likely to respond to drugs.
- Cognitive – loss of confidence, thoughts of suicide, impaired attention and concentration, cognitive slowing (depressive pseudodementia), inappropriate guilt,
- Behavioural – psychomotor retardation or agitation, self-neglect, neglect of jobs, social withdrawal, decreased functioning, at worst depressive stupor.

Psychotic depression also involves:

- Delusions – mood congruent, nihilistic (Cotard’s syndrome – believing they, or part of them, is dead).
- Hallucinations – 2nd person auditory (defamatory, accusatory), olfactory.

Depression can be classified as:

- Mild (often mixed with anxiety), moderate or severe.
- With or without somatic symptoms (mild or moderate depression).
- With or without psychotic symptoms (severe depression).
- Recurrent or single episode.

Aetiology

Multifactorial genetic susceptibility. Environmental factors interact.

Biochemical: reduced 5HT and NA (monoamine hypothesis).

Psychological: cognitive e.g. arbitrary inference, selective abstraction (focussing on specifics), overgeneralisation, persification; psychoanalytic theory (loss and bereavement lead to inward turning of aggression), learned helplessness. Generally, these are regarded as being secondary to low mood rather than causal.

In mild depression, the environment is the most significant. If it is severe, genes play a more major role.

Environmental factors may be:

- Predisposing childhood factors – sexual abuse, parental loss, maternal neglect.
- Precipitating adverse life effects – bereavement, divorce, unemployment.
- Vulnerability factors – lack of confiding relationships, early loss of mother.
Management
Biological: antidepressants, augmentation with lithium (maybe also pindolol, T3, L-tryptophan).
Physical: ECT.
Psychological: counselling, self-help groups, CBT (efficacy is as good as antidepressants in mild and moderate depression and lower rate of relapse), psychoanalytical psychotherapy.
Social: occupational therapy, day hospital, home help, social skills training.

Other affective disorders
Dysthymia – long-standing low mood of insufficient severity for depression. Lack of pleasure in life.
Sometimes thought of a personality trait.
Cyclothymia – like mild chronic bipolar disorder.
Mania and Hypomania

Mania involves:
- Behavioural – increased quantity and speed of activity, decreased need for sleep, talkative (pressure of speech), over familiar, risk taking.
- Cognitive – increased speed of mental activity, difficulty concentrating, distractible, perceptual distortions, flight of ideas, persecutory or grandiose ideation or delusions, mood congruent hallucinations.

ICD-10 recognises three degrees of severity of manic episodes:
- Hypomania - persistent mild elevation of mood with increased activity and difficulty concentrating. Symptoms do not lead to disruption of daily functioning.
- Mania without psychotic symptoms – elevated mood with markedly increased energy, flight of ideas and changed behaviour, lack of insight. Disrupts work and social activities almost completely.
- Mania with psychotic symptoms – as above with grandiose or persecutory delusions and mood congruent 2nd person auditory hallucinations.

Bipolar affective disorder
Characterised by repeated episodes of affective disorder some of which are depression and others hypomania or mania. Recovery is usually complete between episodes. About 90% of patients who have a single manic episode go on to have future episodes. About 50% of patients will have a recurrence of mania within 5 months when they stop mood stabiliser. Manic episodes tend to begin abruptly and last about 3 weeks (with treatment), depression usually lasts longer (3-6 months).
Rapid cycling is when there are >4 episodes per year.
In general, everyone who has mania becomes depressed at some point so will have bipolar disorder. Generally, depression gets more common as disease progresses.

Lifetime risk is about 1%.
This has strong heritability – risk to 1st degree relatives is about 12%.

Management
Primarily pharmacological.
Acute mania – antipsychotics, benzodiazepines.
Prophylaxis – mood stabiliser.
Beware of using antidepressants as can precipitate acute mania.
Personality Development and Disorders

Personality:
Enduring pattern of perceiving, relating to, and thinking about the environment and oneself that is exhibited in a wide range of important social and personal contexts and varies between individuals. Interaction between character and temperament.

Character:
Generally taken to mean those personality traits shaped by developmental processes and life experiences.

Temperament:
Usually refers to biologically based, genetically determined, dispositions that combined with life experiences shape the personality.

Personality disorders are defined in both ICD-10 and DSM IV by concepts:
- Traits which are excessive - significantly different from the way the average individual feels and relates to others within a given culture i.e. statistical criteria.
- Traits which are dysfunctional - lead to social maladaptation, impairment or distress.
- Inflexible and pervasive (broad range of situations).
- Deeply ingrained and stable.

Pathological personality traits are those which prevent a flexible response to the environment, foster vicious circles, lack resilience under stress or cause distress to person or society. Personality disorders are not considered mental illnesses (axis II in DSM IV). They are essentially developmental conditions which appear in adolescence. Diagnosis is generally made clinically on the basis of long standing traits that cause social impairment or distress. Tend to present due to problems caused by disorder.

Criticisms of the concept of personality disorder:
- Clinical assessment of PD is unreliable.
- Current classifications lack empirical validation.
- Pejorative term used to justify therapeutic neglect.
- One person’s PD is another person’s virtue.

Lifetime prevalence is about 3%, may be considerably higher. Rates are higher in lower socioeconomic groups. Most personality disorders persist over many years. Some burn out (immature PD) e.g. antisocial and borderline PDs show a decrease in impulsive and socially unacceptable behaviours with increasing age, whereas others tend to get worse (mature PD) e.g. eccentric cluster.

Types of personality disorder
Three clusters (DSM IV):
- Cluster A - odd, eccentric, bizarre; paranoid, schizoid and schizotypal personalities.
- Cluster B - emotionally unstable, impulsive, self destructive; antisocial (dissocial), borderline (emotionally unstable), histrionic and narcissistic personalities.
- Cluster C - anxious, isolated, unsure; avoidant (anxious), dependent and obsessive-compulsive (anankastic) personalities.

Aetiology
Genetic contribution in dissocial and schizotypal PD. Environment - psychosocial deprivation and upbringing, especially in dissocial PD.
Psychological object relations theory - personality is shaped by the child's early parental relationships e.g. dependent traits result from parental deprivation, borderline PD from traumatically unstable parenting, narcissistic PD from grossly unempathic parenting.

It is also possible to get organic personality disorders, generally due to frontal lobe damage. These are typically characterised by social disinhibition and abnormalities of emotional expression.

**Management**

Personality disorders are costly to society and cause functional disabilities. A large proportion (70% or more) of criminals, alcoholics, self-harmers and drug abusers have personality disorders. Many people with psychiatric illness have co-morbid PD, which affect the course and prognosis of the disease. PDs may make a person more vulnerable to axis 1 disorders.

It is very difficult to treat personality disorders so aim to adapt environment e.g. by finding lifestyle that suits. Avoiding substance misuse is important as this exacerbates most traits. Drug treatments are relatively ineffective. Mood stabilisers, antipsychotics and antidepressants may be useful in treating symptoms. Possible methods are CBT, psychotherapy, group therapy. Crisis intervention strategies may be needed – it is important to avoid an inconsistent or excessive reaction, admission may reinforce behaviour.
<table>
<thead>
<tr>
<th>Disorder</th>
<th>Attitude to self/others</th>
<th>Behaviour</th>
<th>Cognition and emotion</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dissocial</strong></td>
<td>Unconcern for feelings. Blame others. Failure to sustain relationships.</td>
<td>Disregards social norms. Irresponsible, impulsive and aggressive.</td>
<td>Incapacity to learn from experience including punishment.</td>
</tr>
<tr>
<td><strong>Borderline</strong></td>
<td>Disturbed self image. Intense unstable relationships.</td>
<td>Excessive efforts to avoid abandonment. Recurrent self harm. Impulsive.</td>
<td>Emotionally unstable. Transient stress-related paranoid ideation or dissociation.</td>
</tr>
<tr>
<td><strong>Histrionic</strong></td>
<td>Wants to be centre of attention. Concern with physical attractiveness.</td>
<td>Dramatisation, exaggerated expressions of emotion. Seductive or provocative.</td>
<td>Shallow rapidly shifting emotions. Suggestible.</td>
</tr>
<tr>
<td><strong>Narcissistic</strong></td>
<td>Grandiose sense of self importance. Lacks empathy.</td>
<td>Exploitative.</td>
<td>Fantasies of success, power, beauty etc. Unreasonable expectations.</td>
</tr>
<tr>
<td><strong>Paranoid</strong></td>
<td>Suspicious. Self-referential.</td>
<td>Unforgiving, combative and sensitive to slights.</td>
<td>Distort experiences and misinterpret actions as hostile.</td>
</tr>
<tr>
<td><strong>Schizoid</strong></td>
<td>Preference for solitary activity. Indifference.</td>
<td>Cold and detached. Lack of interest or pleasure.</td>
<td>Preoccupation with fantasy and introspection.</td>
</tr>
<tr>
<td><strong>Avoidant</strong></td>
<td>Social avoidance. Views self as inept and inferior.</td>
<td>Restricted lifestyle, won’t take risks.</td>
<td>Fear and anxiety.</td>
</tr>
<tr>
<td><strong>Dependent</strong></td>
<td>Uncomfortable and helpless alone. Encourages others to take responsibility.</td>
<td>Excessively compliant.</td>
<td>Limited capacity to make decisions alone. Fears of abandonment.</td>
</tr>
</tbody>
</table>
Somatisation

The sick role:
- The person is relieved from responsibilities and has the right to expect help.
- In turn they have an obligation to seek and cooperate with treatment.

Somatoform disorders:
- When patients seek medical attention for somatic symptoms or health concerns that are not explained by, or are disproportionate to, the medical findings.
- Must have persisted for >6 months.
- Somatisation is a preoccupation with somatic symptoms.
- Hypochondriasis is an irrational fear or belief that one has a serious medical condition.

These must be differentiated from factitious disorders and malingering. In somatoform disorders, symptoms are outside of voluntary control, whereas in factitious disorders and malingering they are deliberately produced.

Briquet’s Syndrome
This is a severe chronic form of somatisation disorder.
Patients present repetitively with multiple, frequently changing, unexplained physical symptoms. This must go on for at least 2 years and be associated with a persistent refusal to accept reassurance and a degree of functional impairment.
This may account for over 10% of consultations.
It is associated with personality disorders, affective disorders and childhood deprivation and abuse.
This behaviour may be reinforced by:
- Primary gain – attention and sympathy from physician.
- Secondary gain – e.g. avoid responsibilities.

Prognosis is poor.

Hypochondriasis
This is a preoccupation with having a specific serious disease, despite adequate medical evaluation and reassurance.
This is often secondary to an affective disorder.
Repeated reassurance may perpetuate the problem. Management should be to stop investigations and treat other psychiatric disorders.

A specific from of this is dysmorphophobia – patient is preoccupied with an imagined defect in their appearance which causes distress and impairment of functioning. This is a type of overvalued idea.

Dissociative/Conversion disorder
This is an abnormal illness behaviour characterised by sudden onset loss of function in a non-pathological distribution, which is not intentionally produced e.g. sudden muteness, amnesia or paralysis when under stress.
The individual appears unconcerned with the loss of function and tends to recover when the stress is removed.
Psychodynamic theories for this disorder:
- Conversion of psychological stress e.g. 1st world war paralysis
- Gain – perpetuates.
**Management of somatisation disorders**
Stop investigations.
Acknowledge the reality of the patient’s problem and offer patient an alternative way of conceptualising problem.
Treat with psychotherapy and or medication.
Systematically identify perpetuating factors e.g. disordered physiology, misinterpretation of bodily sensations, abnormalities of mood, unhelpful coping behaviour and social stressors.

**Factitious disorder (Munchausen’s disorder)**
This is the deliberate manufacture of somatic symptoms or physical signs.
This is driven by a desire to assume the sick role for primary gains and attention.
The person usually has a personality disorder.
Common presentations include - dermatitis artefacta, haematemesis.

In factitious disorder by proxy the person aims to get attention via dependents i.e. they inflict signs of illness on other people, usually children.
They then seek medical help for the person for primary gain.
These people have severe personality disorders.

Management involves preventing iatrogenic harm, protecting the child if by proxy, confronting at an early stage and trying to manage the patient rather than treating the illness.

**Malingering**
This is manufacture of symptoms for an obvious goal i.e. secondary gains.
Learning Disabilities

A learning disability involves:
- Significantly sub-average intellectual functioning i.e. an I.Q. below 70 (2SD below normal) on an individually administered IQ test.
- Impairment across a wide range of functions and concurrent deficits in adaptive behaviour, taking into account the person's age.
- Onset of intellectual impairment before the age of 18 years (otherwise is dementia).

Learning disabilities are commonly identified before birth from complications or tests, at birth from appearance or after birth from delayed development.

Epidemiology:
- Mild LD affects about 2% of the general population and make up 85% of cases of LD.
- Moderate and severe LD have a prevalence of 0.5%.
- LD are more common in males.
- LD are more common in lower social classes and are associated with overcrowding, poverty and unskilled irregular employment.

Classification (These form part of axis II)

<table>
<thead>
<tr>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Profound</th>
</tr>
</thead>
<tbody>
<tr>
<td>IQ</td>
<td>50-69</td>
<td>35-49</td>
<td>20-34</td>
</tr>
<tr>
<td>Independence</td>
<td>Independent</td>
<td>Substantial autonomy with supervision</td>
<td>Much help needed, usually continent</td>
</tr>
<tr>
<td>Communication</td>
<td>Good</td>
<td>Normally able to</td>
<td>Limited, often not speech.</td>
</tr>
<tr>
<td>Reading/writing</td>
<td>Some</td>
<td>Basic</td>
<td>No</td>
</tr>
<tr>
<td>Ability to work</td>
<td>Semi-skilled</td>
<td>Unskilled</td>
<td>Supervised basic tasks</td>
</tr>
<tr>
<td>Social skills</td>
<td>Normal</td>
<td>Moderate</td>
<td>Few</td>
</tr>
<tr>
<td>Physical problems</td>
<td>Rare</td>
<td>Sometimes</td>
<td>Often physically disabled</td>
</tr>
</tbody>
</table>

Aetiology

30% have no identifiable cause. Genetic – specific abnormalities (commonest Downs, then fragile X), polygenic inheritance of low intelligence. Environmental - social and educational deprivation.

Pathological – infective, toxic, trauma:
- Antenatal – maternal infections (e.g. rubella, toxoplasmosis), maternal intoxication (e.g. alcohol, lead, drugs), maternal illness (e.g. haemorrhage, toxaemia, hypoxia).
- Perinatal – prematurity predisposes to cerebral palsy, physical injury, birth asphyxia, intraventricular haemorrhage.
- Postnatal – infection (e.g. meningitis), injury, metabolic (e.g. hyperbilirubinaemia, hypoglycaemia), status epilepticus, toxicity.
### Specific genetic causes

<table>
<thead>
<tr>
<th>Genetic basis</th>
<th>Clinical features</th>
<th>Prognosis</th>
<th>Medical associations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Down’s</strong></td>
<td>Trisomy 21, mostly nondysjunction. Prevalence 1 per 660 – higher in older mothers.</td>
<td>Characteristic appearance – oblique palpebral fissures, single transverse palmar crease, hypotonia.</td>
<td>IQ 20-55. Gap to normal widens through childhood. Most survive to middle age then decline due to Alzheimer’s. 50% CHD, especially VSD. Hypothyroidism. Alzheimer’s.</td>
</tr>
<tr>
<td><strong>Patau’s</strong></td>
<td>Trisomy 13.</td>
<td>Cleft palate, polydactyly, cardiac and eye defects.</td>
<td>Almost all die by age 3.</td>
</tr>
<tr>
<td><strong>Edward’s</strong></td>
<td>Trisomy 18. More common in females.</td>
<td>Small chin, cardiac abnormalities.</td>
<td>Half die by 2 months. Severe LD.</td>
</tr>
<tr>
<td><strong>Prader-Willi</strong></td>
<td>Lack of paternal 15q.</td>
<td>Obesity, hypogonadism, hypotonia, violent temper, sleep apnoea.</td>
<td>Mild LD. Expressive language disorder.</td>
</tr>
<tr>
<td><strong>Angelman’s</strong></td>
<td>Lack of maternal 15q.</td>
<td>Microcephaly, ataxic jerky movements.</td>
<td>Severe LD. Epilepsy.</td>
</tr>
<tr>
<td><strong>Cri du Chat</strong></td>
<td>5p.</td>
<td>Round flat face, hypertelorism, micrognathia, characteristic cry.</td>
<td>Severe LD.</td>
</tr>
<tr>
<td><strong>XYY</strong></td>
<td>XXY</td>
<td>Assertive, impulsive.</td>
<td>May have LD.</td>
</tr>
<tr>
<td><strong>Neurofibromatosis</strong></td>
<td>AD</td>
<td>Neurofibromata.</td>
<td>10% severe LD. Sarcomatous change.</td>
</tr>
<tr>
<td><strong>Apert</strong></td>
<td>AD, mainly sporadic.</td>
<td>High mortality in 1st year. Learning disabilities.</td>
<td></td>
</tr>
<tr>
<td><strong>PKU</strong></td>
<td>AR Phenylalanine hydroxylase def.</td>
<td>Microcephalic mental impairment. Autism. Epilepsy.</td>
<td></td>
</tr>
<tr>
<td><strong>Rett</strong></td>
<td>X linked dominant.</td>
<td>Fits, agitation, truncal ataxia, depression.</td>
<td>Normal dev for 1 year then lose speech mobility etc.</td>
</tr>
</tbody>
</table>
Programmes to prevent impairments

Screening:
- Universal screening of new-born infants for phenylketonuria, hypothyroidism and cystic fibrosis.
- Screening healthy adult populations.
- Antenatal screening for Down's syndrome – serum testing for alpha fp, unconjugated oestriol, hCG.
- Postnatal testing.
- DNA screening of selected populations at risk.

Prevention:
- Rhesus immunisation.
- Prevent communicable diseases e.g. rubella, CMV.
- Dietary advice and education.
- Accident prevention.

Epilepsy in learning disability

Epilepsy is more common in LDs – nearly 50% of people with severe LD have epilepsy. This is a problem as epilepsy increases the rate of behavioural problems and compliance with anticonvulsants is poor.

Autism

Normal early development:
- 3 months - babble 'conversation' with mother, demand for social interaction
- 2nd year - development of speech, imagination with pretend games

This development is impaired in autism.

Autism involves:
- Impaired social interactions – aloof, passive, no differentiation between friend and stranger, inappropriate eye contact, no relationships, can’t empathise or interpret behaviour
- Impaired communication – limited speech, repetition, difficulty understanding speech and non-verbal communication.
- Restricted repetitive behaviours and interests – repetitive play, no symbolic play, stereotyped behaviour e.g. rocking, need routine.
- Motor abnormalities – rocking, arranging objects in line, repetitive questions, imitating movements, clumsy.

Autism is thought of as a spectrum:
- Kanner’s Autism – severe, little interaction, limited speech (none in 50%), LD in over 2/3s.
- Asperger’s Syndrome – impairment in social interaction with restrict stereotyped behaviours, but no significant abnormalities in language acquisition or cognitive development.

Management:
- Sufficient personal space
- Organised routines
- Appropriate content of activities and physical activities.
- Treatment of other problems especially epilepsy.
- Obsessions – graded change, aim to reduce frequency and restrict to particularly places.
- Set limits – interrupt some behaviours.
• Counsel parents.
• Medication – not very useful, antipsychotics may reduce excitability.

This is a life-long disorder. There is normal life expectancy and some improvement with age. Prognosis is better if early speech and higher intelligence.

**Learning disability and mental illness**

Risk of mental illness is 3 times higher in people with LD than in the general population. This may be due to:

- Physical associations – epilepsy, chronic physical illness, sensory impairment, drug side effects.
- Psychological reasons – poor communication, limited coping, low self-esteem.
- Social reasons – labeling, loss and grief.

Symptoms of psychiatric illness are modified by learning disability – may lack verbal fluency to accurately describe symptoms:

- Depression – may present as diurnal mood variation, wandering due to agitation, behavioural symptoms.
- Mania – may present as challenging behaviour, overactivity, excitement, inappropriate sexual behaviours.
- Schizophrenia – difficult to diagnose but more common, loss of functioning, less elaborate delusions, simple repetitive hallucinations, fear and withdrawal.

**Challenging behaviour**

This is defined as behaviours which make it difficult for the patient to receive care e.g. aggression or withdrawal behaviours.

They may be due to mental illness e.g. depression or hypomania, side effects of medication, physical illness, social or environmental factors.

Management is by treating the cause and functional analysis of the behaviour.
Eating Disorders

Anorexia nervosa

Classification:
- Intentional weight loss leading to a body weight of at least 15% below that expected.
- Self-perception of being fat and intrusive dread of fatness.
- Endocrine disorder leading to amenorrhea.

Typically affects young women, prevalence about 1%.

Aetiology:
- Psychosocial pressures – ‘thinner is better’.
- Changes in body shape through puberty.
- Genetics – about 70% hereditability, COMT gene.
- Personality – negative emotionality, high stress reactivity, harm avoidance, obsessionality, low self esteem, social inhibition.
- Childhood risk factors – exposure to risk factors shared with other psychiatric disorders e.g. abuse, death of relatives, family disharmony.

Clinical features:
- Physical features tend to be obvious but psychopathology may be denied.
  - Mental state – preoccupation with weight, disturbed body image, fear of loss of control.
  - Eating behaviours – calorie counting, food restriction.
  - Deliberate weight loss e.g. self-induced vomiting, excessive exercise.
  - Amenorrhea.

Physical complications:
- GI – dental erosion, acute gastric dilatation.
- CVS – bradycardia, hypotension, arrhythmias.
- Metabolic – hypokalaemia, metabolic alkalosis, refeeding oedema, hypoglycaemia, hypothermia, renal failure.
- Osteoporosis.
- Endocrine – amenorrhea.

Prognosis - long duration predicts poor outcome:
- May become bulimia.
- 20% mortality from complications of starvation or suicide.
- 20% good outcome.
- 60% intermediate.

Management:
- Screening investigations - FBC, U&E, Glucose, LFTs, serum calcium, magnesium, phosphate, TFTs, B12/Folate, serum proteins, ECG.
- Engagement is most important.
- Co-morbid PD, especially borderline, may make management difficult.
- Refer to specialist psychiatric services, admission may be needed.
- Aim to restoring sustainable weight, separate weight from underlying issues, initiate therapeutic work on underlying issues.
- Therapy psychodynamic psychotherapy or CBT for adults, family therapy for younger
**Bulimia nervosa**

**Classification:**
- Recurrent episodes of binging with loss of control.
- Preoccupation with craving.
- Self-induced vomiting, purging, laxative abuse or alternating periods of starvation to counteract binges.
- Self-perception of being too fat.

Typically young females, prevalence is about 4%.

**Aetiology:**
- Similar to anorexia.
- Genetics are less important.
- Childhood risk factors also include premorbid dieting or obesity and critical comments from family about weight, eating or body shape.

**Clinical features:**
May only be apparent from history – behaviours are usually carried out in private.
May be normal weight.

Physical complications: hypokalaemia, dental erosion, parotid enlargement, oesophagitis, Russell's sign (callouses on dorsum of hand).

**Prognosis - 10% poor, 50% good.**

**Management:**
- Screening investigations as above.
- Behaviours may lead to social withdrawal and be hard to talk about.
- This is usually treated as outpatient.
- Pharmacological – high dose SSRI for comorbidly depressed. Fluoxetine is useful, but only whilst being taken.
- Psychological treatments – education, support, CBT, interpersonal psychotherapy.
**Substance Abuse**

**Definition of abuse**
Abuse is culture specific, so best defined as use which is socially disapproved of. There is usually a concept of excess use, or use which is harmful to the individual or society.

Types of abuse include at risk consumption, harmful use, dependence, intoxication.

Drugs of abuse may cause reward by:
- Increasing dopamine function in meso-limbic and cortical areas - stimulants.
- Indirectly increasing dopamine - opiates, alcohol, nicotine.
- Altering serotonergic function - ecstasy, LSD.

Generally, the drugs which increase DA function are associated with dependence and withdrawal. Those which increase serotonin have potential long term neurotoxicity and behavioural impairment.

**Tolerance** – when an individual requires more of a drug to achieve the same effect:
- Tissue – e.g. down regulation of receptors, increased rigidity of cell membrane in chronic alcohol use.
- Metabolic – speed at which the substance is eliminated e.g. induction of hepatic enzymes.
- Psychological – adaptation to the effects, independently of physiological tolerance. This may lead to overdose.

Dependence occurs when the withdrawal of the drug causes a reaction. This may be:
- Physical e.g. sweating, tremor.
- Psychological – i.e. craving.

Dependence syndrome is defined in ICD 10 as having 3 from a cluster of behavioural, cognitive and physiological phenomena:
- A strong desire or sense or compulsion to take the substance.
- Difficulties in controlling use e.g. amount, onset, termination.
- Physical withdrawal state.
- Tolerance.
- Preoccupation with the substance – progressive neglect of other interests.
- Persistence with substance despite detrimental social, cognitive and physical effects.

Neither tolerance nor dependence are essential for, or enough in themselves, to diagnose substance abuse.

Drugs which tend to cause dependence have:
- Positive reinforcement – taking the drug activates dopamine meso-limbic neurones and has a rewarding effect.
- Negative reinforcement – cessation of the drug has a negative effect due to habituation e.g. physical abstinence syndrome or craving.

Drugs with a strong dependence liability are:
- Narcotic analgesics- morphine and heroin.
- General CNS depressants – ethanol, barbiturates.
- Psychomotor stimulants – amphetamines, cocaine, nicotine.
Addiction is a non-specific term, which incorporates tolerance and dependence.

**Aetiology of drug abuse**
Biochemical - mesolimbic dopamine activity causes reward.
Psychological - learning theory.
Personality - antisocial PD
Social influences e.g. peer group.
Genetic – some familial predisposition.

**Alcohol abuse**

Pharmacology:
- Increases fluidity of cell membranes.
- Direct effect on GABA receptor.

Tolerance:
- Tissue – cell membranes become more rigid.
- Metabolic - induced liver enzymes.
- Psychological – get less effect so increased risk of O.D.

Complications of alcohol use:
- Acute intoxication – impulsivity, disinhibition, impaired motor skills.
- Withdrawal.
- Social (may be caused by alcohol or due to confounder e.g. PD) – divorce, violence, unemployment, absenteeism, crime, accidents.
- Physical – neurological, hepatic, GI, anaemia, neoplasms.
- Psychiatric - dysphoric mood (may be caused by chronic drinking or due to self medication for an underlying depressive illness), alcoholic hallucinosis (auditory hallucinations in clear consciousness), pathological jealousy (Othello's Syndrome, delusions of infidelity).

Safe levels of alcohol are considered to be 21 units per week for men and 14 for women.

Neurological complications:
- Wernicke's encephalopathy occurs in a proportion of thiamine deficient alcoholics. It involves confusion, memory deficits, ataxia, lateral rectus palsy. There are degenerative changes in mammillary bodies, hyothalamus, thalamus and cerebellum.
- It progresses to Korsakoff's syndrome. This is an amnesic syndrome involving loss of short term working memory and confabulation.
- Other problems include peripheral neuropathy, cerebellar degeneration, dementia.

Alcohol withdrawal:
- Increased sympathetic tone – tachycardia, hypertension, sweating, tremor.
- Lowered seizure threshold.
- Delirium tremens is the extreme end of the withdrawal syndrome. It occurs in about 5% of people and involves clouding of conciousness, visual hallucinations and fear. It has a significant mortality.
Management:

- Establish treatment goals – wish to reduce drinking, controlled drinking, abstinence.
- Medical aspects.
- Treat withdrawal symptoms – delirium tremens requires admission, rehydration and vitamins. Give reducing doses of benzodiazepines – these reduce withdrawal symptoms due to cross tolerance and thiamine.
- Vitamin replacement.
- Anti-craving drugs - acamprosate (GABA agonist), SSRIs
- Response inhibition – disulfiram (leads to accumulation of acetaldehyde), Ca carbimide.

**Anxiolytics**

The main anxiolytic drugs that can be abused are benzodiazepines. These cause sedation, amnesia, psychomotor impairment, hypnosis, anxiolysis.
Tolerance is rapid and there is a withdrawal reaction due to hyperexcitability of GABA complex. This can involve anxiety, distortion of perception and confusional states.

**Opiates**

E.g. heroin, methadone.
These are very addictive and overdose is common due to tolerance and batch to batch variation. There is a high relapse rate after withdrawal – over 50%, so a harm reduction approach may be more appropriate.

Abstinence causes anxiety, depression, irritability, drug craving, lacrimation, rhinorrhea, yawning, sweating, restless sleep, anorexia, nausea, diarrhoea, tremor and goose-flesh.

Overdose causes respiratory depression. This can be antagonised by naloxone. Other adverse effects are due to the effects of adulterants e.g. quinine and infections.

**Amphetamines**

These give increased drive, confidence, sociability and physical and mental capacity. Later they cause lethargy and depression. High doses cause an increase in body temperature, hallucinations, paranoia and violence.
Chronic use causes amphetamine psychosis.
Ecstasy is a type of amphetamine. This causes euphoria, feelings of intimacy and heightened experiences due to release of serotonin. Acute toxicity is due to hyperthermia, vascular problems and blood coagulation. Sudden death can occur due to a condition like heat-stroke with muscle damage, renal failure and inappropriate secretion of ADH leading to dehydration.

**Cocaine**

High doses cause overactivity of the SNS due to uptake blockade and therefore hypertension, tachycardia, hyperpyrexia, dilated pupils and palpitations. There is no tolerance or physical dependence but it causes psychological dependence. Smoking leads to very rapid dependence.

**Hallucinogens**

Phenycyclidine (PCP) acutely causes delusions, paranoia, disordered thinking and illusions, similar to schizophrenia. Chronically, it causes cognitive impairment, depression and weight loss.
Lysergic acid diethylamide (LSD) is a 5HT2 agonist. Acutely it causes mood swings, delusions, panic, increased BP and heart rate and flashbacks.
These tend not to cause dependence or withdrawal. Flash backs and panic due to bad trip are the main reasons for admission.
General management of substance abuse

Assessment:
- History - detailed consumption, cues, recognised pros and cons, support, environment.
- Collateral history
- Biochemistry e.g. bloods, urine, breathalyser
- Standardised questionnaires

Establish goals

Biological:
- Ease withdrawal or facilitate abstinence
- Treat medical/psychiatric complications

Psychological:
- Motivational interviewing
- Cue exposure to reduce craving

Social:
- Manipulation of environment, including rehabilitation.
- Self-help agencies
Suicide and Deliberate Self Harm

Suicide is a wilful, self-inflicted, life threatening act which has resulted in death.
Suicide accounts for about 1% of all deaths and is the 3rd cause of death in the 15-34 age group.
Suicide attempts are higher in women, but completed suicides are higher in men.
Rates are higher in the unemployed, low social classes and those living alone.

Aetiology of suicide

90% of suicides have some form of psychiatric illness (70% depression, 15% alcoholism).
Psychosocial factors – imitation, adverse life events, lower social, unemployment.
Biological – serotonin deficiency, genetic predisposition – no conclusive data.

Depression:
- 10-15% of depressed patients kill themselves, generally early in the illness
- Specific risk factors include previous history of suicidal ideation, agitation, insomnia, hopelessness, impaired memory and self neglect.

Alcoholism:
- 15% of alcoholics kill themselves, generally late in illness.
- This especially happens if co-morbid depression present.
- Specific risk factors – poor physical health, heavy drinking, unemployment, little or no social support, psychiatric co-morbidity, prior threats of suicide.

Schizophrenia:
- 10% of schizophrenic patients kill themselves, generally early in the illness.
- Specific risk factors - young male with chronic relapsing illness, high pre-morbid function and education, depression, suicidal ideation, akathisia.

Other disorders:
- Neurotic disorders – increased rates in panic disorder and PTSD (especially if guilt), low in OCD.
- Personality disorders – especially sociopathic and borderline.

Other risk factors:
- Physical illness and suicide – high rates in AIDS, multiple sclerosis, epilepsy, peptic ulcer, cancer and head injury. Risk of suicide in general hospital inpatients is 3-4 times that in the general population.
- Prison populations - risk of suicide is 4-5 times higher than in general population. 50% of suicides occur in first 3 months of imprisonment, 90% hang themselves and 33% have a psychiatric history.

Acronym (SUICIDAL – sex, stressful life events, significant others, unsuccessful attempts, unemployment, chronic illness, depression, age, alcohol, availability, lethality).

Common methods of suicide are hanging and gas inhalation (especially males) and poisoning (especially females).
Management of suicide
Identify people at high risk of attempting and completing suicide. Risks for completed suicide include:
- Stable – male, unemployed, isolated, mental or physical illness, drugs, alcohol.
- Dynamic – current mental state.
- Suicide act – planned, final acts, secrecy.

Diagnose and treat psychiatric illness.

Deliberate Self Harm
This is a deliberate, non-fatal act, done in the knowledge that it was potentially harmful.
10% patients with DSH ultimately commit suicide.
50% of suicides have a history of DSH.

Epidemiology:
- Majority of patients are under 35yrs.
- Commonest in females.
- Divorced>single>widowed>married.
- Less than a third have definite psychiatric illness, but most have psychological distress.
- 65% have major life event.

Factors indicating suicidal intent in DSH – isolation, precautions to avoid intervention, note, final acts, violent methods, perceived lethality.

## Bereavement and stress reactions

<table>
<thead>
<tr>
<th>Stress</th>
<th>Acute stress reaction</th>
<th>Adjustment disorder</th>
<th>PTSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Timing</td>
<td>Traumatic</td>
<td>Significant life change</td>
<td>Traumatic</td>
</tr>
<tr>
<td>Emotion</td>
<td>Dazed</td>
<td>Depression, anxiety</td>
<td>Anxiety, irritability, numb</td>
</tr>
<tr>
<td>Cognition</td>
<td>Amnesia or denial</td>
<td>Preoccupation with event</td>
<td>Repeated reliving</td>
</tr>
<tr>
<td>Behaviour</td>
<td>Overactivity or withdrawal</td>
<td>Angry outbursts, disturbed</td>
<td>Avoidance</td>
</tr>
<tr>
<td>Somatic symptoms</td>
<td>Many</td>
<td>Moderate</td>
<td>Startle response exaggerated.</td>
</tr>
<tr>
<td>Management</td>
<td>Individual vulnerability is important.</td>
<td>Reassurance, antidepressants, proportion develop psychiatric illness.</td>
<td></td>
</tr>
</tbody>
</table>
Bereavement
Grief and bereavement are normal experiences.
Bereavement – objective state of having experienced a loss.
Grief – subjective state of experiencing the psychological and physiological reaction to a loss.
Mourning – psychological process of working through a loss.
Complicated grief – failure to return to a pre-loss level of performance or state of emotional wellbeing.

Symptoms – low mood, pining, somatic symptoms e.g. pain, motor symptoms e.g. restlessness, perceptual disturbances e.g. seeing loved one, hostility.
This differs from depression in that the person blames other people rather than themself, has less general guilt and more physical symptoms. Symptoms are fluctuating rather than pervasive.

Phases of the grief reaction:
• Shock, disbelief and denial
• Acute mourning – e.g. weeping, poor sleep, appetite and concentration, guilt
• Restitution

Caring for the bereaved:
• Comfort and consolation.
• Permission to grieve, or stop grieving.
• Help prevent maladaptive means of coping.
• Facilitate restitution.

Grief counselling is useful to increase reality, facilitate grieving and overcome barriers. It focuses on the bereaved and their emotions rather than longer term issues.
Early counselling is to give permission to grieve and reassurance about symptoms.
Later it gives permission to stop grieving and manage intrusive thoughts and images.

Pathological outcomes of bereavement include general ill health, increased mortality, psychosomatic disorder, psychiatric/social problems, altered relationship patterns, vulnerability to loss and increased use of alcohol and prescription drugs.
These outcomes are affected by the relationship with the deceased, type of death, response of family, concurrent stressors, previous losses and socio-demographic factors.

Pathological grief reactions
Factors likely to cause pathological grieving include:
• Circumstances - sudden deaths, blame of survivor.
• Relationship – young children, parents, dependent relationship.
• Personal factors – insecure, other losses.
• Social factors – support, philosophical framework, caring for dependents.
• Pre-existing mental illness.

Pathological grief reactions are defined by timing (more than 1 year, longer for parents of dead child), symptoms and functional impairment. This includes delayed, distorted (out of proportion) and chronic grief. The main treatment is guided or forced mourning. The patient is encouraged to reminisce about the lost person and acknowledge the loss.
Child and Adolescent Psychiatry

This generally presents to GPs or paediatric clinics as behavioural or emotional disorders. The prevalence is probably about 10%, with only about 10% of these seeing psychiatrists.

Aetiology
Biological – genetic, neurological disorder, physical health.
Psychological - IQ, developmental delays, temperament, self-esteem.
Social - family factors, school, abuse, housing.

Clinical syndromes in child psychiatry
Disorders also in adults - anorexia, mood disorders, OCD, schizophrenia, PTSD, self-harm.
Disorders in early childhood – behaviour problems, disorders of elimination, developmental disorders, sleeping difficulties.
Disorders in middle childhood – conduct disorder, hyperkinetic disorder, emotional disorder, tics, Tourette's syndrome.

Behaviour problems
Oppositional defiant disorder:
• Epidemiology – prevalence up to 15%, more common in boys.
• Children are markedly defiant, disruptive and have disobedient behaviour.
• Management – parent training and support, behaviour modification, social support.
• Outcome is better with early intervention but many progress to a conduct disorder.

Disorders of elimination
Enuresis:
• This is involuntary bladder emptying after the age of 5.
• Can be primary, secondary (continence has been achieved, more likely to be related to stress) or nocturnal.
• Aetiology – family history, developmental delay, UTIs, stressful events, deprivation.
• Often co-morbidity with emotional or conduct disorder.
• Manage by excluding physical cause, treating associated psychiatric problems and behavioural methods e.g. restricting fluids, star charts, enuresis alarm. In the short term desmopressin and low dose tricyclics can be used.
• Most children stop wetting before adolescence – about 1% progress to adults.

Encopresis:
• This is the passage of faeces in inappropriate places after age 4.
• Aetiology – more common in boys, physical cause e.g. Hirschsprung’s disease, constipation; symptom of wider psychological disturbance, failed toilet training.
• Management – educate parents and child, treat constipation and psychological problems, behavioural methods.
**Sleep disorders**

Night waking
Night terrors
Nightmares

Generally managed in primary care.

**Conduct disorder**

This has a prevalence of 4-8% and is more common in urban areas and boys. It involves persistent dissocial, aggressive and defiant behaviour. Management involves behaviour modification, parent training, family therapy, support for schools. Medication is only used if there is comorbidity e.g. ADHD. 40% of sufferers are antisocial as adults.

**Hyperkinetic disorder (ADHD)**

This is more common in boys and starts before the age of 7. It involves persistent inattention, overactivity and impulsivity, occurring in more than one setting, which is maladaptive and inconsistent with the child’s developmental level. Management is psychological (behaviour management, parent training, psychiatric comorbidity), and by medication e.g. methyphenidate (stimulant), recently atomoxetine (non-stimulant). Diet may play a role in a minority of cases.

**Emotional disorders**

This includes separation anxiety, phobias, somatic symptoms and school refusal. In older children, this becomes depression and social anxiety. Aetiology includes parental anxiety and family or school stress. Management – psychoeducation, parent training, CBT relaxation training, exposure, response prevention.

**Tics and Tourette’s**

Tics – sudden, repetitive stereotyped movements or utterances (obscenities = coprololia). Tourette’s involves motor and one or more vocal tics, which have been present for at least 1 year. Tics are worsened by anxiety and boredom. Tics are related to obsessional symptoms and linked to OCD. Treat by education, CBT and low dose neuroleptics e.g. risperidone.
**Liason Psychiatry**

People can be referred to liaison psychiatry due to symptoms inadequately explained by organic pathology e.g. somatoform disorders, dissociative or conversion disorders, factitious disorders. In a significant proportion of GI, cardiology and neurology referrals no organic cause is found. In primary care, 20% of all new episodes of physical symptoms have no clear pathology and many of these turn out to have a psychiatric disorder.

Panic disorders can complicate physical disorders and commonly present to A&E due to physical symptoms. Specific phobias e.g. needle phobia can pose particular problems in medical care. Mood disorders are common in inpatients and amplify the disability.

**Psychiatry in Primary Care**

Primary care has a role in:

- Prevention – e.g. bereavement, learning disabilities.
- Early detection and treatment – e.g. postnatal depression.
- Long term management.

Psychiatric problems typically present with being tired all the time, insomnia, tension headache or a physical disease. Problems are referred to secondary care if they are treatment resistant, have an unclear diagnosis, need specialist investigation or need intensive treatment. Some treatments are peculiar to secondary care – ECT, psychotherapies, high doses, CPNs.

**Perinatal Psychiatry**

Suicide is the leading cause of maternal death in United Kingdom. Perinatal psychiatry is concerned with women who develop a psychiatric disorder around pregnancy and women who have a pre-existing illness and get pregnant.

**Illness in pregnancy**

Psychiatric disorder in pregnancy is common, usually mild and likely to improve. The incidence of serious mental illness is markedly reduced in pregnancy. People who are already suffering from a mental illness are not at serious risk during pregnancy. 10-15% develop mild disorders anxiety and depression in 1st trimester. In the 3rd trimester, the incidence of mental illness is lower and this appears to be protective against a recurrence of existing illness. Mild illnesses generally respond to psychosocial interventions. People who have serious conditions should be referred.

**Post natal psychiatric disorders**

PND and 'the blues' are the most common. Psychosis and mania are also important. Everyone is vulnerable after childbirth as it is a physical and emotional challenge, changes role expectations and causes stress. Psychosocial factors are more important in minor conditions, whereas biological factors are more important in serious conditions. People who are more vulnerable – immature, physically ill during pregnancy, genetic inheritance, adversity, positive psychiatric or family history, emergency section, loss of previous child. Risk of admission is increased about 10 fold after childbirth. This is highest in the first 3 months.

The risk of relapse or recurrence after delivery is 1 in 3 for depression or bipolar illness and schizophrenia and about 1 in 2 for severe PND and puerperal psychosis.
The blues typically start 3-10 days after childbirth. They involve emotional lability and tearfulness, anxiety and irritability and exhaustion. This occurs in the majority of women and lasts about 48 hours. It does not require treatment.

Postnatal depression is similar to depression at other times. Incidence is about 10% (severe 3%). Only the incidence of serious depressive illness is increased following childbirth. This may be complicated by anxiety, panic or obsessional symptoms. There are 2 peaks of presentation – 2-4 weeks and 10-14 weeks. Treatment is with antidepressants and counselling. This has a good prognosis but a risk of about 1 in 3 of recurrence with another baby. Untreated PND tends to resolve in 6 months, compared to 2 with treatment. Chronic PND affects the infant's social, emotional and cognitive development.

Puerperal psychosis occurs in 2 in 1000 deliveries and is more likely closer to childbirth (50% in first week). This can be schizoaffective disorder or bipolar disorder. Patients are psychotic and severely disturbed, with hallucinations and delusions. This needs admission. It has a good short term prognosis, but a 1 in 2 risk of recurrence.

**Medication**

Psychological treatment is effective for mild conditions.

Psychotropic medication should be given in the lowest dosage possible divided and only for serious conditions.

There is no rationale for abrupt cessation of medication early in pregnancy. It should be gradually withdrawn.

Conception should be avoided on mood stabilisers. Lithium and benzodiazepines are probably teratogenic. TCAs are probably safe.

Post partum medication should be restarted immediately following delivery.

Breastfeeding should not be carried out on SSRIs, lithium, trazodone, benzos, atypical antipsychotics. Medication in breastfeeding should be given in the lowest dose divided. Avoid feeding at the peak plasma level, take the dose before the infants longest sleep.

**Prevention**

Counsel women with chronic severe mental illness about pregnancy.

For manic depressive illness consider restarting treatment after delivery.

Maintain chronic schizophrenics on medication throughout pregnancy.

For people with a previous history of puerperal psychosis/severe postnatal depression keep close contact 1st few weeks and consider prophylaxis after delivery.

Assess all women at 6 week postnatal check.

**Important Note**

These notes were written by Liz Tatman, as a fourth year medical student in 2006. They are presented in good faith and every effort has been taken to ensure their accuracy. Nevertheless, medical practice changes over time and it is always important to check the information with your clinical teachers and with other reliable sources. Disclaimer: no responsibility can be taken by either the author or publisher for any loss, damage or injury occasioned to any person acting or refraining from action as a result of this information.

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