‘Anxiety’ (Lewis)
1. An emotional state with the subjectively experienced quality of fear
2. An unpleasant emotion which may be accompanied by a feeling of impending death
3. A feeling directed towards the future, perceiving a threat of some kind
4. There may be no recognizable threat or one which, by reasonable standards, is out of proportion to the emotion it seemingly provokes
5. There may be subjective bodily discomfort and manifest bodily disturbance

Classification of fears (Marks, 1971)
A. Normal fear
B. Abnormal fears (phobias)
   Class I  Phobias of external stimuli
   i. agoraphobia
   ii. social phobia
   iii. animal phobia
   iv. miscellaneous specific phobias
   Class II Phobias of internal stimuli
   i. illness phobias
   ii. obsessive phobias

Biological aspects of anxiety

Cardiac function
• higher basal rate
• less deceleration after stress
• more beat-to-beat fluctuation
• increased awareness of heart function

Electrodermal response
• increased skin conductance
• decreased habituation
• more spontaneous fluctuation

Peripheral blood flow
• more vasodilatation
• decreased renal and splanchnic flow

Neurotransmitter abnormalities
• increased circulating adrenaline
• increased circulating noradrenaline
• increased platelet MAO
• increased central NA and 5-HT activity

Responses to sodium lactate infusion
• provokes panic in susceptible patients, compared to controls
• IMIPRAMINE is effective in blocking lactate-induced panic
• panic due to:
  • ? abnormal metabolism
  • ? production of alkalosis
  • ? reduction of ionized calcium
  • ? interacts with hyperactive β-adrenergic receptors

Mitral valve prolapse
• incidence of MVP in patients with panic disorder or agoraphobia of 40-50 %
  (general population 5-20 %) (Parisier et al.)
• evidence does not suggest that MVP causes panic attacks
• MVP and panic may form part of a general syndrome of primary autonomic
dysfunction, or MVP may act as autonomic precipitant interacting with genetic
predisposition to panic disorder

Hyperventilation syndrome
• more common in young women
• if three minutes of overbreathing induces similar symptoms, the diagnosis can be
  made
• physiological effects of reduced $P_{CO_2}$:
  • vasoconstriction of cerebral arteries
  • reduced availability of $O_2$ in oxyhaemoglobin
  • increased irritability of autonomic, sensory, and motor nervous systems
  • bronchoconstriction and tachycardia
  • exaggerated sinus rhythm
  • headache, paraesthesiae, visual disturbance

Behaviour therapies

Systematic desensitization
• gradual exposure to phobic stimulus along hierarchy of increasing intensity until
  patient habituates and avoidance response is extinguished
• includes relaxation training, then practice in fantasy before situational exposure
• good response associated with:
  • more specific phobias
  • good relaxation response
  • patient motivation
  • encouragement and support from therapist
  • positive involvement of relatives
• poor response associated with:
  • presence of free-floating anxiety
  • poor motivation
  • presence of secondary gain
  • severe obsessions

Flooding (implosion)
• fantasy or in vivo
• supervised maximum exposure to feared stimulus until anxiety reduction, or exhaustion
• effective for phobias where free-floating anxiety is prominent

Modelling
• observation of therapist engaging in non-avoidance behaviour with the feared stimulus
• a combination of flooding, associated modelling, and moderate doses of DIAZEPAM given 4 hours before sessions may be particularly effective in agoraphobia
F40 Phobic anxiety disorders

F40.0 Agoraphobia
   .00 without panic disorder
   .01 with panic disorder
F40.1 Social phobia
F40.2 Specific isolated phobias
F40.8 Other phobic anxiety disorders
F40.9 Phobic anxiety disorder, unspecified

Predisposing and associated factors
- passive, anxious, and dependent premorbid personalities
- stable families
- similar to general population in terms of education, social class
- often precipitated by major life events
- history of childhood fears, and enuresis
- higher incidence of sexual problems in female group compared to control population

no association with:
- major depressive disorder
- schizophrenia

Epidemiology
- The ECA study:
  - lifetime prevalence for all phobias = 7.8-23.3%
  - six-month prevalence for agoraphobia = 2.8-5.8%
  - six-month prevalence of simple phobia = 4.5-11.8%
  - six-month prevalence for social phobia = 1.2-2.2%
- The Edmonton Study (Dick et al. 1994):
  - lifetime prevalence for all phobias = 8.9% (12% for females, 6% for males)
  - age of first symptoms 6 in females, 12 in males
  - high rates of comorbidity with depression, alcohol and drug abuse, and obsessive compulsive disorder

phobic anxiety disorders affect females more than males, except social phobia where the sex ratio is equal

The heritability of anxiety disorders
- Kendler et al. 1993
  - panic disorder: 35-40%
  - generalized anxiety disorder: 30%
Mowrer’s two-step conditioning
• explains fear, and phobias
  1. pairing of stimulus with fear (classical conditioning)
  2. reinforcing avoidance by anxiety reduction (operant conditioning)

Eysenck’s theory of the neuroses (1976)
• proposed that certain fears are adaptive
• used Seligman’s concept of ‘preparedness’ to account for the development of
  particular fears

The 3 pathways model (Rachman, 1978)
• there are 3 ways in which fears can develop:
  1. direct conditioning
  2. observational learning
  3. informational learning
Agoraphobia (F40.0)

Clinical features
1. the psychological or autonomic symptoms must be primarily manifestations of anxiety and not secondary to other symptoms, such as delusions or obsessional thoughts
2. the anxiety must be restricted to (or occur mainly in) at least two of the following situations:
   - crowds
   - public places
   - travelling away from home
   - travelling alone
3. avoidance of the phobic situation must be, or have been, a prominent feature
   - the most common themes are distance from home, overcrowding, and confinement
   - panic attacks and anxious cognitions are more marked in agoraphobia than other kinds of phobic disorder
   - a.k.a. ‘housebound housewife syndrome’

Epidemiology
- lifetime prevalence = 0.6 - 6 %
- F:M = 4:1
- mean age of onset = 28 years old
- long history before treatment
- 80 % married
- nearly all unemployed or housewife

Aetiology
1. Biological
   a) similar biochemical factors to panic disorder
   b) genetically:
      i) risk of panic attacks in 1st degree relatives of both panic disorder and agoraphobic patients is 20 %
      ii) however, there is an increased risk of agoraphobia only in the relatives of agoraphobic patients
2. Psychosocial
   a) again, similarities exist between agoraphobia and panic disorder
   b) agoraphobics tend to have a more unstable home background
   c) the condition is often said to coincide with a major life change in people who have previously exhibited dependent traits

Treatment
- see panic disorder
- the psychological side may involve more work with the sufferer’s family as they may be helping to maintain and reinforce the behaviour
Course and prognosis

- agoraphobia without panic disorder is often disabling and crippling
- often has a fluctuating course
- 50% have other DSM III diagnosis
  - 39% have concomitant depression or dysthymia
  - 12% have social phobia
  - 10% have simple phobia

Social phobia (F40.1)

Clinical features
1. the psychological or autonomic symptoms must be primarily manifestations of anxiety and not secondary to other symptoms, such as delusions or obsessional thoughts
2. the anxiety must be restricted to or predominate in particular social situations:
   - marked fear of being the focus of attention, or fear of behaving in a way that will be embarrassing or humiliating e.g. eating or speaking in public, encountering known individuals in public, entering small group situations
3. avoidance of the phobic situations must be a prominent feature

- social phobias may be secondary to a depressive illness, as social performance drops:
  - mainly female
  - onset age 30 - 50
  - continuous course

Epidemiology

- 6-month prevalence = 2-3 / 1000
- age of onset: late adolescence; most cases start between the ages of 17 and 30
- age range = 5-35 years
- F:M = 3:2/ equal incidence
- the first episode occurs in a public place, usually without any reason
- transcultural variants include taijin-kyofu-sho in Japan

Associations

- alcohol abuse is more common in social phobia than other phobias

Aetiology

1. Biological
   a) neurochemical theories:
      i) social phobics may release more catecholamines than non-phobics
      ii) dopaminergic activity may be related to the disorder - MAOIs are effective treatments
b) **genetic:**
   i) 1st degree relatives of social phobics are 3 x more likely to be affected
   ii) MZ twins may be more concordant than DZ twins

2. **Psychosocial**
   a) see specific phobias
   b) parents of sufferers are said to be:
      i) less caring
      ii) more rejecting
      iii) more overprotective

**Treatment**

1. **Psychological**
   a) *Cognitive behavioural therapy:*
      i) cognitive retraining
      ii) desensitization
      iii) rehearsal during sessions, such as social skills training
      iv) homework assignments

2. **Pharmacotherapy**
   a) *MAOIs,* especially **PHENELZINE** are effective in the treatment of the generalized form of social phobia - perhaps by increasing risk-taking behaviours and acting as *psychological activators*
   b) **SSRIs** are effective treatments, especially **PAROXETINE**
   c) tricyclics and **BUSPIRONE** are relatively ineffective
   d) the social phobia associated with performance anxiety is best treated with small doses of β-blockers such as **PROPANOLOL**

**Course and prognosis**

- generally present for life
- complications:
  - alcohol and drug dependence (usually prescribed)
  - secondary depression

**Specific phobias (F40.2)**

**Definition of a phobia (Marks)**

1. fear out of proportion to the demands of the situation
2. it cannot be reasoned away
3. it is beyond voluntary control
4. fear leads to avoidance of the feared situation
5. (leads to some degree of impairment of normal life)

**Epidemiology**

- 6-month prevalence = 5 - 10 %
- lifetime prevalence = 4 % in men, 13 % in women
• F:M = 2:1 (1:1 for needle/ injection phobia)
• age of onset: 5 - 9 years
• environmental and injury phobias: mid 20’s
• frequency of phobias (in descending order):
  • animals
  • storms
  • heights
  • illness
  • injury
  • death

Types of phobia
1. **animal phobias**
   a) onset usually before age 8
   b) F>M:
      i) 95 % of people presenting for treatment are women
      ii) boys tend to grow out of their phobias in adolescence
   c) often little generalization
   d) few associated symptoms
2. **blood / injury phobia**
   a) more likely to have a family history than other phobias – strongest evidence of a genetic component
   b) after initial tachycardia, the patient gets vasovagal bradycardia and a drop in blood pressure, resulting in nausea and fainting
3. **space phobia**
   a) characterized by fear of absent visuospatial support and of falling
   b) onset 55 years
   c) seldom accompanied by (generalized) anxiety or depression
   d) generally associated with a stable personality
   e) seen in association with neurological and cardiovascular disorders
   f) does not respond well to behavioural psychotherapy
   g) may indicate disturbed integration of vestibulo-ocular reflexes due to diverse lesions in the neck or more centrally; nystagmus is commonly to the left and dizziness can often occur on turning the head to the right
   h) the fear of falling may be associated with inappropriately generated rescue reactions
4. **Illness phobia**
   a) equal sex incidence
   b) chronic ruminations, but no attempts at resistance
   c) previous illness in relative or individual may be a precipitant
5. **Phobia of dental treatment**
   a) prevalence of 5 %

Aetiology
1. **Biological**
   a) **genetic:**
      i) up to ¾ of affected probands have a 1st degree relative with the same phobia
2. **Psychosocial**
   a) *Behavioural theories:*
      i) phobia develops due to *classical conditioning*
      ii) *operant conditioning* is used to explain the persistence of a phobic response such that the avoidance of the feared situation is in itself rewarding and hence reinforcing
      iii) phobias may also develop through *modelling* (i.e. watching a parent) or by *information transfer* (i.e. being taught to be frightened of something)
   b) *Psychodynamic theories:*
      i) Freud viewed phobias as a *displacement* of a sexual conflict from a person to a seemingly irrelevant object or situation, which then has the power to arouse the full range of emotions
      ii) avoidance is seen as part of this defence

**Treatment**

1. **Psychological**
   a) *Exposure therapy:*
      i) flooding – best when free-floating anxiety is present
      ii) systematic desensitization – best when free-floating anxiety is absent
   
2. **Pharmacotherapy**
   a) *β-blockers* or *benzodiazepines* may help the patient to engage more easily in exposure therapy

**Prognosis**

- simple phobias that originate in childhood continue for many years
- phobias starting in adult life after stressful events have a better prognosis
- systematic desensitization:
  - good response with:
    - more specific phobias
    - good relaxation response
    - patient motivation
    - relative support
  - poor response with:
    - free-floating anxiety
    - poor motivation
    - secondary gain
    - severe obsessions
F41 Other anxiety disorders

F41.0 Panic disorder
F41.1 Generalized anxiety disorder
F41.2 Mixed anxiety and depressive disorder
F41.3 Other mixed anxiety disorders
F41.8 Other specified anxiety disorders
F41.9 Anxiety disorder, unspecified

Panic disorder (F41.0)

Clinical features

- a panic attack may be defined as a sudden onset of a discrete period of severe anxiety in which at least four or more of the following symptoms have been experienced:
  1. palpitations
  2. sweating
  3. trembling or shaking
  4. sensation of shortness of breath
  5. feeling of choking
  6. chest pain / discomfort
  7. nausea / butterflies
  8. dizziness / lightheadedness
  9. derealization / depersonalization
  10. fear of losing control / going crazy
  11. fear of dying
  12. paraesthesia
  13. chills or hot flushes

- in order to make a diagnosis of panic disorder, the patient should have experienced at least three panic attacks within a three week period. The attacks should occur:
  a) in circumstances where there is no objective danger
  b) without being confined to known or predictable situations
  c) with comparative freedom from anxiety symptoms between attacks
     (although anticipatory anxiety is common)

Epidemiology

- 6-month prevalence = 0.6 - 1 %
- lifetime prevalence = 1.5-3 %
- F:M = 2:1
- mean age of onset = 25 years old; rare after the age of 40
- age range 25-44
Aetiology

1. Biological
   a) major neurotransmitter systems involved are:

<table>
<thead>
<tr>
<th>System</th>
<th>Panic-inducer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Noradrenergic</td>
<td>Yohimbine</td>
</tr>
<tr>
<td>Serotonergic</td>
<td>d-Fenfluramine</td>
</tr>
<tr>
<td>GABA</td>
<td>Flumazenil</td>
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</tbody>
</table>

   b) panic can also be induced with CHOLECYSTOKININ, MCCP (5-HT receptor agonist), SODIUM LACTATE, and the inhalation of carbon dioxide
   c) genetic studies have reported a 4-8 x increase in panic disorder amongst the relatives of affected probands

2. Psychosocial
   a) separation in early life is associated with panic - there is an increase in frequency of separation of parents and separation from mother before the age of 17 in patients with panic disorder
   b) cognitive behavioural theories stress that anxiety is a learned response and the genesis and maintenance of panic attacks is explained by a combination of classical conditioning and the negative catastrophic thoughts that patients have during attacks
   c) psychoanalytical models view panic attacks as arising from unsuccessful attempts to defend against anxiety provoking impulses

Brain imaging
- PET scanning shows abnormal increases in right para-hippocampal area:
  * increased blood flow
  * increased blood volume
  * increased O₂ metabolism

Treatment

1. Psychological
   a) Cognitive behavioural therapy combines both exposure to the feared stimulus with relaxation, and work around the patient’s false cognition and giving information about the panic attack.
   b) This is probably the treatment of choice at this time.

2. Pharmacotherapy
   a) Antidepressants of most classes are useful in the treatment of panic
      i) they all may cause a degree of over-stimulation during the initiation of treatment.
      ii) the most frequently used are IMIPRAMINE (low doses), CLOMIPRAMINE, FLUOXETINE and PHENELZINE
      iii) they should be given at least 8 to 12 weeks to exert their full effects and be continued for 8 to 12 months.
   b) Benzodiazepines may also be useful adjuncts to treatment, especially during the initial phase of antidepressant treatment
      i) the drug of choice is ALPRAZOLAM (Xanax®) - unfortunately this cannot be prescribed on the NHS
ii) therefore one may use short courses of diazepam at relatively low doses.

Course and prognosis
- 30-40% of patients appear to be symptom free at long term follow up
- 50% have very mild symptoms
- 10-20% continue to have disabling symptoms
- poor outcome predicted by lower social class and long duration of illness
- depression may occur in 40 - 80% of panic patients
- alcohol and substance dependency may occur in up to 20 - 40% of patients
- increased risk of suicide compared to the normal population

Generalized anxiety disorder (F41.1)

Clinical features
- the essential feature of this disorder is anxiety, which is generalized and persistent but not restricted to, or even strongly predominating in, any particular environmental circumstances (i.e. it is "free floating")
- The symptoms are variable but in order to make the diagnosis the sufferer must have primary symptoms of anxiety most days for at least several weeks at a time, and usually for several months. These symptoms should usually involve elements of:
  a) apprehension (worries about future misfortunes, feeling "on edge", difficulty in concentrating etc.)
  b) motor tension (restless fidgeting tension headaches, trembling, inability to relax)
  c) autonomic overactivity (lightheadedness, sweating, tachycardia or tachypnoea, epigastric discomfort, dizziness, dry mouth, etc.)
- if symptoms of Generalized Anxiety Disorder (GAD) and Depression are present, but neither set of symptoms, considered separately, is sufficiently severe to justify a diagnosis then the category Mixed Anxiety and Depressive Disorder may be used

Epidemiology
- 1 year prevalence = 3 -8%
- F:M = 2:1
- mean age of onset = any age; but as a general rule, its onset is earlier than for other phobic disorders, and it is more gradual
- early onset GAD is more likely to be female, have a history of childhood fears, and have marital or sexual disturbance
- more common in 20’s

Aetiology
1. Biological
   a) the two most implicated receptor groups are:
      i) serotinergic
ii) GABAminergic

b) genetically:
  i) about 25% of first degree relatives of sufferers have GAD
  ii) male relatives are more likely to have an alcohol misuse disorder
  iii) some twin studies report a MZ concordance of 50%, and a DZ concordance of 15%

c) there is an association with *mitral valve prolapse*

2. **Psychosocial**
   a) *cognitive behavioural* theories suggest that GAD arises from selective attention being given to negative details in the environment, by distortions in information processing, and by an overly negative view of one’s ability to cope
   b) *psychoanalytical* models hypothesize that anxiety is a symptom of unresolved unconscious conflicts
   c) environmental factors (such as loss of a parent) contribute a higher vulnerability for the development of generalized anxiety disorder than other disorders, such as panic disorder

Treatment

1. **Psychological**
   a) *Cognitive behavioural therapy* addresses both the distorted negative thought processes and some of the somatic symptoms directly, through relaxation and biofeedback
   b) *Supportive therapy* has doubtful long term clinical efficacy, although patients report a lessening of their anxiety if they can discuss their worries with a sympathetic individual
   c) *Psychodynamic psychotherapy* mediates its effects by increasing the patient’s anxiety tolerance

2. **Pharmacotherapy**
   a) *Benzodiazepines* are often useful in the short term or to allow patients to cope with the psychotherapeutic approaches
      i) intermediate half-life BZDs should be used at low, divided doses
      ii) effective in 40%
   b) *Antidepressants* including IMIPRAMINE and the SSRIs are often useful, especially if there is significant affective overlay
   c) BUSPIRONE, a 5-HT₁A agonist is effective in around 60-80% of patients
      i) more effective on the cognitive than somatic symptoms

Course and prognosis
- generally a chronic disorder, with a lifelong fluctuating course
- 70% have mild or no impairment, 9% have severe impairment
- 50% will develop a major depressive illness
- poorer prognosis with:
  - severe symptoms
  - syncopal episodes
  - agitation
  - derealization
  - hysterical features
• suicidal ideas
F42.0 Obsessive compulsive disorder

F42.0 Predominantly obsessional thoughts or ruminations
F42.1 Predominantly compulsive acts (rituals)
F42.2 Mixed obsessional thoughts and acts
F42.8 Other obsessive-compulsive disorders
F42.9 Obsessive-compulsive disorder, unspecified

Subtypes
1. Neurodevelopmental
   a) more common in males
   b) earlier onset
   c) chronic course
   d) more severe symptoms
   e) associated with soft neurological signs e.g. tics, visuospatial deficits
   f) poor response to SSRIs
2. Primary
   a) more common in females
   b) later age of onset
   c) episodic
   d) less severe
   e) associated with frontal neuropsychological deficits, mood, anxiety, and eating disorders
   f) good response to SSRIs

Diagnostic guidelines
1. OC symptoms must be present on most days for at least 2 successive weeks, and be the source of distress or interference with activities
2. They should have the following characteristics:
   a) they must be recognized as the individual’s own thoughts or impulses
   b) there must be at least one thought or act that is still resisted unsuccessfully, even though others may be present which the sufferer no longer resists
   c) the thought of carrying out the act must not in itself be pleasurable (simple relief of tension or anxiety is not regarded as pleasurable)
   d) the thoughts, images, or impulses must be unpleasantly repetitive

  • resistance is not an essential component (Stern and Cobb, 1978)

Characteristics of compulsive acts (DSM III-R)
1. the act has to be a purposeful one
2. it has to be performed in accordance with a certain set of rules
3. the act is not an end in itself, but is designed to bring about another state of affairs (e.g. averting disaster)
4. there has to be a disconnection between the act itself and the state of affairs it is likely to engender - a magical quality between what the patient is doing and what he is trying to achieve or prevent must be present

Clinical features
• obsessions tend to increase anxiety in a sufferer of OCD, whilst carrying out a compulsive ritual tends to decrease anxiety

1. **obsessional thoughts / ideas**
   a) repeated, intrusive thoughts interfering with normal train of thought, causing distress
   b) may be single words, phrases, rhymes, or puns - often violent, obscene or blasphemous
   c) attempts to exclude them lead to distress

2. **obsessional images**
   a) vivid, not hallucinations
   b) often of a violent or sexual nature

3. **obsessive ruminations**
   a) endless inconclusive internal debates

4. **obsessional doubts**
   a) concern over actions, e.g. gas not switched off, doors not closed

5. **obsessional convictions**
   a) notions that thought equal acts e.g. if I think about him he will die
   b) may be delusional in intensity

6. **compulsive rituals**
   a) mental rituals such as counting
   b) physical activities like washing and checking
   c) may be related to thoughts, or be unconnected
   d) handwashing is more common in women

7. **obsessional slowness**
   a) slow activity out of proportion to other symptoms
   b) affects goal directed activity - automatic activity is still carried out quickly
   c) more common in men

Phenomenology
• obsessive doubts 42 %
• fears of contamination 45 %
• bodily fears 36 %
• insistence on symmetry 31 %
• aggressive thoughts 28 %
• checking compulsions 63 %
• washing 50 %
• counting 36 %

Epidemiology
• lifetime prevalence (ECA study) = 1.9 - 3.1 %
• M:F = 1:1
  • peak for women occurs in the 24 - 35 age group
  • peak for men occurs later
• mean age at onset = 20 years
  • bimodal age of onset, with peaks occurring age 12-14 and 20-22 years
• mean age of presenting to psychiatric services = 27.5 years (Rasmussen and Tsuang, 1986)
• figures do not show a cumulative age effect - the same peak effect occurs in the younger age group:
  • ? OCD getting more common
  • patients in older group have suffered from episodes of OCD but forgotten about them

Aetiology
1. Biological
   a) dysregulation of serotonin function
      i) blunted cortisol and prolactin release to M-CPP (mixed 5-HT agonist/antagonist)
      ii) however, neuroendocrine tests of 5-HT function are not abnormal in patients with OCD
   b) genetic:
      i) MZ: DZ = 50-80%: 25%
      ii) 35% of 1st degree relatives also have OCD
      iii) between 11-80% of Tourette’s patients have obsessional symptoms; 20% of OCD patients suffer from tics
2. Psychosocial
   a) behavioural theorists argue that obsessions are conditioned stimuli and that compulsions are established as learned avoidance strategies to lessen anxiety
   b) psychoanalytical models see OCD as arising from a combination of defence mechanisms protecting the person from conflicting desires and drives:
      i) isolation (separation of an impulse or idea from its emotional content)
      ii) undoing (reversing the consequences of an action, usually with a compulsive act)
      iii) reaction formation (behaving in an opposite way to your underlying impulses)
      iv) magical thinking (everything you think about comes true)
      v) ambivalence (both loving and hating the same object)
   c) personality:
      i) 15 - 35% of OCD patients have been noted to have previous anankastic personality traits
      ii) some personality traits which are said to characterize OCD sufferers are:
         a) abnormally high expectations of unpleasant outcomes
         b) failure to live up to perfectionist ideals should be punished
c) magical rituals can prevent catastrophes  
d) erroneous perception of threat  
e) deficiency in ability to link concepts and integrate them  
f) give single events undue credence  
g) “islands of certainty amid confusion”

Brain imaging
- increased blood flow in:  
  - the frontal lobes (fronto-orbital gyrus)  
  - basal ganglia (esp. the caudate)  
  - anterior cingulate gyrus  
- static imaging has shown the caudate to be reduced in size bilaterally in the same type of patient  
- abnormalities are normalized with effective pharmacotherapy  
- may be abnormal activity in a neurological circuit involving the orbitofrontal cortex, cingulate gyrus, and caudate nucleus

Treatment
1. Psychological  
   a) Cognitive behavioural therapy is as / more effective than pharmacotherapy for OCD:  
      i) exposure and response  
      ii) response prevention – useful in ritualistic behaviour  
          a) reduces rituals in 2/3 of chronic OCD patients  
      iii) ‘thought stopping’ may be helpful in ruminations  
      iv) paradox and loop tapes  
      v) behavioural therapy is less effective for obsessional thoughts than for rituals  
      vi) 90% make worthwhile gains  
      vii) 51% show 70% reduction in symptoms  
      viii) 39% show 30-69% reduction  
   b) Supportive therapy may be useful
2. Pharmacotherapy  
   a) effective in approx. 50-80% of OCD patients  
      i) CLOMIPRAMINE and SSRIs  
          a) effects are rarely seen for the first 6-8 weeks, building to a maximum after 8-16 weeks  
          b) may be augmented with HALOPERIDOL, especially if tics are evident  
      ii) relapse rate is high when medication is stopped if BT has not been used
3. Psychosurgery  
   a) still indicated for severe, intractable OCD  
   b) most common operation is stereotactic cingulotomy  
      i) produces symptomatic improvement in about 65%  
   c) subcaudate tractotomy is also used
Prognosis

- better with:
  - mild symptoms
  - predominance of phobic ruminative ideas, absence of compulsions
  - short duration of symptoms
  - no childhood symptoms or abnormal personality traits

- worse if:
  - symptoms involving the need for symmetry and exactness
  - male sex
  - early onset
  - family history of OCD
  - presence of hopelessness, hallucinations, or delusions
F43  Reaction to severe stress, and adjustment disorders

F43.0  Acute stress reaction
   .00  Mild
   .01  Moderate
   .02  Severe
F43.1  Post-traumatic stress disorder
F43.2  Adjustment disorders
F43.8  Other reactions to severe stress

Acute stress reaction (F43.0)

Clinical features
- a transient disorder of significant severity which develops in an individual without any apparent mental disorder in response to exceptional physical and / or mental stress, and which usually subsides within hours or days
- there must be an immediate and clear relationship between the impact of an emotional stressor and the onset of symptoms
- onset is usually within a few minutes, if not immediately
- the symptoms:
  a) show a mixed and usually changing picture; in addition to the initial state of ‘daze’, depression, anxiety, despair, overactivity, and withdrawal may all be seen, but none predominates for long
  b) resolve rapidly (within a few hours at the most); in cases where the stress continues or cannot be reversed, the symptoms usually begin to diminish after 24-48 hrs and are usually minimal after about 3 days

Aetiology
- occur in response to a variety of stressors
- the severity is a complex function of degree, quantity, duration, reversibility, environment and personal context
- psychodynamic
  - personality structure and the use of appropriate coping mechanisms are apparent key factors

Treatment
- the condition is self-limiting and therefore supportive treatment is all that is required
Post traumatic stress disorder (F43.1)

Clinical features

- arises as a delayed and/or protracted response to a stressful event or situation of an exceptionally threatening or catastrophic nature, which is likely to cause pervasive distress in almost anyone
- the event usually involves the threat of severe injury or death, or a threat to physical integrity
- should not be diagnosed unless it arises within 6 months of the traumatic event
- the symptoms fall into three categories:

1. **Re-experiencing** of the traumatic event
   a) recurrent and intrusive distressing recollections of the event, including images, thoughts, and perceptions
   b) recurrent distressing dreams of the event
   c) acting or feeling as if the event was reoccurring
   d) intense physiological distress/reactivity at exposure to internal or external cues that represent the traumatic event

2. **Persistent avoidance** of stimuli associated with the trauma, and **numbing** of general responsiveness
   a) efforts to avoid thoughts, feelings, or conversations associated with the trauma
   b) efforts to avoid activities, places, or people that arouse recollections of the trauma
   c) inability to recall an important aspect of the trauma
   d) markedly diminished interest or participation in significant activities, and a feeling of detachment from others
   e) restricted range of affect, and a sense of foreshortened future (“doom”)

3. Persistent symptoms of **increased arousal**
   a) difficulty falling or staying asleep
   b) irritability or outbursts of anger
   c) hypervigilance
   d) difficulty concentrating
   e) exaggerated startle response

Epidemiology

- lifetime prevalence: 1 - 3 % (5-15 % may have subclinical disorder)
- lifetime prevalence in community = 1-9 %
- chronic PTSD seen in 1.3% males, 4.7 % females (Breslau et al. 1995)
- Vietnam veterans: c. 30 % (25 % more have subclinical disorder)
- F:M = ?
- age of onset: any age

Aetiology

1. **Stressor**
   a) no direct correlation between severity and likelihood or severity of PTSD
   b) about 25% exposed to potentially traumatic event develop PTSD
c) the subjective meaning of the event to the individual appears to be one of the most important factors

2. **Vulnerability factors**
   a) presence of childhood trauma (sufferers of PTSD report childhood sexual abuse more often than expected)
   b) borderline, paranoid, or dependent personality traits (psychopathic traits are protective)
   c) inadequate support system
e) female
   f) family psychiatric history
   g) recent stressful life changes
   h) loss of self-determination
   i) recent heavy alcohol intake

3. **Psychological**
   a) the *cognitive model* holds that sufferers are unable to process the trauma adequately, and use avoidance techniques to minimize reliving the stressor
   b) the *behavioural model* holds that PTSD arises through pairing of the stressor with physical or mental reminders of the trauma
   c) the *psychodynamic model* suggests that the trauma reactivates a previously quiescent, yet unresolved psychological conflict

4. **Biological**
   a) increased autonomic arousal:
      i) increased urinary noradrenaline
      ii) reduced amount of cortisol in both the blood and urine of chronic PTSD sufferers
   b) some evidence of dysregulation of the endogenous opiate system
c) sleep architecture is disturbed
d) impaired short term memory
e) reduced hippocampal volume on MRI scan
f) hyperresponsivity of NA neurones in locus coeruleus due to minor psychological changes
g) NMDA receptors may also be involved
h) NA dysfunction may underlie recurrent memories/ flashbacks in PTSD
i) general hyperresponsiveness of NA system observed in PTSD patients (Southwick *et al.* 1993)

Treatment

1. **Psychological**
   a) time limited *cognitive therapy*
      i) degree of exposure
      ii) stress management
   b) *family therapy* and *group therapy* increase support for the sufferer and victims of mass trauma respectively
c) *hypnosis*
d) *EMDR*

2. **Pharmacotherapy**
a) IMIPRAMINE and AMITRIPTYLINE have been used with some success in a number of trials
i) should be given at full dosage
ii) treat for at least a year
iii) less effect on avoidance, denial, and emotional numbing
b) FLUOXETINE has also shown to be effective

Adjustment disorders (F43.2)
- a maladaptive reaction to a psychosocial stressor - it is maladaptive because it interferes with the affected person’s social or occupational functioning
- the disorder is expected to remit when the stressor recedes

Clinical features
- onset is usually within one month of the stressful event or life change
- duration is not longer than six months

a) Brief depressive reaction:
   - transient mild depression
   - duration < 1 month
b) Prolonged depressive reaction:
   - mild depression in response to chronic stressor
   - duration < 2 years
c) Mixed anxiety and depressive reaction

Epidemiology
- any age of onset
- young single women are over represented
- medical/surgical patients 5% of admissions
- psychiatric patients 10% of sample population

Aetiology
- see aetiology of acute stress reaction

Treatment
1. Psychological
   a) Group therapy
      i) may reinforce positive coping strategies
   b) Individual therapy
      i) must be time limited to prevent dependence
   c) Crisis counselling
      i) is of doubtful benefit except in those with no other supports

2. Pharmacotherapy
   i) short courses of medications may be useful for brief symptom relief such as insomnia
F44 Dissociative (Conversion) Disorders

History
- Sydenham, 1681 – proposed that hysteria was a disease of the mind
- Thomas Willis – suggested hysteria was caused by a disorder of the brain
- Charcot – due to a functional disorder of the brain, and rendered patients susceptible to hypnosis
- Pierre Janet – proposed that the disorder in hysteria was a tendency to dissociate, i.e. to lose the normal integration between various parts of mental functioning
- Freud and Breuer – ‘On the psychical mechanisms of hysterical phenomena’
- Freud – suggested that hysteria was due to emotionally-charged ideas which had become lodged in the unconscious of the patient at some previous time, and which were excluded from consciousness by repression
- Kretschmer – believed that hysterical symptoms could develop in psychologically stable people as a result of a ‘reflex’ biological mechanism that counteracts highly stressful experiences

Clinical features
- primary gain
- secondary gain
- choice of symptom modeled closely on recent experience in self or others
- manipulation of others and the environment
- hysterical personality type/disorder

- common theme is a partial or complete loss of the normal integration between memories of the past, awareness of identity and immediate sensations, and control of bodily movements
- for a definite diagnosis, the following should be present:
  1. the clinical features for the individual disorders described below
  2. no evidence for a physical disorder that might explain the symptoms
  3. evidence for psychological causation, in the form of a clear association in time with stressful events and problems, or disturbed relationships (even if denied by the patient)

- primary gain: the exclusion from consciousness of anxiety due to psychological conflict
- secondary gain: the disorder confers some advantage to the patient, e.g. excused from normal responsibilities
  - secondary gain is largely socially determined

- somatic manifestations of psychiatric illness are more common in:
  - the elderly
  - children
  - lower social classes
Eastern Countries

**Dissociative Amnesia (F44.0)**

**Clinical features**
- core feature is a patchy loss of memory, usually of recent traumatic events
- perplexity, distress, and varying degrees of attention-seeking behaviour may be evident
- purposeless local wandering may occur - usually accompanied by self neglect and rarely lasts more than a day or two
- there must be an absence of organic brain disorders, intoxication, or excessive fatigue

**Epidemiology**
- thought to occur more commonly in women and more often is younger rather than older adults
- associated with organic disease:
  - epilepsy
  - multiple sclerosis
  - head injury

**Aetiology**
1. **Neuropsychological:**
   a) may be an extreme form of state dependent learning such that memory of a traumatic event is not easily recalled as the circumstances surrounding the event are so out of the ordinary
2. **Psychodynamic:**
   a) amnesia is seen as a defence mechanism used alongside repression and denial as a way of dealing with an emotional conflict or an external stressor

**Treatment**
- attempts to try and restore the patient’s lost memories by:
  - initial interview
  - informant history
  - ‘abreaction’ via medication or hypnosis

**Course and prognosis**
- the symptoms usually terminate abruptly and recovery is generally complete with few recurrences
Dissociative Fugue (F44.1)

Clinical features
- has all the features of dissociate amnesia plus an apparently purposeful journey away from home or work, during which self-care is maintained
- in some cases a new identity is assumed sometimes for long periods of time but usually only for a few days

Aetiology, course, and treatment
- as for dissociative amnesia
- often a history of:
  - disturbed relationship with parents in childhood
  - habitual liars

Dissociative disorders of movement and sensation (F44.4-44.7)

Dissociative motor disorders
Dissociative convulsions
Dissociative anaesthesia and sensory loss

Clinical features
- the symptoms often resemble the patient's idea of a physical disorder
- the core feature is loss of function which does not appear to be under voluntary control
- 'La Belle Indifference' is often seen, but it is not universal
- in order to make the diagnosis:
  1. there should be no evidence of physical disorder
  2. enough must be known about the psychological and social setting and personal relationships of the patient to allow a convincing formulation to be made for the appearance of the disorder

Epidemiology
- annual incidence: 22 per 100,000
- F:M = 2:5:1
- age of onset: more common in adolescents and young adults (may occur at any age)
- more common in:
  - rural populations
  - lower socio-economic groups
  - lower educational status
  - military personnel who have been exposed to combat situations

Aetiology
1. Biological
a) brain imaging studies suggest that the production of symptoms may be related to a defect in the communication between the hemispheres
   i) inhibits the patient's awareness of bodily sensation by setting up negative feedback loops during periods of increased cortical arousal
b) evoked responses in patients with hysterical anaesthesias suggest two underlying mechanisms:
   i) a lowering of peripheral receptor sensitivity
   ii) a central mechanism of inhibition along different pathways

2. **Psychosocial**
   a) **Psychodynamic**
      i) holds that dissociative physical symptoms are caused by the repression of unconscious intrapsychic conflict and the conversion of the anxiety produced into a physical symptom
      a) production of the symptoms may therefore allow the patient to avoid facing prohibited wishes or urges
      b) may communicate the desire for special consideration and treatment

**Treatment**
- resolution is usually spontaneous, although often helped by supportive or behaviour therapy aimed at increasing insight and issues of stress and coping

**Münchausen syndrome**
- Asher, 1951
- a.k.a. ‘Hospital addiction’ syndrome

**Epidemiology**
- M=F
- mean age of presentation = 36 years

**Aetiology**
- generally due to severe personality difficulties
- a small minority are due to depressive illness

**Clinical features**
- patients who repeatedly present to hospitals with symptoms suggestive of serious physical illness
- most common symptoms are GI symptoms, and haemoptysis
- evidence of:
  - conscious stimulation of symptoms
  - deception of medical staff
Chronic fatigue syndrome – Neurasthenia

Epidemiology
- prevalence = 7.4 per 100,000
- presentation between 20 and 50 years
- F > M
- no clear association with socioeconomic status

Aetiology
- neurological evidence inconclusive
- high prevalence of antecedent/ lifetime psychiatric illness:
  - minor depression
  - anxiety
  - somatization

Treatment
- CBT
- stress management
- biofeedback
- psychotherapy
- self-help groups
- antidepressants

Compensation Neurosis
- described by Miller (1961)
- refers to psychologically determined physical or mental symptoms occurring when there is an unsettled claim for compensation
- it has been suggested that settlement was followed by recovery, but this has not been substantiated – quite often the symptoms do not resolve
F45  Somatoform disorders

F45.0  Somatization disorder
F45.1  Undifferentiated somatoform disorder
F45.2  Hypochondriacal disorder
   includes body dysmorphic disorder
F45.3  Somatoform autonomic dysfunction
   includes Da Costa’s syndrome, irritable bowel syndrome

Somatization disorder (F45.0)

Clinical features
- a disorder characterized by multiple, recurrent, and frequent changing physical symptoms, usually present for (at least two) years
- symptoms may affect any system but the most frequent are:
  - GI tract
  - skin
  - sexual or menstrual symptoms
- in order to make the diagnosis, the patient must have refused to accept reassurance and there must be some social / occupational impairment
- previously known as Briquet’s syndrome

Epidemiology
- lifetime prevalence: 0.1 - 0.2 %
- F:M ratio = 5-20:1
- age of onset: before 30 years old (usually in the teens)

Associations
- co-morbidity high with a range of diagnoses:
  - anxiety
  - depression
  - substance abuse (esp. alcohol abuse)
  - histrionic personality disorder
  - antisocial personality disorder
- 2/3 of patients with SD have PD

Aetiology
1. Biological
   a) there is some evidence for faulty processing of sensory inputs in patients with this disorder
      i) overactivity of the ANS with smooth muscle contraction
      ii) increased tension in voluntary muscles
      iii) hyperventilation leading to biochemical changes - Lewis
iv) arousal leading to changes in endocrine activity
b) genetic:
   i) about 10-20% of 1st degree relatives of affected probands also suffer from this disorder

2. **Psychosocial**
a) often viewed as a form of communication or as substitutions for repressed instinctual urges
b) conversion of repressed affect - *Freud*
c) need to be cared for - *Menninger*
d) **The Sick Role** (*Parsons*)
i) 2 rights, and 2 obligations:
   a) exemption from normal society and responsibilities
   b) right not to be held responsible for illness
   c) expectation that the sick role is undesirable
   d) should seek help, and accept medical advice and treatment

ii) The Social Role of the Doctor includes:
   a) defining illness
   b) legitimizing illness
   c) imposing an illness diagnosis if necessary
   d) offering appropriate help

e) **Illness Behaviour** - the way in which symptoms are perceived, evaluated, and acted upon (*Mechanic*)
i) abnormal illness behaviour (*Pilowsky*)
f) somatizers in one study were found to be less depressed, have less social dissatisfaction, be less sympathetic to mentally ill, and were more likely to have been in hospital than ‘psychologisers’ (*Bridges, 1991*)
g) **Alexithymia** (*Nemiah and Sifneos*)
i) no words for mood

h) **Culture**
i) psychologization is a 20th Century phenomenon
ii) sanctions against expression of emotions
iii) idiosyncratic explanations for affective states

i) **Childhood / Family experience**
i) role of early learning / exposure to illness
ii) some families only provide care for somatization
iii) family teaches us how to evaluate mood and physical symptoms, and how to complain

j) **Cognitive coping mechanisms**
k) **Environmental systems**
i) doctors ‘somatize’ and medical care is a strong social support system
ii) family social network is a powerful source of secondary gain
iii) work-disability system

**Treatment**

1. **Psychological**
a) **Psychotherapeutic**
i) some patients may benefit, but only once they have acknowledged some psychological basis for their problems

b) Consistency
   i) patient should be seen by the same physician each time
   ii) attempts to steer the patient towards a psychological explanation

2. Specific approaches
   a) CBT
      i) Chronic fatigue syndrome
      ii) hypochondriasis
      iii) chronic pain
   b) Psychodynamic
      i) irritable bowel syndrome
   c) Behavioural
      i) chronic pain
      ii) somatization disorder
      iii) body dysmorphic disorder
   d) Pharmacological
      i) chronic pain
   e) Group therapy
      i) hypochondriasis
      ii) chronic pain
      iii) Chronic fatigue syndrome

Course and prognosis
• the disorder is often chronic, with a presentation at least once a year and a new symptom occurring when stressed

Hypochondriacal disorder (F45.2)
• includes non-delusional dysmorphophobia - a preoccupation with an imagined bodily defect
  • about 1/5 of patients attempt suicide (in contrast to most patients with hypochondriacal disorder)

Clinical features
• preoccupation with the possibility of having one or more serious and progressive physical disorders, and even a disfigurement
• any mild physical symptoms experienced by the patient are often interpreted in this context but the degree of conviction about potential diagnoses may vary between consultations
• as for SD, the patient maintains his beliefs despite repeated investigations and reassurances

Epidemiology
• six month prevalence: 4-6 %
• F:M = 1:1
• age of onset usually 20-30 years, but may occur at any age

Associations
• up to 80% may have co-existing anxiety or depressive illness
• PD is 3 x more common

Aetiology
1. four major themes:
   a) disorder may be due to a faulty cognitive scheme such that sufferers focus on bodily sensations, misinterpret them as serious, and therefore become alarmed
   b) patients are attempting to enter the sick role as a way out of insurmountable problems or events
   c) may be a variant in the presentation of anxiety or mood disorders
   d) complaints of physical illness are a displacement of aggressive and hostile wishes towards others, or as an expression of guilt and low self-esteem

Treatment
• similar to that for somatization disorder

Course and prognosis
• usually episodic, often with long gaps between presentations
• 1/3 - ½ eventually improve significantly

Somatoform autonomic dysfunction (F45.3)
• presenting symptoms relate to a system or organ largely under autonomic control, i.e. GI, cardiovascular, or respiratory systems
• the symptoms of autonomic arousal are combined with subjective symptoms referred to a specific organ or system

- Type One
  • associated with objective evidence of autonomic arousal, e.g. palpitations, sweating, flushing, tremor

- Type Two
  • characterized by more subjective symptoms, e.g. sensations of burning, tightness, bloating

Somatoform Pain Disorder

Clinical features
• inconsistent with anatomical distribution of the nervous system
• continuous over long periods by day
• may prevent getting to sleep, but doesn’t cause wakening
• may have symbolic significance, e.g. chest pain where father died of MI
• restricted insight
• responds better to psychotropics than analgesics

Epidemiology
• peak around middle age
• more common in women