GUIDELINES

FOR

THE MANAGEMENT OF

COMMON MENTAL DISORDERS

Ministry of Health & Family Welfare

Government of India
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PSYCHOSIS

Introduction

Broadly, psychiatric illnesses presenting in general health care settings can be classified into two groups, ‘Common Mental Disorders’ (CMDs) e.g. depression, anxiety disorders and ‘Severe Mental Illnesses’ (SMIs) which refers to a group of mental illnesses causing marked disruption in socio occupational life i.e. schizophrenia and bipolar disorder.

Psychosis (Greek word, ‘psyche” refers to mind/soul; and ‘-osis’ refers to abnormal condition or derangement) means ‘an abnormal condition of the mind’. Psychosis refers to a state characterized by an individual’s loss of contact with reality. It may involve abnormality in thinking and perceptions, as well as disturbances in emotions. It is categorized as a severe mental disorder causing dysfunction in the life of the patient and family.

Schizophrenia and other forms of psychoses that affect young people represent a major public health problem. It is common myth in general public to consider it due to religious and supernatural causes than illness, which often leads to delay in treatment seeking. Moreover, due to stigma attached to mental health services, there is often a delay in reaching to the psychiatrists. Thus, general physicians can be effective bridge in diagnosing, managing and effectively referring such patients.

Psychosis occurs in a number of psychiatric disorders e.g. depression, bipolar disorder (manic-depressive illness), puerperal psychosis and sometimes with drug and alcohol abuse. It can also occur in a number of medical and neurological conditions and caused due to medications.

Clinical features

Patients can present with following complaints related to psychosis:

- Abnormal/disorganized behavior /unusual appearance
- Un-understandable talk/Non-sensible speech
- False beliefs (e.g. ‘people are planning to kill me’)
- False perception (e.g. hearing voices of people not around)
- Social withdrawal
- Neglect of usual responsibilities related to work/school/domestic chores
- Talking to self
- Violent/aggressive behavior
- Restless and running here and there

Clinical symptoms and signs for evaluation of psychosis for ease of understanding can be divided into following two groups:

1. **Positive symptoms**: They are called ‘positive’ since the person’s thoughts, beliefs or sensations seems to be ‘abnormally expanded or greater’ than normal. They suggest person having lost contact from reality and of having created one’s false world. They include:
   - **Hallucinations**: It refers to perceiving any sensations in absence of a real stimulus, e.g. hearing or seeing things that are not there.
   - **Delusion**: It refers to false beliefs that are held with extraordinarily conviction and are not shared by other members of the society.
   - **Formal Thought disorder**: It refers to the disorganization in thought as reflected by speech or sentences which are disjointed or cannot be understood e.g. while speaking person looses chain of thought or one thought has no connection with other.

2. **Negative symptoms**: They are called ‘negative’ since they involve ‘decrease’ in a person’s usual experiences and functioning. They include:
   - Little/ no drive to do things
   - Lack of energy and interest
   - Little display of feelings
   - Speaking very less
Diagnosis

There are no laboratory tests available for diagnosis of psychosis. Diagnosis is entirely clinical. The following set of symptoms when present together can be helpful in making diagnosis:

Schizophrenia

More than two symptoms of the following if present for more than one month duration causing disturbance in functioning:

1. Delusions
2. Hallucinations
3. Disorganized speech
4. Grossly disorganized or catatonic behavior
5. Negative symptoms

Subtypes of schizophrenia

1. **Paranoid Schizophrenia**: Delusions; Hallucinations (e.g. belief people are making plans to kill me or hearing of voices; most common subtype; better prognosis

2. **Catatonic Schizophrenia**: Stupor; Rigidity; Mutism; Maintaining odd postures for long time; Good prognosis

3. **Hebephrenic Schizophrenia**: Disorganized speech; Self care and behavior e.g. vagabonds on street; Poor prognosis

4. **Simple schizophrenia**: Negative symptoms; gradual onset; Long lasting; Poor prognosis

5. **Residual schizophrenia**: Earlier fulfill criteria for Schizophrenia; Partially attenuated form mainly as negative symptoms
**Acute Psychosis**

It is characterized by appearance of psychotic symptoms within 2 weeks. It usually has rapidly changing clinical picture and complete improvement within 1 to 3 months, spontaneously or with effective treatment.

**Persistent delusional disorder**

It is characterized by the presence of delusion, usually centered on one theme with absence of hallucinations. Person’s social and occupational functioning in all areas other than the theme of delusion is unaffected. Delusion can be persecutory (e.g. suspicious or fearful of being harmed), grandiose (e.g. believes self to be in possession of extra power, worth knowledge, ability or identity) or somatic (e.g. false belief of having an abnormal somatic sensation or illness).

**Affective psychosis**

**Mania with psychotic symptoms**

Presence of psychotic symptoms in background of following symptoms for 1 week:

Markedly elevated or irritable mood

Excessive energy and activity

Excessive talking

Usually psychotic symptoms are grandiose delusion and auditory hallucinations

**Depression with psychotic symptoms**

Presence of psychotic symptoms in the background of following symptoms for 2 weeks:

Depressed mood
Decreased energy and activity

Usually psychotic symptoms are nilhistic delusion (belief of having every body organ inside having decayed) and auditory hallucinations

**Organic Psychosis due to general medical condition**

Psychosis associated with medical or surgical condition/ drug-induced

**Assessment**

*Good clinical history, thorough medical examination and mental status examination are the keys to make diagnosis.* Take history from patient, accompanying caregivers and screen relevant treatment and investigation records. Cover following points in your questions to assess symptoms and other relevant history:

a. What is the nature of the hallucination or delusion?

b. What is the time span?

c. Is there a recurring theme?

d. Is there insight into it being unreal?

e. Is there history of any medical illness?

f. Have there been any recent major life-events?

g. Is there a history of substance abuse (alcohol or drugs)?

h. Does the patient's past behavior suggest psychological vulnerability, e.g. irritability, uneasiness, suspiciousness and withdrawn mood?

i. Is there a family history of mental illness?

While taking the history it is possible to make an assessment of the patient's mental state:

a. Is there loss of touch with reality; are there delusions or hallucinations?

b. Is thought or speech disorganized, abstract or vague?

c. Is emotion normal and appropriate? Remember that such experiences will naturally cause extreme anxiety but are there inappropriate emotional outbursts?
d. Is there excitement or confusion?
e. Is there depression or suicidal ideation?

**Investigations**

Though no specific test is diagnostic, but depending on the possible list of differentials for causes of organic psychosis, investigations can be advised. Look for concurrent conditions e.g. alcohol use, signs or symptoms suggestive of stroke/ diabetes/ hypertension/ HIV or AIDS/ cerebral malaria/ medications usage (e.g. steroids, ATT).

**Treatment**

A comprehensive treatment program includes:

1. Antipsychotic medication, which forms the cornerstone of treatment of psychosis
2. Education of the individual about his/her illness and treatment
3. Family education and support
4. Support groups and social skills training
5. Rehabilitation to improve the activities of daily living
6. Vocational and recreational support

**Pharmacological management**

After identification of the case, antipsychotic medications should be started depending on clinical status. The treatment can be broadly divided into two phases acute and maintenance. The goals of acute phase of treatment are to reduce symptoms, to prevent harm to self/others and improve biological functions. The goal of maintenance phase of treatment is to prevent relapse and to help patient improve one’s level of functioning.
Selection of antipsychotic drug: Two classes of antipsychotic drugs are available, typical antipsychotics (haloperidol, chlorpromazine, trifluoperazine) and atypical antipsychotics (risperidone, olanzapine, quetiapine). Both the groups are equally effective, but differ in their side effect profiles. In typical antipsychotics, high potency drugs (e.g. haloperidol) have more extrapyramidal side (EPS) effects and low potency drugs (e.g. chlorpromazine, fluphenazine, trifluoperazine) have more anticholinergic side effects (e.g. dryness of mouth, urinary retention, constipation) and cardiovascular side effects (e.g. tachycardia, postural hypotension).

Managing side effects

Monitoring for common acute side effects is essential:

1. Extrapyramidal side effects (drooling of saliva, rigidity, fine tremors in hands), acute dystonia (sudden sustained contraction of a group of muscles, most commonly neck and oral musculatures are affected), oculogyric crisis (sudden up rolling of eyeballs), rabbit syndrome (fine perioral tremors). Manage extrapyramidal side effect by reducing antipsychotic dosage or addition of oral anticholinergic drug e.g. trihexyphenidyl or acutely by giving injection promethazine.

2. Cardiovascular side effects (hypotension, bradycardia, QTc prolongation in ECG): These side-effects require reducing dosage or switching to other agents.
<table>
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<th>Antipsychotic</th>
<th>Adult dose range (mg/day)</th>
<th>Side effects</th>
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<tbody>
<tr>
<td></td>
<td><strong>Acute</strong></td>
<td><strong>Maintenance phase</strong></td>
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<tr>
<td>Chlorpromazine</td>
<td>100-1600 mg oral; 25-400 mg IM</td>
<td>50-40 mg oral</td>
</tr>
<tr>
<td>Trifluoperazine</td>
<td>4-40 mg oral</td>
<td>5-20 mg oral</td>
</tr>
<tr>
<td>Fluphenazine</td>
<td></td>
<td>12.5-50 mg IM (deaconate, fortnightly)</td>
</tr>
<tr>
<td>Haloperidol</td>
<td>5-20 mg oral; 1-5 mg oral;</td>
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<tr>
<td></td>
<td>5-20 mg IM (deconate, monthly)</td>
<td>25-200 mg IM</td>
</tr>
<tr>
<td>Risperidone</td>
<td>4-6 mg oral</td>
<td>Sedation, extra pyramidal side effects, amenorrhea</td>
</tr>
<tr>
<td>Olanzapine</td>
<td>7.5-30 mg oral</td>
<td>Sedation, postural hypotension, weight gain</td>
</tr>
<tr>
<td>Quetiapine</td>
<td>300-800 mg oral</td>
<td>Sedation, postural hypotension, dizziness</td>
</tr>
</tbody>
</table>
Non pharmacological management

Psycho-education: Discuss with the patient and family regarding:

- The person’s ability to recover;
- The importance of continuing regular social, educational and occupational activities, as far as possible
- The suffering and problems can be reduced with treatment
- The importance of taking medication regularly;
- The right of the person to be involved in every decision that concerns his or her treatment
- Importance of staying healthy (e.g. following healthy diet, staying physically active, maintaining personal hygiene).

- Additional messages to family members of people with psychosis
  -- The person with psychosis may hear voices or may firmly believe things that are untrue
  -- The person with psychosis often does not agree that he or she is ill and may sometimes be hostile
  -- The importance of recognizing the return/worsening of symptoms and of coming back for re-assessment should be stressed
  -- The importance of including the person in family and other social activities should be stressed
  -- Family members should avoid expressing constant or severe criticism or hostility towards the person with psychosis.
  -- Person with psychosis may have difficulties recovering or functioning in high-stress working or living environments.
  -- It is best for the person to have a job or to be otherwise meaningfully occupied.
**Follow up care**

People with psychosis require regular follow-up. In this phase, particularly general physicians can be of great help. Once a patient is in remission, or behaviourally stable, can continue to follow up locally with nearest general physician. Subsequently, patient can be referred to specialised mental health services only on need basis.

**Follow up frequency:**

Acute phase: Follow up once or twice weekly.

Maintenance phase: Follow up every one to three month.

**Follow up assessment:** During follow up visits, assess for the following:

- Level of symptoms
- Side-effects of medications
- Treatment adherence: Treatment non-adherence is common, address it
- Assess for and manage concurrent medical conditions
- Assess for the need of psychosocial interventions at each follow-up
- Maintain realistic hope and optimism during treatment
- Involvement of carers is critical during such periods

**Referrals**

In following situations, consider need for referral to specialized mental health services:

1. **Nature of illness**

   - Severe level of symptoms and distress
   - Suicide or risk of harm to others
   - Marked violent aggressive behavior
   - Catatonic symptoms
   - Poor general medical status
-Refusal to accept orally (meals/ medications)

2. Nature of treatment

-Partial or no response to treatment

-Need to start modified electroconvulsive therapy

-Need to start clozapine

-Need of specific psychological therapies or vocational rehabilitation

3. Support system

-Poor social support system (e.g. homelessness)

-Family needs psycho-education about nature of illness and need of treatment
DEPRESSION AND OTHER AFFECTIVE DISORDERS

Affective disorders encompass all disorders of mood that are persistent and pervasive and lead to socio-occupational dysfunction. By far, Depression is the most commonly encountered mental disorder by a general practitioner and hence a comprehensive understanding of the disorder is indispensable. This is the broad classification of affective disorders.

Introduction and epidemiology

Mood disorders mainly manifest as either manic or depressive episodes. The lifetime risk of depression varies from 10-15% with the risk being twice more in women than men. Depression has a multifactorial aetiology as depicted below. The disorder may manifest at any age but the incidence seems to peak in 20s. The disorder may have a variable course but if untreated can run a chronic and disabling course. Thus early identification and treatment increases the likelihood of remission and recovery.
Diagnosis:

For a depressive episode: diagnostic criteria

For at least 2 weeks, has the person had at least 2 of the following core symptoms:

- Depressed mood (most of the day, almost every day), (for children and adolescents- irritability or depressed mood)
- Loss of interest or pleasure in activities that are normally pleasurable
- Decreased energy or easily fatigued

During the last 2 weeks has the person had at least 2 other features of depression

- Reduced concentration and attention
- Reduced self-esteem and self-confidence
- Ideas of guilt and unworthiness
- Bleak and pessimistic view of the future
- Ideas or acts of self-harm or suicide
- Disturbed sleep
- Diminished appetite

Symptoms should be causing significant socio occupational dysfunction

Rule out bereavement in past 2 months

Rule out the possibility of bipolar depression. (Ask about prior episode of manic symptoms such as elevated, expansive or irritable mood, increased activity and talkativeness, flight of ideas, decreased need for

If 2 other features Mild depressive episode

If 3 other features Moderate depressive episode

If 3 of the core features and 4 other symptoms Severe depressive episode It may be with or without psychotic

Risk factors for depression

- Female sex
- Age <40 years
- Impaired interpersonal relationships
- Divorced, widowers
- Family history of depression
Recurrent depressive disorder

Recurrent episodes of depression with current episode being mild, moderate or severe

Dysthymia

A chronic depressive state that does not meet criteria for a depressive episode anytime and lasts for at least 2 years

An episode can be with or without **Somatic syndrome** (4 or more of the following)

- Loss of interest in activities and lack of emotional reactivity
- Loss of appetite
- Weight loss
- Insomnia mainly as early morning awakening
- Depression worse in morning
- Low libido
- Psychomotor retardation or agitation
Clinical features

As seen in the diagnostic criteria the clinical features of depression encompass domains of psychological, somatic and psychotic symptoms if severe.

<table>
<thead>
<tr>
<th>Psychological</th>
<th>Somatic</th>
<th>Psychotic</th>
<th>Suicidal thoughts</th>
</tr>
</thead>
<tbody>
<tr>
<td>• sadness</td>
<td>• weight loss or gain</td>
<td>• delusions (pertaining to ideas of sin,</td>
<td>• always enquire about ideas of self harm</td>
</tr>
<tr>
<td>• hopelessness</td>
<td>• insomnia or hypsomnia</td>
<td>poverty, deserved punishment, imminent</td>
<td>proactively</td>
</tr>
<tr>
<td>• helplessness</td>
<td>• loss of libido</td>
<td>disasters for which patient feels responsible)</td>
<td></td>
</tr>
<tr>
<td>• worthlessness</td>
<td>• psychomotor slowing or agitation</td>
<td>• hallucinations (usually auditory of</td>
<td></td>
</tr>
<tr>
<td>• guilt</td>
<td></td>
<td>accusations and defamation or olfactory of</td>
<td></td>
</tr>
<tr>
<td>• pessimism</td>
<td></td>
<td>rotting things)</td>
<td></td>
</tr>
<tr>
<td>• anhedonia</td>
<td></td>
<td>• stupor (if marked slowing)</td>
<td></td>
</tr>
<tr>
<td>• difficulty</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• concentrating</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• loss of interest</td>
<td></td>
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Following medical conditions, drugs and psychiatric disorders may be comorbid with depression and should always be explored and only then can the patient be treated holistically.

<table>
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<tr>
<th>General medical conditions</th>
<th>Other psychiatric disorders</th>
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<tbody>
<tr>
<td>Endocrine</td>
<td>Anxiety disorders</td>
</tr>
<tr>
<td>Thyroid / glucocorticoid disturbance</td>
<td>Substance use disorders</td>
</tr>
</tbody>
</table>
Meningitis, encephalitis, syphilis, HIV
Systemic (UTI, pneumonia, sepsis)

**Cardiovascular**
Shock, cardiac failures

**Cerebrovascular**
Infarcts, haemorrhage

**Metabolic**
Dyselectrolytemia, renal/hepatic failures, hypo/hyperglycaemia

**Nutritional**
Vitamin B12, Folate deficiency

**Tumours**
Cerebral/systemic

**Trauma**
Subdural haematoma, cerebral contusions

Drugs

**Withdrawal of**
caffeine, nicotine, sedatives, amphetamines

**Prolonged use of**
Antihypertensive drugs
Anti-inflammatory/ analgesics
Steroids
Antineoplastic drugs
Anti-parkinsonism drugs
Antipsychotics
Sedatives
Many antibacterial/ antifungals

**Mental status examination**

Generalised psychomotor retardation (or agitation in elderly), stooped posture, slow spontaneous movements, downcast gaze, decreased rate of speech with increased reaction time, depressed mood and restricted affect, with delusions or hallucinations in perceptual disturbance may be seen. Thought process may be slow with poverty of ideas. Though sensorium is intact memory may be impaired with increased impulsivity (assess for suicide) with impaired judgement (too pessimist) and insight may be impaired. Usually the classic triad of pessimist thoughts about self, future and others may be seen.

A differential diagnosis needs to be done from the comorbid conditions as listed above as treating the underline cause may change the course of Depression. Also the distinction of unipolar and bipolar depression is inevitable for optimal treatment.
Management:

Goals

- Psychoeducation
- Relieve the symptoms of present episode (remission)
- Frequent follow ups until remission and also later
- Restore premorbid functioning of individual
- Maintain treatment to prevent relapse (usually if no or minimal depressive symptoms for 9 – 12 months and ability to carry out routine activities for that time period consider tapering and stopping the antidepressant)

PHARMACOLOGICAL

Tricyclic antidepressants (TCA): Imipramine, Amitryptiline
Selective Serotonin Reuptake Inhibitors (SSRI): Fluoxetine, Sertraline, Escitalopram
Serotonin Norepinephrine receptor inhibitors: Venlafaxine
Others: Mirtazepine

PSYCHOLOGICAL

Psychoeducation, cognitive behavioural therapy (CBT)

PSYCHOSOCIAL

Psychoeducation, address current psychosocial stressors, reactivate social networks, activity scheduling, physical activity
Select an antidepressant from the **WHO Formulary**. Fluoxetine and amitriptyline (as well as other tricyclic antidepressants (TCAs) are antidepressants mentioned in the WHO Formulary.

In the **national list of essential medicines** Fluoxetine, Imipramine and Amitriptyline have been mentioned.

**National formulary of India** advises use of Fluoxetine, Escitalopram, Imipramine and Amitriptyline.

Following table summarises the drug information.

<table>
<thead>
<tr>
<th>SSRI's</th>
<th>TCA</th>
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<tr>
<td><strong>Common side-effects</strong> (mostly self-limiting)</td>
<td><strong>Common side-effects</strong> (mostly self-limiting)</td>
</tr>
<tr>
<td>Restlessness, nervousness, insomnia, anorexia, gastrointestinal disturbances, headache, sexual dysfunction</td>
<td>Orthostatic hypotension (fall risk), dry mouth, constipation, difficulty urinating, dizziness, blurred vision and sedation.</td>
</tr>
<tr>
<td>Risk of switching to mania in people with bipolar disorder.</td>
<td><strong>Serious side-effects</strong>: cardiac arrhythmia.</td>
</tr>
<tr>
<td><strong>Time to response</strong>: 4 – 6 weeks after adequate dose</td>
<td>Risk of switching, lethal in overdose, sedating</td>
</tr>
<tr>
<td><strong>Dose for fluoxetine</strong>: Initiate treatment with 20</td>
<td><strong>Time to response</strong>: 4 – 6 weeks after adequate dose</td>
</tr>
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</table>

**At primary health care level**, it may be feasible to

- Select a drug from national formulary and give according to instructions given below.
- Give psychosocial interventions along with, depending on severity of depressive symptoms.
- Exploring the psychosocial stressors of patient in privacy, empathetically and helping problem solve them may be needed. Family and friends can be used to build up support system for patient. Any pertinent stress like abuse may need to be dealt with in patient’s environment.
- Patient should be encouraged to resume the earlier pleasurable hobbies and social meetings to reactivate his social circuits.
- Patient may need to be helped with a schedule according to his strengths and weaknesses and appropriately rewarded and applauded for the same with the help of family members initially.
- Some sort of physical activity or yoga may be advised (45 minutes 3-5 times/week)
- Any comorbid medical condition should be treated adequately at primary health care level
mg daily (if not tolerated even 10 mg) once
daily and increase to 20 mg if the medication is
tolerated. If no response in 4 – 6 weeks or
partial response in 6 weeks, increase dose by
20 mg (maximum dose 60 mg)

For children and elderly start with 10 mg and
increase gradually to 20 mg. wait for 6-12
weeks before up titration.

Monitor for emergence of suicidal ideation in
adolescent population and refer to specialist if
no response.

**Dose of amitriptyline:**

Start with 50 mg at night and reach up to 100-
150 by 4-6 week and if no/partial response
increase further(maximum 200 mg)

Do not prescribe for adolescent population

For elderly start low with 25 mg and go slow
with wait up to 12 weeks and maximum dosage
being 100 mg.

DO NOT prescribe TCA to people at risk of
serious cardiac arrhythmias or with recent
myocardial infarction. Monitor blood pressure
for all cardiovascular patients on TCA.

**Special population**

<table>
<thead>
<tr>
<th><strong>Individuals with concurrent conditions</strong></th>
<th><strong>Pregnancy and breast feeding</strong></th>
<th><strong>children</strong></th>
<th><strong>Elderly</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Treat both the depression and the concurrent condition</td>
<td>Avoid psychotropic in pregnancy, and manage with non-pharmacological means, however weigh the risk benefit ratio of continuing an antidepressant during pregnancy. Avoid long acting fluoxetine during breast feeding</td>
<td>Offer pharmacotherapy with fluoxetine (&gt; 12yrs) only if non pharmacological treatments fail. Monitor for emergence of suicidal ideation.</td>
<td>Avoid Tricyclic antidepressants, watch for drug interactions, expect slower response, increase dosages gradually and treat with lower dosages</td>
</tr>
</tbody>
</table>

**When to refer**

- Patient has severe depression with psychotic features
- Suicidal ideations
- Failure of 2 or more antidepressants
- Psychological intervention needed (lack of resources)
- Bipolar affective disorder
- Multiple comorbid conditions
- Special population if not manageable
- Any other emergency comorbid with mood disorder
Bipolar affective disorder (BAD)

BAD is characterised by an episode of mania or hypomania. It may or may not be followed by a subsequent mood episode. About 5-10% of patients with initial diagnosis of major depressive episode have a manic episode later on.

Diagnostic criteria

Mania

Elevated, expansive or irritable mood + 3 of the following (4 if mood is irritable)

- Increased activity, restlessness, excitement
- Increased talkativeness
- Loss of social inhibitions
- Decreased need for sleep
- Overspending or other reckless behaviours
- Distractibility
- Elevated sexual energy

Hypomania

Same features as mania but with a lesser severity and duration.

Multiple symptoms, lasting for at least 1 week
Severe enough to interfere significantly with work and social activities or requiring hospitalization

Some symptoms, lasting for at least 4 days
Cause some interference with work and social activities but do not require hospitalization or have psychotic features

So a bipolar disorder may manifest as single manic episode or a manic/hypomanic episode with depression.
Clinical features and MSE

Mood Changes as elation, euphoria or Irritable. The level of goal directed and pleasurable activities increase that are usually reckless and involve risk. Patient is distractible with poor concentration. Speech is rapid and patient jumps from one topic to another. Patient may have delusions mainly grandiose delusions of power, worth or identity. There is decrease in need of sleep and still high levels of energy, increased appetite, increased libido and sexual activity, increased desire for substances. Also the disorder may be comorbid with other general medical conditions as depicted under depression. Comorbid psychiatric conditions and substances are mentioned here.

PREDICTORS OF BIPOLARITY

- Early age of onset with marked psychomotor retardation
- Postpartum depression
- Rapid onset and offset of depressive episodes
- Recurrent depression (5 or more episodes)
- Atypical symptoms (increase in appetite and sleep)
- Bipolar family history
- Antidepressant induced hypomania
- Seasonal episodes

Other psychiatric disorders
Anxiety disorders
Substance use disorders

Drugs
Withdrawal of
Sedatives, alcohol
Prolonged use of
Amphetamines, cocaine
Management

Goals

- Psycho education
- Treat the present episode
- Prevent relapse by maintenance treatment (at least 2 years after last bipolar episode)
- Know when to refer
- Rehabilitation (appropriate economic, social, cultural and educational activities)

If present episode is mania/hypomania

Begin treatment of acute mania with mood stabilizer (lithium, valproate, and carbamazepine) or with antipsychotics*

Consider short-term benzodiazepine* such as diazepam for behavioural disturbance or agitation.

Discontinue any antidepressants.

If present episode is depression but there is history of past bipolar affective disorder or past manic/hypomaniac episode

Begin treatment with a mood-stabilizer

Consider antidepressant combined with mood stabilizer for moderate/severe depression with monitoring of switching to mania. However, referral to a specialist should be done. Consider non-pharmacological management of mild bipolar depression along with mood

Stimulants (methylphenidate)
Steroids
Bromocriptine, levodopa
Baclofen
Opioids
*National formulary of India- Antipsychotics for acute mania (Olanzapine, Haloperidol, Chlorpromazine, Fluphenazine) and short term Benzodiazepines (Diazepam, Lorazepam, Alprazolam, Nitrazepam)

When to refer

- If acute episode is severe and not being controlled at primary health care setting
- A patient with bipolar depression
- Medical emergencies comorbid with Bipolar mood disorder
- Special population like elderly, pregnant women and children and adolescent
- Toxicity with drugs

Primary mood stabiliser can be lithium, valproate or carbamazepine as shown in the table below.
<table>
<thead>
<tr>
<th><strong>Lithium</strong></th>
<th><strong>valproate</strong></th>
<th><strong>Carbamazepine</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Starting dose:</strong> 300</td>
<td><strong>Starting dose:</strong> 500</td>
<td><strong>Starting dose:</strong> 200</td>
</tr>
<tr>
<td><strong>Usual dose:</strong> 600-1200</td>
<td><strong>Usual dose:</strong> 1000-2000</td>
<td><strong>Usual dose:</strong> 400-600</td>
</tr>
<tr>
<td><strong>Levels:</strong> 0.6 – 1.0 mEq / litre</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Mania:</strong> 0.8 – 1.0 mEq / litre</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Maintenance:</strong> 0.6 – 0.8 mEq / litre.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Regular serum level monitoring critical. Monitor TSH and S. creatinine levels also.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Side effects:</strong></td>
<td>Tremors, sedation, weight gain, hepatotoxicity, leukopenia. Watch for hepatitis, pancreatitis, thrombocytopenia,</td>
<td>Tremors, sedation, weight gain, hepatotoxicity, leukopenia, hepatotoxicity. Watch for diplopia, impaired coordination, rash, liver enzyme elevations, Stevens-Johnson syndrome, aplastic anaemia.</td>
</tr>
<tr>
<td>Tremors, sedation, weight gain, hepatotoxicity, leukopenia.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Impaired coordination, polyuria, polydipsia, cognitive problems, cardiac arrhythmias, diabetes insipidus, hypothyroidism</td>
<td></td>
<td></td>
</tr>
<tr>
<td>START only if clinical and laboratory monitoring is available. Effective against the relapse of both mania and depression Educate about lithium toxicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No level monitoring required. Get hepatic status before initiating. Ex-plain the signs and symptoms of blood and liver disorders. Monitor hepatic and blood indices.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Avoid in pregnancy and advice contraception to woman of child bearing age along with folate supplementation. Lithium also associated with birth defects (mainly cardiac) though at a lower rate than antiepileptic</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Avoid in pregnancy and advice contraception to woman of child bearing age along with folate supplementation. Lithium also associated with birth defects (mainly cardiac) though at a lower rate than antiepileptic.
*Carbamazepine and Lithium carbonate are included in National formulary of India while Lithium and Valproate are mentioned in national list of essential medicines.

**Special population** should be referred to psychiatrist. Females of child bearing age on antiepileptic mood stabiliser should receive folate supplementation and be advised to practice contraception. Even for Lithium a risk benefit assessment needs to be done before continuing in pregnancy as it is also associated with teratogenic effects though less than that of antiepileptic mood stabilisers. Thus a referral needs to be done.

- Take plenty of fluids (summers) with no salt restriction
- Reduce or stop lithium temporarily on developing fever, loose motions, vomiting or any condition leading to fluid loss
- ACE inhibitors, diuretics and NSAIDs along with Lithium increase risk of toxicity
- Gastrointestinal upset, dysarthria, ataxia, coarse tremor followed by impaired consciousness, fasciculation, myoclonus, seizures, and coma are signs of toxicity
ANXIETY DISORDERS

INTRODUCTION AND EPIDEMIOLOGY

ANXIETY: diffuse, vague, unpleasant sense of apprehension that is felt as psychic tension and restlessness or autonomic arousal like palpitation, perspiration, headache, chest tightness, abdominal discomfort, inability to sit still. It is usually insidious to an unknown, internal, vague, conflictual threat.

FEAR: response to external, known, definite, non conflictual threat that alerts the individual

Anxiety is normal if it is alerting in response to external stress and leads to optimal performance. However, if anxiety leads to excessive arousal and impairment in the ability to function normally it is pathological and should be treated.

It is said that the 12-month prevalence rate of anxiety disorder is 17.7 percent with women having more prevalence than men. Patients seeking treatment for somatic and cognitive symptoms of anxiety may be most commonly encountered by a GP.

TYPES OF ANXIETY DISORDERS

- Phobic anxiety disorder
- Panic disorder
- Generalised anxiety disorder
- Obsessive compulsive disorder
- Acute stress disorder
- Post-traumatic stress disorder
- Anxiety disorders due to general medical condition and substances
PHOBIC ANXIETY DISORDER

These are by far one of the commonest mental disorders, with females more afflicted than males and onset usually seen in teens.

It can be of 3 types broadly

1. Agoraphobia
2. Social phobia
3. Specific phobia

AGORAPHOBIA

DIAGNOSTIC CRITERIA AND CLINICAL FEATURES

Agoraphobia

Marked and consistent fear or avoidance of at least two of the following situations: crowds; public places; travelling alone; travelling away from home.

Symptoms of anxiety in the feared situation at some time since the onset of the disorder, with at least two symptoms

Autonomic arousal symptoms

Palpitations, Sweating, Trembling, Dry mouth

Symptoms concerning chest and abdomen

Difficulty breathing, Feeling of choking, Chest pain or discomfort, abdominal distress

Symptoms concerning mental state

Feeling dizzy or light-headed. Feelings that objects are unreal (derealisation), or that one's self is unreal (depersonalization), Fear of losing going crazy, Fear of dying.

General symptoms

Hot flushes or cold chills, Numbness or tingling sensations.

Significant emotional distress due to the avoidance or the anxiety symptoms, and a recognition that these are excessive or unreasonable.
SOCIAL PHOBIA

DIAGNOSTIC CRITERIA AND CLINICAL FEATURES

Social phobias

A. Either (1) or (2):

(1) Marked fear of being the focus of attention, or fear of behaving in a way that will be embarrassing or humiliating

(2) Marked avoidance of being the focus of attention or situations in which there is fear of behaving in an embarrassing or humiliating way.

These fears are manifested in social situations (eating or speaking in public; encountering known individuals in public; or entering or enduring small group situations, such as parties, meetings and classrooms)

At least two symptoms of anxiety in the feared situation at some time since the onset of the disorder, as defined in Agoraphobia and in addition one of the following symptoms: Blushing; Fear of vomiting; Urgency or fear of micturition or defecation.

Significant emotional distress due to the symptoms or to the avoidance and recognition that the symptoms or the avoidance are excessive or unreasonable.

SPECIFIC PHOBIA

DIAGNOSTIC CRITERIA AND CLINICAL FEATURES

Specific (isolated) phobias

Either (1) or (2):

(1) marked fear of a specific object or situation other than agoraphobia or social phobia (2) marked avoidance of such objects or situations (animals, birds, insects, heights, thunder, flying, small enclosed spaces, sight of blood or injury, injections, dentists and hospitals) B. Symptoms of anxiety in the feared situation at some time since the onset of the disorder, as defined in agoraphobia.

Significant emotional distress due to the symptoms or the avoidance, and a recognition that these are excessive or unreasonable.

Symptoms are restricted to the feared situation, or when thinking about it. It can be: animal type (e.g. insects, dogs); nature-forces type (e.g. storms, water); blood, injection and injury type; situational type (e.g. elevators, tunnels); other type
DIFFERENTIALS
Phobias need to be differentiated from normal shyness, substance induced anxiety symptoms, central nervous symptoms pathologies. Schizophrenia patients may have specific or social phobia but as part of their broader psychosis.

PANIC DISORDER
The lifetime prevalence of this disorder is 1-4% with women being twice more likely to be affected. Mean age of presentation is 25 years.

DIAGNOSTIC CRITERIA AND CLINICAL FEATURES

Recurrent panic attacks that are not consistently associated with a specific situation or object and often occurring spontaneously

The panic attacks are not associated with marked exertion or with exposure to dangerous or life-threatening situations.

A panic attack is characterized by all of the following: (a) it is a discrete episode of intense fear or discomfort; (b) it starts abruptly; (c) it reaches a crescendo within a few minutes and lasts at least some minutes; (d) at least four symptoms of anxiety must be present as described in phobic disorder above

Panic disorder is characterised by unexpected discrete episodes of fear and physical symptoms of anxiety that swarm in a few minutes and subside in next few. Usually they are mistaken for acute cardiac events and patients may have several emergency visits before being diagnosed as panic disorder as seen in this case. So a detailed history and thorough medical workup may help bring upon timely intervention for the patient. Also, this patient is self-treating his anticipatory anxiety with alcohol. Substance abuse may be comorbid with anxiety disorders.

COMMON DIFFERENTIALS
Mostly panic attacks are confused with acute cardiac syndromes like anaemia, cardiac failure, arrhythmias or angina and most of the initial workup is done on that line. Respiratory emergencies like asthma should be ruled out. Endocrine disorders like thyrotoxicosis, Pheochromocytoma, hypoglycaemia, menopausal or premenstrual syndromes may manifest as extreme anxiety. Some drug intoxication or withdrawal states or allergic states like anaphylaxis should be ruled out
GENERALISED ANXIETY DISORDER (GAD)

They constitute one fourth of all anxiety disorder with onset seen in late adolescence and more in females.

DIAGNOSTIC CRITERIA AND CLINICAL FEATURES

A period of at least six months with prominent tension, worry and feelings of apprehension, about every-day events and problems.

Four symptoms out of the following list

**Autonomic** arousal symptoms

Palpitations, Sweating Trembling, Dry mouth

Symptoms concerning **chest and abdomen**

Difficulty breathing, Feeling of choking, Chest pain or discomfort, abdominal distress

Symptoms concerning **mental state**

Feeling dizzy or light-headed. Feelings that objects are unreal (derealisation), or that one’s self is unreal (depersonalization), Fear of losing going crazy, Fear of dying.

**General** symptoms

Hot flushes or cold chills, Numbness or tingling sensations.

Symptoms of **tension**

Muscle tension or aches and pains, Restlessness and inability to relax, Feeling keyed up, or on edge, or of mental tension, A sensation of a lump in the throat, or difficulty with swallowing.

Other **non-specific** symptoms

OBSESSIVE COMPULSIVE DISORDER

It is the fourth most common psychiatric disorder with equal prevalence in men and women and mean age of onset in 20 years.
DIAGNOSTIC CRITERIA AND CLINICAL FEATURES

Either obsessions or compulsions (or both), present on most days for a period of at least two weeks.

Obsessions (thoughts, ideas or images) and compulsions (acts) share the following features

1. They are acknowledged as originating in the mind of the patient and are not imposed by outside persons or influences.
2. They are repetitive and unpleasant, and at least one obsession or compulsion must be present that is acknowledged as excessive or unreasonable.
3. The subject tries to resist them but at least one obsession or compulsion must be present which is unsuccessfully resisted.
4. Carrying out the obsessive thought or compulsive act is not in itself pleasurable.

The obsessions or compulsions cause distress or interfere with the subject's social or individual functioning, usually by wasting time.

ACUTE STRESS DISORDER

A young male patient was brought to the OPD as he was very agitated after hearing the news of death of his wife suddenly in an accident few minutes back. Relatives reported on hearing the news he did not respond and after a while started saying irrelevant things and occasionally misnamed relatives. On being asked he even abused and became violent with his brother. Patient appeared confused, disoriented, dizzy and restless and every now and then used to pace around in the room. He had tachycardia, flushing and sweating and shortness of breath. He had no past history of any illness.

Exposure to an exceptional mental or physical stressor.

Followed by an immediate onset of symptoms (within one hour) (Two groups of symptoms) are given

1. The criteria for symptoms as mentioned in generalized anxiety disorder

2. Withdrawal from expected social interaction; narrowing of attention; apparent disorientation; anger or verbal aggression; despair or hopelessness; purposeless over-activity; uncontrollable and excessive grief (judged by local cultural standards).

If the stressor is transient or can be relieved, the symptoms must begin to diminish after not more than eight hours. If the stressor continues, the symptoms must begin to diminish after not more than 48 hours.
Post-traumatic stress disorder

Exposure to a stressful event or situation of exceptionally threatening or catastrophic nature, which is likely to cause pervasive distress in almost anyone.

Persistent remembering or reliving the stressor by intrusive flash backs, vivid memories, recurring dreams, or by experiencing distress when exposed to circumstances resembling or associated with the stressor.

Avoidance of circumstances resembling or associated with the stressor

Either (1) or (2):

1. Inability to recall, either partially or completely, some important aspects of the period of exposure to the stressor

2. Persistent symptoms of increased psychological sensitivity and arousal shown by any two of the following: difficulty in falling asleep, irritability, difficulty in concentrating; hyper-vigilance; exaggerated startle response.

Symptoms occurred within six months of the stressful event, or the end of a period of stress.

These symptoms are seen in patients who are exposed to some life threatening event like natural calamities followed by a triad of re-experiencing, avoidance and increased arousal.

**MANAGEMENT**

<table>
<thead>
<tr>
<th>Pharmacological</th>
<th>Non pharmacological</th>
</tr>
</thead>
<tbody>
<tr>
<td>✤ Short term treatment with benzodiazepines (BZD’s)</td>
<td>✤ Psycho education about the disorder</td>
</tr>
<tr>
<td>✤ Withdraw BZD’s after initial 2-4 weeks.</td>
<td>✤ Exploring the concomitant stressors and addressing the worries in a reassuring, empathic and neutral manner</td>
</tr>
<tr>
<td>✤ Always taper BZD’s</td>
<td>✤ Cognitive behavioural therapy (CBT) ( include relaxation, biofeedback and addressing cognitive distortions)</td>
</tr>
<tr>
<td>✤ Long term treatment with antidepressants preferably Selective Serotonin Reuptake Inhibitors (SSRI’s) followed by tricyclic antidepressants and MAO inhibitors</td>
<td>✤ Supportive psychotherapy</td>
</tr>
<tr>
<td></td>
<td>✤ Insight oriented psychotherapy</td>
</tr>
<tr>
<td></td>
<td>✤ Regular exercises, yoga, breathing exercises may be advised along with</td>
</tr>
</tbody>
</table>
COMORBID CONDITIONS WITH ANXIETY DISORDERS

<table>
<thead>
<tr>
<th>General medical conditions</th>
<th>Other psychiatric disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Endocrine</strong></td>
<td>Substance use disorders</td>
</tr>
<tr>
<td>Hyperthyroidism, hypoparathyroidism, Cushing’s syndrome, hypoglycaemia, Pheochromocytoma, Addison’s disease</td>
<td>Schizophrenia</td>
</tr>
<tr>
<td></td>
<td>Mood disorders</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>Drugs</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>--------------------------------------------</td>
</tr>
<tr>
<td>Asthma, hyperventilation</td>
<td></td>
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<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Withdrawal of</td>
</tr>
<tr>
<td>Anaemia, cardiac failures, mitral valve prolapse, hypertension, angina, myocardial infarction</td>
<td>Alcohol, opiates, sedatives, hypnotics</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurological</td>
<td>Intoxication</td>
</tr>
<tr>
<td>Infarcts, haemorrhage, epilepsy, migraine, Wilson’s disease, basal ganglia disease like Sydenham’s chorea and Huntington’s disease(OCD)</td>
<td>Nicotine, amphetamine, cocaine, theophylline, amyl nitrite, hallucinogens</td>
</tr>
<tr>
<td></td>
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<tr>
<td>Metabolic</td>
<td></td>
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<tr>
<td>Dyselectrolytemia, renal/hepatic failures</td>
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<td></td>
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<tr>
<td>Nutritional</td>
<td></td>
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<tr>
<td>Vitamin B12, Folate deficiency</td>
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<td></td>
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<tr>
<td>Tumours</td>
<td></td>
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<tr>
<td>Cerebral/systemic</td>
<td></td>
</tr>
</tbody>
</table>
The following table is an outline for the use of drugs for treating anxiety disorders.

<table>
<thead>
<tr>
<th>Group of drug</th>
<th>Dosages (mg/day)</th>
<th>Adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Selective serotonin reuptake inhibitors (SSRI’s)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sertraline</td>
<td>50-200</td>
<td>Sleep disturbance, gastrointestinal side effects, headache, anxiety, prolonged bleeding time, hyponatremia, sexual dysfunction, discontinuation syndrome</td>
</tr>
<tr>
<td>Escitalopram*</td>
<td>10-20</td>
<td></td>
</tr>
<tr>
<td>Paroxetine</td>
<td>20-50</td>
<td></td>
</tr>
<tr>
<td>Citalopram</td>
<td>20-40</td>
<td></td>
</tr>
<tr>
<td>Fluoxetine*</td>
<td>20-60</td>
<td></td>
</tr>
<tr>
<td>Fluvoxamine</td>
<td>100-300</td>
<td>sedation, hypotension, cardiac side effects mainly prolonged QT interval, anticholinergic effects, weight gain</td>
</tr>
<tr>
<td><strong>Tricyclic antidepressants (TCA)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clomipramine</td>
<td>75-300</td>
<td></td>
</tr>
<tr>
<td>Imipramine*</td>
<td>75-300</td>
<td></td>
</tr>
<tr>
<td>Amitriptyline</td>
<td>75-200</td>
<td></td>
</tr>
<tr>
<td><strong>Benzodiazepines (BZD’s)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clonazepam</td>
<td>0.5-2</td>
<td>On long term use-Tolerance, dependence and withdrawal, ataxia, dizziness, daytime drowsiness, amnesia, in overdose respiratory depression</td>
</tr>
<tr>
<td>Alprazolam*</td>
<td>0.5-4</td>
<td></td>
</tr>
<tr>
<td>Lorazepam*</td>
<td>1-4</td>
<td></td>
</tr>
<tr>
<td>Diazepam*</td>
<td>5-15</td>
<td></td>
</tr>
<tr>
<td><strong>Beta-blockers</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Propranolol</td>
<td>10 – 20 mg bid or tds</td>
<td>Hypotension, bradycardia, worsening of asthma and hypoglycaemia, GI side effects</td>
</tr>
</tbody>
</table>

*In National formulary of India

**WHEN TO REFER**

- If 2 adequate trial of treatments (antidepressant(usually 8-16weeks) or psychotherapy or combination) fails
- Need for specialised psychological intervention and lack of resources
- Severe or chronic disorders or those with comorbid psychiatric conditions that need multidimensional approach
- Secondary depressive features are severe enough to warrant independent attention
SOMATOFORM DISORDERS

Introduction

Somatoform Disorders are a broad group of illnesses that have bodily signs and symptoms as a major component. According to ICD – 10 [International Classification of Diseases – 10th revision], Somatoform Disorders are described as ‘Repeated Presentation of physical symptoms together with persistent requests for medical investigations, although patients have been reassured by their Physicians that the symptoms have no physical basis’. In people who have a somatoform disorder, history and physical examination do not indicate the presence of a medical condition. Medical test results are either normal or do not explain the person’s symptoms. Patients are convinced that their symptoms result from some type of undetected and untreated bodily derangement. These complaints are not imaginary and patients do actually experience these symptoms. These disorders encompass mind – body interactions in which the brain sends various signals that impinge on the patient’s awareness, indicating a severe problem in the body.

Epidemiology

Somatoform Disorders are more common in females as compared to males with approximate ratio of 4: 1. The overall prevalence of these disorders varies from 1-5 %. It is found in 5-10 percent of patients presenting to General Practitioners, hence it is important to identify at primary level. Usually, the symptoms begin appearing during adolescence, and patients are diagnosed before the age of 30 years. They are more common in people with low education & low income. Symptoms are sometimes similar to those of other illnesses and may last for several years before initial presentation. Symptoms may occur across cultures and gender.

Clinical Features

The ICD – 10 classifies Somatoform Disorders into various categories with a common underlying pre-occupation with bodily signs and symptoms. These categories are Somatization disorder, Hypochondriacal disorder, Un-differentiated somatoform disorder, Somatoform autonomic dysfunction, Persistent somatoform pain disorder and others.

Somatization disorder - The main features are multiple, recurrent, and frequently changing physical symptoms, which have usually been present for several. The patients believe they have been sick most of their lives. Most patients have a long and complicated history of contact with both primary
and specialist medical services, during which many negative investigations or operations may have been carried out. Symptoms commonly involve gastrointestinal sensations (pain, belching, regurgitation, vomiting, nausea, etc.), abnormal skin sensations (itching, burning, tingling, numbness, soreness, etc.), headache and blotchiness. Sexual, menstrual and neurologic problems may also be reported.

The course of the disorder is chronic and fluctuating. People with this disorder tend to be referred to various specialists, and have many tests and investigations. Dependence upon or abuse of medication (usually sedatives and analgesics) is often found.

**Hypochondriasis** - This is a disorder where people fear that minor symptoms may be due to a serious disease. For example, that a minor headache may be caused by a brain tumor, or a mild rash is the start of skin cancer. People with this disorder have many such fears and spend a lot of time thinking about their symptoms.

**Body dysmorphic disorder** - People with this disorder are obsessed and exaggerate a physical flaw. Patients may also imagine a flaw they don’t have. The preoccupation may involve any part of the body such as wrinkles, hair, or the size or shape of the eyes, nose, or breasts. Some people consult a cosmetic surgeon to have the imagined or trivial defect corrected.

**Pain disorder** - The predominant complaint is of persistent, severe, and distressing pain, which cannot be explained fully by a physiological process or a physical disorder. Pain usually starts with a psychological stress or trauma. For example, they develop an unexplained, chronic headache after a stressful life event.

**Somatoform autonomic dysfunction** - The symptoms are presented by the patient as if they were due to a disorder of a system or organ that is under autonomic innervations i.e. the cardiovascular, gastrointestinal, or respiratory system although there is no evidence of a significant disturbance. Presenting symptoms usually are palpitations, sweating, flushing, tremor, sensations of fleeting aches and pains, burning, heaviness, tightness, and sensations of being bloated.

**Somatoform disorder not otherwise specified** - People with this type do not meet the full criteria for any other somatoform disorders. Example includes pseudocyesis (mistaken belief of being pregnant based on other signs of pregnancy, including an expanding abdomen; feeling labor pains, nausea, fetal movement etc.)

Somatoform Disorders are commonly associated with other psychiatric disorders, namely Personality Disorders, Substance related Disorders, Generated Anxiety Disorder and Phobia. Suicidal threats are common but actual suicide is rare. Patient’s medical histories are usually vague, inconsistent and confusing. Patients classically described their complaints in a dramatic, emotional and exaggerated fashion. They would emphasize the need for further investigations or examinations.

**Assessment and Diagnosis**

The general practitioners and medical officers play a vital role in the management of patients with somatoform disorders. They act as the 'gatekeeper' and have the potential to limit the patient’s access to specialized hospital resources, many of which are not only expensive, but also carry the
possibility of iatrogenic harm. Appropriate and timely diagnosis in primary care combined with collaborative psychiatric and medical interventions may decrease significant long-term morbidity and suffering.

The assessment aims to clarify patient's somatic symptoms and their effects on daily life, Understand what patient wants from treatment, Elicit fears and beliefs about illness, Exclude organic disease, Identify relevant psychosocial Stressors, Identify co-morbid psychiatric disorders, Risk of suicide and Dependence on medications. Previous referrals, investigations and treatment history needs detailed assessment (Box - 1). Doctors may need to perform laboratory tests to rule out other possible causes before they diagnose somatoform disorder.

**Box - 1: Objectives in assessment interview**

- Clarify patient's complaints
- Understand what patient wants
- Elicit fears and beliefs about illness
- Previous referrals, investigations and treatments
- Exclude organic disease
- Identify relevant psychosocial Stressors
- Order relevant investigations
- Identify drug misuse

**Differential Diagnosis**

- Malingering (Intentionally producing symptoms to achieve an environmental goal)
- Factitious disorder (Fabrication of symptoms or self-inflicted injury to assume the sick role)
- Psychosomatic illness (A real medical condition affected by stress or psychological factors)
- Depression

**Treatment of Somatoform Disorders**

Somatoform disorders are difficult to treat as patients often cling to the belief that their symptoms have an underlying physical cause. Reassurance by a doctor does not usually help as they feel their doctors cannot find the cause for their symptoms.
A strong doctor-patient relationship is a key to getting help in patients with somatoform disorders. Seeing a single health care provider with experience managing somatoform disorders can help cut down on unnecessary tests and treatments (Box - 2).

The focus of treatment is on improving daily functioning, not on managing symptoms. Stress reduction is often an important part of getting better. Stress management techniques, Counseling for family and friends, Promotion of self-care activities, Relaxation and breathing exercises, Lifestyle change and Occupational Counseling may be useful and can be done at primary level. Medications do not have much of a role except if the disorder is associated with underlying mental illnesses such as depression, anxiety or substance abuse. Still, antidepressants such as amitriptyline, imipramine and selective serotonin reuptake inhibitors (SSRIs) along with short-term antianxiety agents such as benzodiazepines have been found effective.

**Box - 2 : Specific treatments for somatoform disorders**

- *General advice*
  - Lifestyle change
  - Relaxation

- *Drug treatments*
  - Antidepressants
  - Anti-Anxiety drugs (short duration)

- *Occupational and social*
  - Occupational counseling
  - Problem-solving for social problems

- *Psychological treatments*
  - Cognitive-behavioral and other therapies

- Regular appointments at fixed, pre-arranged intervals
- Minimize use of drugs
When to Refer?

Some important indicators that should guide the primary care provider to refer to psychiatrists include:

- Diagnostic dilemma
- Chronic & Severe illness
- Suicidal threats and attempts.
- Co-morbid Psychiatric problems like severe depression and substance abuse.
- No relief on initial treatment
EMOTIONAL AND BEHAVIOURAL PROBLEMS IN
CHILDREN & ADOLESCENTS

INTRODUCTION
The child is continually developing. Symptoms & Behaviour problems change with developmental changes, as do emotional needs. The developmental stage can influence the presentation, significance & course of a psychiatric disorder. It is essential that all individuals involved in the care of children have at least a basic understanding of ‘normal’ and ‘abnormal’ childhood experience and behaviour.

Transient symptoms and behavioural disturbances are common in children of all ages. Parents are becoming increasingly aware of emotional health problems in Children and seek help for various concerns regarding their children.

Parents may seek help for various concerns regarding their children. These concerns may include children not able to sit still, not able to pay attention, not able to engage in verbal, non-verbal, or social communication with others, seems to be withdrawn, depressed, angry, and violent, poor scholastic performance, drug abuse etc.

The major epidemiological features of childhood psychiatric disorder are:
- Boys are affected twice as often as girls for most diagnoses.
- Commonest diagnoses are emotional and conduct disorder.
- Disorders are commoner in children with mental retardation, epilepsy or medical illness.

ETIOLOGY IN CHILD PSYCHIATRY
- The same broad range of etiological factors operates in childhood as in adulthood.
- There is a genetic component to most disorders, mediated partly through its influence on intelligence & temperament. The disorders involve interplay of genetic and environmental factors.
- The major environmental factors are the family & social circumstances. The family factors include parenting styles, parental conflict/ separation and social factors include deprivation. A deprived child is one who is: Without proper parental care or control, subsistence, education as required by law, or other care or control necessary for his physical, mental, or emotional health or morals, has been placed for care or adoption in violation of law, has been abandoned by parents or other legal custodian, is without a parent, guardian, or custodian.
- Other factors like medical disorder (Epilepsy) might also increase the risk.

RANGE OF CHILDHOOD PSYCHIATRIC DISORDERS
The Mental Health problems affecting children and adolescents may be considered from a number of perspectives. The disorders listed below are identified as priority areas based on
higher frequency of occurrence and degree of associated impairment, therapeutic possibilities and long-term care consequences.

Internalising behavioural problems are characterised by an overcontrol of emotions and include being withdrawn, demanding attention, being too dependent or clingy and feeling worthless or inferior.

**DESCRIPTION OF CHILDHOOD PSYCHIATRIC DISORDERS**

(A) **LEARNING DISORDERS:**

Learning disorders have a high incidence and prevalence with serious implications for future productivity. Learning disorders are diagnosed when the individual’s academic achievement in reading, mathematics, or writing is substantially below what would be expected for age, schooling and intellectual ability (APA, 1994). Specific Learning Disorder (SLD) is a disorder in one or more of the basic psychological processes involved in understanding or in using spoken or written language. The disability may be exhibited as an imperfect ability to listen, think, speak, read, write, spell, or do mathematical calculations.

**Types of Learning Disorders:**

| Reading disorders: | • Reading skills significantly below expectancy  
|                   | • Significantly interferes with academic achievement or activities requiring reading skills |
| Mathematics disorders: | • Mathematics skills significantly below expectancy  
|                     | • Significantly interferes with academic achievement or activities requiring mathematics skills |
| Disorder of written expression | • Writing skills significantly below expectancy  
|                               | • Significantly interferes with academic achievement or activities requiring mathematics skills |
The most common treatment for learning disabilities is special education. Other professionals such as speech and language therapists also may be involved. This usually requires referral to a higher center.

(B) PERVASIVE DEVELOPMENTAL DISORDERS

These are Developmental disorders where the development follows a deviant path and there is a deviation in development of communication and socialization.

The disorders included in this category are

- Autism
- Aspergers disorder
- Retts disorder
- Other disintegrative disorders
- PDD unspecified

AUTISM:

Autism is a pervasive developmental disorder and characterized by significant problems in all three domains of:

- Communication
- Social interactions
- And stereotyped patterns of behaviour

Characteristic features:

- Difficulties with social interactions
- Marked impairment in the use of non-verbal behaviours, such as eye-to-eye gaze, facial expression, body postures, and gestures to regulate social interaction
- A decrease or lack of spontaneous seeking to share enjoyment, interests, or achievements with other people
- Decreased or lack of social or emotional reciprocity
- Failure to develop peer relationships appropriate to developmental level
- Impairments in communication
- Delay or total lack of development of spoken language
- Stereotyped or repetitive use of language or idiosyncratic language
- Lack of varied, spontaneous make-believe play or social imitative play appropriate to developmental level.

Common co-occurring conditions in autism:

- Mental Retardation: up to 75% of people with autism have mental retardation
- Seizures: 25% of autistic individuals also develop seizures, some in early childhood and others as they go through puberty

Treatment:

- Behavioural, psychotherapeutic as well as pharmacologic approaches can be used to address numerous problems, but unfortunately there is no cure for the core disorder
- The goal of treatment is not merely to lessen symptoms, but to help the individual achieve the maximally feasible quality of life
MENTAL RETARDATION/ INTELLECTUAL DISABILITY:

- Mental retardation is one of the most prevalent of the developmental disorders, occurring in approximately 1 – 3% of school-aged children and estimated to be 2.5%.
- Mental Retardation or Intellectual disability involves impairments of general mental abilities that impact adaptive functioning in three domains, or areas. These domains determine how well an individual copes with everyday tasks:
  - The conceptual domain includes skills in language, reading, writing, math, reasoning, knowledge, and memory.
  - The social domain refers to empathy, social judgment, interpersonal communication skills, the ability to make and retain friendships, and similar capacities.
  - The practical domain centers on self-management in areas such as personal care, job responsibilities, money management, recreation, and organizing school and work tasks.

<table>
<thead>
<tr>
<th>CATEGORY</th>
<th>IQ RANGE</th>
<th>LEVEL OF FUNCTIONING</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild MR</td>
<td>50-70</td>
<td>• Individuals educable.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Can be minimally self supporting.</td>
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<tr>
<td></td>
<td></td>
<td>• Can acquire basic social and vocational skills.</td>
</tr>
<tr>
<td>Moderate MR</td>
<td>35-49</td>
<td>• They are trainable.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Able to perform academic activities comparable to a child in Grade two.</td>
</tr>
<tr>
<td>Severe MR</td>
<td>20-34</td>
<td>• Seriously impaired in their motor and speech development</td>
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<tr>
<td></td>
<td></td>
<td>• Only learn basic language and practice hygiene after age six.</td>
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<tr>
<td></td>
<td></td>
<td>• Usually permanently dependent on others and therefore spend their lives with family,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• in community homes or in institutions.</td>
</tr>
<tr>
<td>Profound MR</td>
<td>Less Than 20</td>
<td>• Fully reliant on caregivers.</td>
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<tr>
<td></td>
<td></td>
<td>• Often diagnosed with a neurological disorder and epilepsy, spasticity and mutism.</td>
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</tbody>
</table>
Etiology:

Mental retardation may be the end result of one of the following categories of risk.

- **Biomedical** - these are factors that have a deleterious impact on the child’s central nervous system
- **Social** - inadequacies in the social and/or family environment can diminish cognitive and social growth and development
- **Behavioral** - these include trauma, maternal substance abuse
- **Educational** - the availability and quality of training programmes can affect intellectual development and influence whether or not the child functions in the range of mental retardation.
- Interaction between risk factors

Assessment of mental retardation:

The diagnosis of the cause of mental retardation involves pediatricians and pediatric neurologists.

The assessment of a child with suspected mental retardation encompasses:

- Making the diagnosis
- Evaluating concurrent psychiatric problems

Management:

Mental retardation is rarely treatable and management is aimed at maximizing potential and quality of life, and treating concurrent psychiatric problems. The physician should help the family to prepare for the future. The clinician should ensure, that over time, the family accepts the prognosis and has realistic expectations for the child’s future.

(D) HYPERKINETIC DISORDER:

- It is also called **ADHD**: Attention Deficit Hyperactivity Disorder.
- Prevalence of this disorder is estimated to be around 3-5% in pre-pubertal children.
- It is much more common in boys than girls (4:1)
- The onset is usually in early childhood (before age 7) but the problem is usually identified when the child starts going to school.

**Characteristic features:** This disorder is characterized by: Hyperkinetic behaviour and poor attention span.

<table>
<thead>
<tr>
<th>The main feature is hyperkinetic behaviour:</th>
<th>The second characteristic is poor attention span</th>
</tr>
</thead>
<tbody>
<tr>
<td>As children they are:</td>
<td>These children do not concentrate on any task and leave most tasks unfinished.</td>
</tr>
<tr>
<td>- Restless and cannot sit still.</td>
<td>- These patients may have many other associated symptoms like impulsivity, emotional lability, poor scholastic progress and antisocial behaviour.</td>
</tr>
<tr>
<td>- Fiddle with everything around</td>
<td></td>
</tr>
<tr>
<td>- Emotionally excitable</td>
<td></td>
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<tr>
<td>- Show episodes of rage or crying</td>
<td></td>
</tr>
<tr>
<td>- Impulsive</td>
<td></td>
</tr>
<tr>
<td>- Show temper tantrums</td>
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</table>
Etiology:
- Genetic factors seem to play some part.
- Majority of these patients do show developmental delays. Soft neurological signs are common.
- Others believe that the disorder occurs due to maturational lag of the central nervous system. The condition improves with maturation.
- Psychosocial factors may play some part. This disorder is more common in orphanages where proper rearing atmosphere is not available.

Diagnosis:
- Based on presence of hyperactivity and poor attention span, which is out of proportion of the age and intelligence of the child.
- Some children may show conduct disorders in form of truancy, lying, aggression etc.

Course and Prognosis:
- About three-fourths improve by adolescence.
- Some may continue to have poor attention span.
- Others may continue showing both hyperactivity and poor attention.
- However, a small group may develop dissociative personality disorder and others may persist with difficulties in social adaptation.

MANAGEMENT
- Pharmacotherapy:
  - The CNS stimulants: These drugs reduce hyperactivity and improve attention span.
  - Dextroamphetamine and Methylphenidate are the drug of choice. They are to be given in the morning and at noon because nighttime dose may produce sleep difficulty.
  - Dextroamphetamine is given in a dose of 5-10 mg/day.
  - Methylphenidate in doses of 0.25-1 mg/kg/day is effective.
  - Non-stimulant: Atomoxetine (1-1.4 mg/kg qd)

- Psychological Treatment
The parents and teachers are advised not to retaliate against the child but their hyperactivity could be channelized into outdoors sports and their poor attention can be improved by appropriate educational technology.

(E) CONDUCT DISORDER:
Characterized by repetitive and persistent dissociative and aggressive/defiant behaviour. This behaviour should be persistent and should not be limited to one or two situations. 0.5 to 1% children may show conduct disorder before 18 years of age.

Etiology:
- Genetic Factors
- Familial Factors
iii) Socio-cultural Factors
iv) Psychological Factors
v) Neurological Factors

Clinical Features
These children may show any one or more of the following:
- excessive level of fighting or bullying;
- cruelty to animals or other people;
- fire setting;
- stealing;
- repeated lying;
- truancy from school and running away from home
- frequent and severe temper tantrums; defiant provocative behavior and persistent severe disobedience

Conduct disorders
Conduct disorders can be further subdivided into the following:

a) Conduct disorder confined to family context - Dissocial acts in relation with their family members
b) Unsocialized Conduct Disorders – These children find it difficult to develop enduring relationship with peers. They are usually isolated.
c) Socialized Conduct Disorder – These children become part of a delinquent group and indulge in antisocial activities as described earlier. Basically these children are able to form stable relationship with other children. They may establish relationships with normal children or with children from delinquent gangs.

Management:
- Conduct disorders require psychological management. Following principles are to be kept in mind:
  - If conduct disorder is associated with family disruption or poor parenting, the child may have to be shifted to some other place or a children\'s home
  - Family should always be involved and attempt should be made to help the family to provide consistent upbringing
  - These children may be involved with a group of normal children which may provide them proper role models to emulate
  - Behaviour modification with positive reinforcement may be helpful
- Medication may be needed to control aggressiveness or hyperactivity. Haloperidol in small doses & anticonvulsant (carbamazepine) may be helpful.

(F) DISORDER OF SOCIAL FUNCTIONING WITH ONSET SPECIFIC TO CHILDHOOD AND ADOLESCENCE

These disorders usually develop in response to disturbed environment. Common disorders included are:

- Non-Organic Enuresis: Majority of children become dry by 4 to 5 years. Enuresis is seen in around 1% of children after this age. Enuresis is characterized by involuntary voiding of urine by
day or night. If it is caused by neurological or other organic factors then it is called organic enuresis. Enuresis is divided into primary and secondary:

**Etiology**
Definite etiology is not known. Genetic factors may play a role. Some children may have excessive production of urine due to reduced activity of antidiuretic Hormone. Psychological factors are more important in secondary enuresis. In primary enuresis, reactive psychological problems may lead to poor self-image and familial tensions.

**Clinical Picture**
These children wet the bed at night or in the daytime. The frequency can be varying from daily to once or twice a week. These children usually become self-conscious and tense. They may avoid going to places where they have to pass the night. Family tensions may also develop.

**Differential Diagnosis**
Organic causes of enuresis like spina-bifida-occult, cystitis, seizures etc. should be excluded.

**Management**
- Behavioural treatment has been found effective. The children should be made to void urine before going to bed. Evening intake of fluid should be restricted. The child may be woken up in the middle of night and made to pass urine..
- **Pharmacology:** Imipramine in dosage of 25 to 50 mg at bedtime reduces enuresis. But many children relapse after stopping the drug.
- **Psychological Treatment:** This is effective only in secondary enuresis. These children should be helped to overcome their anxiety and feelings of insecurity by providing them stable parental relationship.

**Non-Organic Encopresis:**
This is characterized by passing of stools at inappropriate places even after the age of bowel control has been attained (5 years). These children may pass stool in their underwear. This condition should be differentiated from organic encopresis e.g. overflow incontinence resulting from constipation and, ganglionic megacolon (Hirschprung’s disease). These children do not have any desire to defecate yet they may have incontinence. Encopresis is usually associated with some other psychiatric disorders like pervasive developmental disorders.

**Management:**
These children are usually very tense and families are disturbed. The tension should be eased out by explaining to them that this is a disorder and we should help the child to have change of underwear’s without much fuss. Behavioural training is useful. Treatment of associated psychiatric condition can be helpful.
G. Emotional Disorders (with specific onset to childhood)

Separation anxiety disorder

Anxiety on separation from a primary care giver is a developmentally normal phenomenon in children less than 1 yr and may last until 2 yrs of age.

When developmentally inappropriate and excessive anxiety concerning separation from home or primary care giver occurs, it is referred to as Separation Anxiety Disorder.

Clinical features:

Its onset may be during preschool years or commonly between age of 6-7 yrs. The onset has to be prior to 18 yrs of age

The anxiety is expressed as:

- Excessive distress when separation occurs
- Excessive worry about anticipated separation
- Reluctance to be in a situation in which separation is anticipated. Eg: School, sleeping alone
- Physical symptoms as stomachache, headache etc.

Management:

- Detailed assessment of child’s temperament, Mothers attitude towards the child and their bonding as well as the psychosocial background in the family
- Multimodal treatment is advised – includes CBT, family education, and psychosocial intervention with the family.
- Graded contact as a form of behaviour modification

SCHOOL REFUSAL

Many children try to avoid going to school from time to time. Some do it regularly. School refusal is not a psychiatric disorder, but it is a common cause of referral to child psychiatrists and is frequently attributable to an emotional disorder.

ASSESSMENT OF SUSPECTED SCHOOL REFUSAL

- Why is the child absent from school?
- What does the school refusal reflect?
  Reluctance to leave home (i.e. secondary to separation anxiety)
  A specific ‘school-phobia’ (e.g. of getting there or of being bullied)
  A more generalized disorder (e.g. social phobia, depressive disorder)
What are the other factors affecting the presentation of management?
Recent life events (e.g. bereavement)
Recent events at school (e.g. change of class)
Parental characteristics: (e.g. overprotective).

Management is aimed at a rapid return to school before avoidance is too ingrained. Sometimes a graded re-exposure is needed. Address any specific fears of stresses, and treat any associated psychiatric disorder.

The prognosis in younger children is good. In older children, the problem may become prolonged and other psychiatric problems may emerge.

CHILDHOOD AND ADOLESCENT DEPRESSION

Childhood major depressive disorder (MDD) is characterized by a significant, often recurrent emotional and behavioral change from baseline to a dysphoric or irritable mood, loss of pleasure or fun, and with decreased functioning at home, school and with peers.

Incidence figures are hard to measure in this population.

- Infants and preschoolers- 1%. It mainly presents as failure to thrive, as well as attachment, separation and behavioral problems
- School age children- 2%. Closer fit to adolescent/ adult criteria. It typically has a more protracted, recurrent or severe course. Suicide attempts and completions uncommon to rare.
- Adolescents- 5% or more. Presents more like the adult syndrome and typically require evaluation of suicidality and substance use.

Etiology and contributing factors

These include genetic factors (biological offspring’s of depressed parents more likely to have depression), environmental factors, organic factors, developmental and transactional factors.

Making the diagnosis-

- Infant/ toddler- both the persistently passive, unresponsive infant and the irritable, unsoothable, crying infant may suffer from the mood dysregulation associated with depression. Their clinical presentation may evolve into a quiet, inhibited toddler with arrested social development or an overactive, impulsive and irritable preschooler.
- School age- in school age children with MDD there may be humiliation, defeat, irritability and self-doubt. The child may become sad, isolated, rejected and accident-prone. Temper
tantrums or morbid preoccupations with bodily injury, illness, abandonment or death may emerge. There may be multiple somatic complains.

- Adolescents- they may have difficulty containing intense negative feelings. They may have direct negative actions inwards towards themselves by disregarding food, sleep and hygiene. Some turn to alcohol and drugs or engage in self-destructive behavior. Adolescents with MDD often appear irritable, angry and resentful. This age group should be given special attention considering the harmful behavior they may indulge in.

**Management**

Mild or moderate cases of depression can be safely cared for as outpatients. Urgent psychiatric consultation is indicated when the child or adolescent might be acutely suicidal, abusing substances, having psychotic features or otherwise difficult to manage. The treatment strategies include-

- Medications –SSRI’s (Fluoxetine) are the drugs of choice for this population, but they have to be used with caution
- Cognitive behavior therapy
- Family therapy
- Environmental manipulation
- Parental guidance.

**Psychological issues related to Adolescence:**

Adolescence is not just a blend of childhood and adulthood; it is a stage with unique biological and social characteristics of its own. Biological factors defining adolescence includes the achievement of sexual maturity as well as the physical and cognitive changes resulting from hormonal shifts. Behavioural problems and disorders during this period of “stress and storm” often represent exaggerations or unresolved versions of the normal development tasks of adolescence. Some of the psychological and social tasks of adolescence include: identity formation (a strong sense of differentiated self in the individual and includes a social/sexual/work and moral identity), self determination, and the dual process of exploration (“keeping options open”) and commitment. Some societies provide structured rituals to assist adolescent with the transition from childhood to adulthood.

Disorders of adolescence are classified in terms of:

- Residual childhood problems (e.g. conduct disorder)
- Problems of adolescent transition (eating disorder, Para suicide, drug abuse)
- Early adult disorders (e.g. Schizophrenia, bipolar disorder, OCD)
GENERAL PRINCIPLES OF MANAGEMENT IN CHILD PSYCHIATRY

Management of children with psychiatric disorder is based upon several principles:

i. Take the child’s developmental stage and overall level of functioning into account.

ii. Most problems are treated initially with reassurance, support and behavioural interventions.

iii. Whatever the treatment, involve the family.

iv. Avoid removal from school or home wherever possible

v. Medication has a limited role in most disorders.

General principles of behaviour therapy: Behaviour therapy may not be feasible at PHC level and might require referral to a higher centre. However, some of the following principles may be useful in management:

- Behaviour modification in children can be brought about by using behavioural principles.
- Every behaviour is determined by an Antecedent (what happens before a behaviour) and a Consequence (what happens after a behaviour).
- Antecedent or Consequence can be altered to alter a behaviour.
- Desired behaviours need to be reinforced and unwanted behaviour should be decreased.

<table>
<thead>
<tr>
<th>Consider referral when:</th>
<th>Refer to</th>
</tr>
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<tbody>
<tr>
<td>Problem</td>
<td></td>
</tr>
<tr>
<td>Mental retardation</td>
<td>Pediatrician, neurologist,</td>
</tr>
<tr>
<td></td>
<td>Psychologist</td>
</tr>
<tr>
<td>Autism</td>
<td>Psychiatrist, Psychologist</td>
</tr>
<tr>
<td>Significant learning/ academic problems</td>
<td>Psychiatrist</td>
</tr>
<tr>
<td>Suicidal or self-injurious behaviour, severe aggression, Substance abuse</td>
<td>Psychiatrist</td>
</tr>
<tr>
<td>Psychotic symptoms</td>
<td>Psychiatrist</td>
</tr>
<tr>
<td>Attention Deficit hyperactivity disorder</td>
<td>Psychiatrist, psychologist</td>
</tr>
<tr>
<td>Mood disorder, anxiety disorder</td>
<td>Psychiatrist, psychologist</td>
</tr>
</tbody>
</table>
- **Positive reinforcement** is used to promote desired behaviours. It involves the use of rewards which include Tangible rewards as food, objects, etc, and social rewards as praise.
- Initially tangible rewards may be used as reinforcement and later replaced by social rewards to sustain the change in behaviour. Rewards must be chosen on the basis of child’s preferences.
- Unwanted behaviours can be decreased by ignoring them or taking away existing privileges or reinforcement. For example: withholding TV viewing or a regular excursion.
- Punishment should not be used or only sparingly used to bring about a behaviour change.
- If wanted behaviours are reinforced at the same time ignoring the unwanted behaviours, which is also called **differential reinforcement** the benefits may be marked.
- One has to be consistent in providing reinforcements and they should be contingent to the behaviour.
PSYCHIATRIC PROBLEMS IN ELDERLY

Introduction

The elderly population of India is steadily increasing. It is projected that by 2050, proportion of persons who are of age 60 years and above living in India will rise to 19% from current 8% of total population (United Nations Population Division, 2010). The demographic profile depicts that in the years 2000-2050, the overall population in India will grow by 55% whereas aged population 60 years and above will increase by 326% (United Nations 2002). This has resulted in “Greying of India”.

Despite improvement in health care system, Indian elderly is facing a huge burden of communicable and non-communicable diseases. This vulnerable population is more prone to psychiatric problems. These psychiatric problems in elderly are more complex and challenging than those encountered in young- and middle-aged patients due to host of medical and environmental conditions. Most common psychiatric illnesses in Indian elderly population are depression, dementia and anxiety disorders which need attention of General Practitioners.

Clinical picture

Dementia

Dementia is also known as ‘chronic brain failure’, more frequent after 65 years of age. Dementia is a syndrome characterised by concurrent impairment in cognition, behaviour and activities of daily living. Its cardinal feature is memory impairment in the presence of clear consciousness. It can be understood through ‘ABC of dementia’:

57
A- Problems in Activities of Daily Living (ADL)

B- Problems in Behaviour i.e. Behavioral & Psychological symptoms of Dementia (BPSD)

C- Problems in Cognition i.e. memory impairment

A: Dementia interferes with activities of daily routine like dressing, washing, eating, personal hygiene & toilet training.

B: Psychological symptoms in dementia assessed on the basis of interviews with patients and caregivers include delusions, paranoia, hallucinations, anxiety and depressed mood. Behavioural symptoms identified on the basis of observation of the patient include physical aggression, screaming, restlessness, agitation, wandering, sexually inappropriate behaviours, and hoarding.

C: Patients with dementia have problems in cognition i.e. memory, more particularly learning new information. There is a definitive decline in memory from previous state reported by caregivers. Other problems associated with cognitive decline include language, orientation, judgment, executive function and thinking. Memory loss is usually progressive with prevalence doubling every 5 years after the age of 60 years.

**Delirium**

Delirium or acute confusion is a transient organic brain syndrome, a type of medical emergency encountered in health care settings, characterised by an alteration in consciousness caused due to underlying medical conditions. This clinical condition is acute in onset and shorter in duration (few hours to days). Patient with delirium is presented with global impairment of cognitive function (i.e. confused state of mind, disorientation to
time/place and disturbance in sleep-awake cycle). This condition has a diurnal variation, more worsening in night hours (‘Sun downing phenomenon’). Emotional disturbances like anxiety, fear, irritability, anger, apathy and withdrawn, may be associated with delirium in elderly. Mortality among hospitalized patients is high as much as 40%. Hence, clinicians need serious attention to identify and manage this medical emergency

**Depression**

Depression is considered to be ‘Disorder of losses’-loss of mood (sadness of mood); loss of interest in pleasurable activities (anhedonia); and loss of energy (easy fatigability). Depressed/sad mood remains the most common presentation of depression in elderly but may not be spontaneous complaint. In addition to weight loss, persistent anhedonia is common reported symptom in elderly. Psychotic symptoms may also present in elderly depressives. Appetite loss and suicidal ideation or acts may also present in old age depression. Depression may be associated with medical conditions. Elderly patients often report bodily symptoms (aches & pains, gastro-intestinal disturbances) rather than sadness of mood. They also report subjective experience of forgetfulness or memory loss and slow thinking (Pseudo-dementia; ‘dementia of depression’) which should be differentiated from dementia. They may also present with anxiety symptoms, agitation and psychotic symptoms. There is always a high risk of suicide associated with depression in late life.

**Late-onset anxiety disorders**

Anxiety disorders in elderly may be associated with medical disorders or exist as independent disorders (Generalised anxiety disorder, panic disorder & obsessive compulsive disorder). They may be presented with physical symptoms like palpitation, chest pain, choking, giddiness, and tingling numbness; and psychological symptoms like worrying, nervousness, tension, apprehension, anticipatory fear, lack of concentration.
Late-onset psychosis

Elderly may also prone to psychosis like young adults. In late-onset psychosis (also called ‘Paraphrenia’), delusions of persecution & reference and auditory & visual hallucinations are usually present.

Management

Assessment

Dementia

Diagnostic exercise of patient with memory problem starts with a good history from both family members and the patient regarding chronological presentation of symptoms and problem behaviours. Evaluation includes questions related to memory loss like becoming lost in a familiar place, asking food again and again, and losing objects. A thorough evaluation of medical history and examination of bodily systems, including neurological examination is must for patient with memory loss. The nature and degree of the cognitive function and activities of daily living should be assessed with Hindi adaptation of Mini-Mental State Examination (HMSE) and Everyday Abilities Scale for India (EASI). The laboratory tests to rule out reversible causes for dementia are: thyroid stimulating hormone, vitamin B12 & folate levels, electrocardiogram, blood sugar & creatinine. Computed tomography of brain is required for patients with memory loss.

Delirium

Delirium is seen more in intensive medical and surgical units and among patients recovering from surgery or on multiple medications. It needs thorough clinical evaluation, including intoxication, electrolyte disturbances, infections, hypoxia, hypoglycaemia, physical restraint, bladder catheter use and concomitant medications.
Depression

Older persons may not reveal depressive symptoms so easily, hence needs good observation and clinical skills. More focus of interview is towards motor activity, hopelessness, worthlessness, hallucinations, suicidal ideations and memory problems. The laboratory tests to rule out co-morbid medical conditions are: complete blood counts, thyroid stimulating hormone, vitamin B12 & folate levels (vegetarians), electrocardiogram, Fasting blood sugar, serum electrolytes, blood urea & creatinine. Computed tomography of brain is optional for suspected cases of cerebro-vascular lesions.

Late-onset anxiety disorders

Anxiety symptom may be part of medical and psychiatric disorders like hypoglycaemia, hyperthyroidism, cardiac arrhythmias, pulmonary emboli, delirium, depression, dementia and psychotic disorder. It could be related to medications like ephedrine, anticholinergic drugs and benzodiazepine withdrawal.

Late-onset psychosis

Psychotic symptoms in the form of suspiciousness, delusions, hallucinations and agitation are present in schizophrenia and delusional disorders. Family members should be interviewed to explore behavioural disturbances.

Treatment approaches

Once the psychiatric problem is formulated by the clinician, the usual treatment approaches must be individualized depending on clinical status and also targeted to reverse the underlying disorder. Emphasis should be on identifying above mentioned problems early and referring appropriately. Also, the assessment should include evaluation of psychiatric and comorbid medical illnesses, which is a rule rather than exception in the elderly. It is also essential to include drug history keeping in mind the drug-drug/ drug-disease interactions.
Counselling & education regarding disorder as well as diet and medicines is the mainstay for the successful treatment.

**Dementia**

Early identification and treatment may delay or arrest the process of cognitive decline. Treatment of dementia is based on the cholinergic hypothesis of memory. Cholinesterase inhibitors like donepezil, rivastigmine and galantamine are indicated for patients with dementia. Donepezil should be started with dosage of 5mg/day which may titrate to 10mg/day in 2 weeks. Antipsychotics may be indicated for controlling aggressive behaviour, psychotic symptoms and severe agitation. Risperidone (1 to 3 mg) and quetiapine (25 to 400 mg) may be used.

**Delirium**

For patients with delirium, initial treatment includes medical support i.e. maintenance of vital parameters (adequate airway, blood pressure, pulse and temperature). The cause of delirium is established through history, physical examination, and laboratory tests. Specific therapy/intervention is started after identification of precipitant of confusional state. Clinicians also look after and manage excess and confused environmental stimuli present in the vicinity of patient. Patient should be kept in quiet, well-lighted and simply furnished room. Family members and hospital staff should regularly acquaint the patient regarding time, place and person. Restraints should be avoided. For agitated behaviour, low dose antipsychotics (Haloperidol or Risperidone, either oral or intramuscular) may be prescribed.

**Depression**

Antidepressants remain mainstay for treatment of geriatric depression. Newer antidepressants, Specific Serotonin Reuptake Inhibitors (SSRIs) like fluoxetine, sertraline, citalopram and escitalopram can be initiated at lower doses (preferably half of young adult dosage). Gastrointestinal disturbances, weight loss and agitation are commonly reported side
effects with SSRIs. Tricyclic antidepressants may be avoided due to postural hypotension and anticholinergic side-effects. Elderly depressives who do not respond to or tolerate antidepressants may better respond to Electroconvulsive Therapy (ECT). Families should be involved for support building and motivating patient in activities.

**Late-onset anxiety disorders**

Short-acting benzodiazepines like alprazolam (0.125-0.25mg) and lorazepam (2-4mg) may be indicated in patients with anxiety disorders. SSRIs (Escitalopram, citalopram and sertaline) are useful in treatment of anxiety disorders.

**Late-onset psychosis**

Patient with psychosis requires a safe environment. Agitated and suspicious elderly needs inpatient hospitalization. Antipsychotic medications used in late-onset psychotic disorders are risperidone (1-3 mg/day), quetipine (25-300mg/day) and haloperidol (0.5-10mg/day).

**Guidelines for referral**

Refer patients of following conditions to specialist care:

1. Dementia with behavioural and psychological symptoms
2. Complicated delirium
3. Depression not responding to antidepressants
4. Suicidal patient
5. Depression with psychotic/cognitive symptoms
6. Anxiety disorders not responding with medications
7. Agitated and suspicious elderly patient
After completing this chapter, the participants should be able to:

1. Identify a case of Epileptic seizure
2. Classify the patients in different types of epilepsy
3. Understand about the management strategy of epilepsy:
   - How long the medications (AED) is to be continued
   - When to initiate and stop the treatment
   - When to Refer the cases of epilepsy

INTRODUCTION

- Epilepsy is one of the common neuropsychiatric conditions encountered in general practice. It is a diagnosis which carries social stigma and hence the diagnosis should not be made without thorough consideration. For some patients, epilepsy might be a lifelong disorder requiring indefinite treatment.
- Epilepsy is characterized by Recurrent Seizures. Seizures occur due to episodic excessive, abnormal neuronal activity in the brain that produces a sudden change in the neurological functions.
- Its incidence is about 5-8/1000. However, increased incidence is seen in neonates, children, and elderly.

CLASSIFICATION

Summary of international classification of epileptic seizures

I. Partial (Focal) seizures
   a) Simple partial seizures (consciousness not impaired)
   b) Complex partial seizures (temporal lobe or psychomotor seizures, consciousness impaired)
   c) Partial seizures evolving to secondarily generalized seizures [tonic-clonic (grand mal), tonic or clonic]

II. Generalized seizures (convulsive or nonconvulsive)
   a) Absence (petit mal) seizures
   b) Myoclonic seizures
   c) Tonic seizures
   d) Atonic seizures
   e) Clonic seizures
   f) Tonic-clonic (grand mal) seizures

III. Unclassified epileptic seizures
SIMPLE PARTIAL SEIZURES

- Sudden onset and cessation
- Due to focal cortical pathology
- No alteration in consciousness
- Focal symptoms or signs:
  - Motor
  - Sensory or special sensory
  - Psychic (dysphasic, dysmnestic, cognitive, affective, illusions, hallucinations)
- Partial seizures may progress to Complex Partial Seizure (CPS) or to generalized tonic clonic seizures

COMPLEX PARTIAL SEIZURES (TEMPORAL LOBE/PSYCHOMOTOR EPILEPSY)

Seizure may begin with no warning or with motor, sensory, autonomic, psychic signs & symptoms

- Sudden onset and gradual recovery
- Aura (focal symptoms/signs as in simple partial seizures)
- Altered consciousness—motionless stare & unresponsiveness
- Automatism (chewing, lip smacking, fiddling, rubbing of hands, circling, running, mimicry, gestural, ambulatory, verbal, responsive), co-ordinated involuntary motor activity
- Amnesia
- CPS patients may have combination of Aura and altered consciousness and automatism
- CPS may progress to generalized tonic clonic seizures
- Due to focal cortical pathology
- Temporal lobe CPS—60% extra temporal, especially frontal cortex -30%

TYPICAL ABSENCE SEIZURES (PETIT MAL)

- Sudden onset and cessation, (few seconds, average:10 seconds)
- Cessation of motor activity: Blank stare
- Blinking of eyes, jerking and brief automatism may be present
- Impaired responsiveness, Posture and tone preserved
- Several 100/day especially with sleep awake cycle
- EEG—Generalized 3/sec spike and wave pattern
- Precipitated by hyperventilation
- Easily controlled with AED

ATYPICAL ABSENCE SEIZURES

- Absence (blank stare)
- Impaired responsiveness
- Focal signs prominent, include tone changes, clonic jerking, motor spasm, automatism
- Onset and cessation often gradual, and seizure may be prolonged
- In-patients with diffuse cerebral damage, mental retardation
- Difficult to control
MYOCLOMIC SEIZURES
- Brief jerk, singly or in series + induced by stimulus
- Intensity varies from slight tremor to massive jerk
- Distribution varies, from single muscle to generalized jerking
- Consciousness preserved
- Rapid onset and cessation on waking or going off to sleep
- Myoclonus may precede a generalized tonic clonic seizure
- EEG generalized spike and polyspike and wave discharge
- Occurs as part of: Idiopathic generalized epilepsy (JME), progressive myoclonus epilepsy; or in patients with diffuse cerebral damage associates with mental retardation

PRIMARILY GENERALIZED TONIC-CLONIC SEIZURES (GRAND MAL)

Ictus
- Loss of consciousness-shrill cry- tonic phase (10-30 sec)
- Respiration ceases
- Clonic phase, labored breathing,
- Tongue bite, frothing, incontinence, cyanosis, vocalization, autonomic features (30-60 sec)
- Flaccidity of muscles (2-30 minutes)
- Sudden onset and gradual recovery

Post ictal
- Confusion, drowsiness, sleep, headache, muscle pain
- May be modified with treatment-tonic, clonic or atonic
- May have precipitating factors or diurnal variation

FEBRILE CONVULSIONS
- Seizures are associated with fever
- Common (2-5% of all children) (3 months-6 years)
- Peak age of onset (6 month-2 years)
- Simple, complex types, febrile SE-young children
- Recurrence – early age of onset & positive febrile history
- Seizure usually at onset of febrile illness
- Complex febrile convolution: > 30 min; partial seizure, recur within 24h
- Risk of continuing epilepsy is small

The first step in the evaluation of a patient with possible epilepsy is to determine whether the patient does or does not have seizures.
- History from the patient and eyewitness: The history obtained from the patient and a reliable informant/eye witnesses is often the most important information in establishing diagnosis of a seizure disorder.
- Questions related to parts of a seizure: Begin by asking in detail about the events before, during and after the seizure, namely
  - Prodrome
To determine the cause (etiology) of the seizure, determine:
- Whether there is a family history of epilepsy
- Personal history of head trauma, birth complications, febrile convulsions, middle ear or sinus infection, alcohol or drug abuse or symptoms of cancer

**Physical examination:** A thorough physical examination: look for disorders associated with epilepsy as well as signs of head trauma, infections of middle ear or sinuses, congenital malformations, alcohol or drug abuse and symptoms of cancer.

**Electroencephalographic studies (EEG):** In approximately 50% of patients with epilepsy, a single EEG may show no abnormalities at all. An EEG provides information about:
- Confirmation regarding presence or absence of abnormal electrical activity
- Information regarding type of seizure disorder
- Location of seizure focus

**Laboratory tests and Neuro-imaging:** The following tests are done to determine the cause of newly diagnosed seizure disorder:
- Electrolyte and liver function test
- EEG in sleeping and waking states
- MRI or CT brain: to rule out serious structural lesion
- Screening tests for toxic substances if alcohol or drug abuse or withdrawal is suspected
- Lumbar puncture performed if fever or cancer suspected

**Note:** The request for the investigations by the medical officers should be judicious. A high degree of clinical suspicion should govern the need for investigations for the patients with seizure disorder. These should include for eg. Cases of recurrent seizures even on regular & adequate treatment, persons with first episode of seizure, post encephalitic sequelae, history of head injury etc.

**DIFFERENTIAL DIAGNOSIS:**

**Disorders that must be differentiated from epilepsy:**
- Non-epileptic attack disorder (Pseudo seizures)
- Syncopal attacks
- Transient Ischaemic attacks (TIA)
- Breath holding spells

**Non Epileptic Attack Disorder or Pseudo-seizure**
- Attacks with motor phenomenon
- Thrashing of limbs (people around)
- Prominent pelvic movements and back arching
• Distractability/interactions with the environment
• Excessive salivation, teeth clenching
• Commoner in females than males
• Obvious secondary gains
• Overt psychiatric illness may be there

**Syncopal Attacks**
• Evidence of precipitant factors
• Lightheadedness, dizziness, nausea
• Ringing in the ears
• Bilateral loss of vision
• Pallor & Collapse
• Some twitching tonic movement
• Irregular myoclonic jerking
• Rapid recovery when supine
• Sweating, subsequent flushing

**Transient Ischaemic Attacks**
• Focal Neurological Deficit
• Loss of consciousness – rare
• May not be repetitive

**Breath Holding Spells**

**CAUSES OF SEIZURES ACCORDING TO AGE:**

<table>
<thead>
<tr>
<th>Neonate-3 years</th>
<th>3-20 years</th>
<th>20-60 years</th>
<th>&gt;60 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Prenatal injury</td>
<td>- Genetic predisposition</td>
<td>- Brain tumor</td>
<td>- Vascular disease</td>
</tr>
<tr>
<td>- Perinatal injury</td>
<td>- Infection</td>
<td>- Trauma</td>
<td>- Brain tumors</td>
</tr>
<tr>
<td>- Post natal trauma</td>
<td>- Trauma</td>
<td>- Vascular disease</td>
<td>- Trauma</td>
</tr>
<tr>
<td>- Metabolic def.</td>
<td>- Cong. Malformation</td>
<td>- Infection</td>
<td>- Metabolic Disorders</td>
</tr>
<tr>
<td>- Congenital Malformation.</td>
<td>- Metabolic def.</td>
<td></td>
<td>- Infections</td>
</tr>
<tr>
<td>- CNS Injury</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Features of in increased risk of seizure recurrence*
• Seizure types (e.g. partial, tonic and atypical absence seizures etc).
• High previous seizure frequency
• Long duration of epilepsy
• Structural abnormality or Psychiatric disorder
• Epileptic syndromes (e.g. JME, LGS etc)
• Inadequate AED/Misdiagnosis
Neuro-imaging in Epilepsy: Indications for structural imaging include:

- Partial seizures or grossly abnormal EEG
- Fixed/progressive neurological or psychological deficit
- Generalized seizures before the age of 1 year and after 20 years
- Refractory seizures/status epilepticus/seizures in elderly

Treatment:

The initiation of antiepileptic drug treatment: Always ask

- Is the diagnosis of epilepsy certain?
- What are the risks of recurrence of seizures without treatment?
- What are the risks of recurrence of seizures with treatment?
- Are the presences of precipitating factors relevant?
- Are the seizure characteristics relevant?
- What are the risks of side effects to treatment?

Consider drug therapy after a first unprovoked seizure if:

- The patient has a neurological deficit
- The EEG shows unequivocal epileptic activity
- The patient or their family/carers consider the risk of having a further seizure unacceptable
- Brain imaging shows a structural abnormality

Choice of Anti Epileptic Drugs (AEDs) for different seizure types:

<table>
<thead>
<tr>
<th>Seizure type</th>
<th>First choice</th>
<th>Second choice</th>
</tr>
</thead>
<tbody>
<tr>
<td>Partial seizures</td>
<td>Carbamazepine, valproate</td>
<td>Phenytoin, Lamotrigine,</td>
</tr>
<tr>
<td>Simple partial, complex</td>
<td></td>
<td>Clobazam, Phenobarbitone</td>
</tr>
<tr>
<td>partial, secondarily</td>
<td></td>
<td></td>
</tr>
<tr>
<td>generalized</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Generalized seizures</td>
<td>Valproate</td>
<td>Lamotrigine,</td>
</tr>
<tr>
<td>Tonic-clonic, clonic</td>
<td>Carbamazepine</td>
<td>Clobazam, Phenobarbitone</td>
</tr>
<tr>
<td>Absence</td>
<td>Valproate, Ethosuximide</td>
<td>Lamotrigine, Clonazepam</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Acetazolamide</td>
</tr>
</tbody>
</table>
The newer drugs (gabapentin, lamotrigine, levetiracetam, oxcarbazepine, tiagabine, topiramate, and vigabatrin) are recommended for managing epilepsy in people who have not benefited from treatment with the older antiepileptic drugs or for whom the older antiepileptic drugs are unsuitable. However this should be carried out at higher centres under specialist care.

### Anti Epileptic Drugs: starting and maintenance doses for adults

<table>
<thead>
<tr>
<th>AED</th>
<th>Starting dose (mg)</th>
<th>Average maintenance dose (total mg/day)</th>
<th>Doses/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbamazepine</td>
<td>100</td>
<td>600-1800</td>
<td>2-3 (retard:2)</td>
</tr>
<tr>
<td>Clobazam</td>
<td>10</td>
<td>10-30</td>
<td>1-2</td>
</tr>
<tr>
<td>Clonazepam</td>
<td>0.5</td>
<td>0.5-3</td>
<td>1-2</td>
</tr>
<tr>
<td>Ethosuximide</td>
<td>250</td>
<td>500-1500</td>
<td>1-2</td>
</tr>
<tr>
<td>Lamotrigine</td>
<td>50</td>
<td>200-400</td>
<td>2</td>
</tr>
<tr>
<td>Oxcarbazepine</td>
<td>300</td>
<td>900-2400</td>
<td>2-3</td>
</tr>
<tr>
<td>Phenobarbitone</td>
<td>60</td>
<td>60-180</td>
<td>1</td>
</tr>
<tr>
<td>Phenytoin</td>
<td>200-300</td>
<td>200-400</td>
<td>1-2</td>
</tr>
<tr>
<td>Valproate</td>
<td>500</td>
<td>1000-2500</td>
<td>1-2</td>
</tr>
</tbody>
</table>

**Note:**
- The clinician should be aware of the special populations with seizure disorders (children elderly and women with pregnancy). The doses of antiepileptics need to be adjusted accordingly. Also the common drug interactions with AEDs like Phenytoin, Carbamazepine and valproate should be made note of, while prescribing them.
- All women on anticonvulsants should start taking folic acid supplements of 5 mg per day well before the pregnancy.
Anticonvulsant drugs can increase the risk of fetal malformation, although the absolute risk is low. The risk is particularly increased in women taking valproate and phenytoin.

Women with epilepsy (and their partners if appropriate) should receive accurate information and counseling about contraception, conception, pregnancy, caring for children, breast feeding, and the menopause.

**STEPWISE ‘AED’ PLAN FOR NEWLY DIAGNOSED PATIENT**

**Step 1:** Precipitating factors: fever, fatigue, alcohol and drug abuse

**Step 2:** Reasons for AED treatment, expectations, limitations, duration of therapy, need for regular tablet taking, AED do not provide a cure for epilepsy, have to be taken for a long time, 70-80% of patients seizures controlled by regular medication

**Step 3:** Start with small dose of AED (1st line), dose increments gradually to therapeutic level, may increase to the maximum tolerated dose, if necessary

**Step 4:** If seizures continue in spite of maximally tolerated dose of a first-line AED:

- Review the diagnosis of epilepsy
- If there is any underlying structural lesion: imaging of the brain
- Whether patient is complaint by non-confrontational enquiry of patient and carers
- Counting the number of tablets, measurement of serum drug level: Low serum AED suggests non-compliance.
- Start the 2nd AED and taper the first AED.

**Duration of treatment:**

- Patients are more likely to remain free from seizures if they have been free from seizures for more than two years while taking antiepileptic drugs
- Decision to withdraw anticonvulsant should be done under the guidance of a specialist.

**Referral to specialist care:**

- Refer patients with first episode seizure to specialist care
- Refer patients with prolonged seizures, status epilepticus
- Refer if there may be difficulties in monitoring the patient’s condition
PSYCHIATRIC EMERGENCIES

Introduction:

A psychiatric emergency is defined as any disturbance in thoughts, feelings or actions for which immediate therapeutic interventions are necessary. Such patients may cause harm to themselves or others or suffer immense acute distress which is intolerable. These are clinical situations which are unique as they may pose risk and danger not only for the patient but also for the people around him. The emergent task at hand is triage, evaluation, formulation and disposition or thereafter referral. The challenge is ensuring the safety of the patient, others and oneself. The handling of a psychiatric emergency can be very intimidating for a medical professional, however the management becomes much simpler if they understand the nature of the underlying psychiatric disorder and follow some simple steps. The management of a psychiatric emergency is a team effort in which both medical professionals and hospital staff have crucial roles. Prompt and appropriate management of psychiatric emergencies saves lives.

A psychiatric emergency can include any of the following clinical scenarios:-

Box No. 1: PSYCHIATRIC EMERGENCIES

| 1. Suicidal patient |
| 2. Violent patient |
| 3. Substance withdrawal |
| 4. Severe anxiety / panic attack |
| 5. Drug toxicity / intoxication |
| 6. Drug induced parkinsonism |

The following text discusses the presentation and management of two of the most common and important psychiatric emergencies in detail, namely suicide and violence and briefly discusses the others.
SUICIDE

Introduction

Suicide is the termination of one’s life intentionally. It is the third leading cause of death between ages 15 and 24. A suicidal patient is one who has attempted or tried to attempt or frequently thought of attempting suicide. A suicidal patient needs emergent intervention as this can prevent a completed suicide. As opposed to popular belief suicide is more often a culminating event of an underlying psychiatric disorder (in 95% of cases) than of any circumstantial life event. The prevalence of suicide is 10-15% in patients of depression, 10% in schizophrenia and this number rises when alcoholism complicates the picture. Hence the evaluation for an underlying psychiatric disorder is a must in all such patients. Slitting of wrist, hanging, overdose of sleeping pills are some common means applied by such patients. Risk assessment is an integral part of evaluation and management.

Presentation of a suicidal patient:

Such patients have either frequently thought of/ contemplated suicide or have planned the act or have attempted suicide.

They are often brought by family.

Box No. 3- Warning signs of suicide

<table>
<thead>
<tr>
<th>Warning signs of suicide</th>
</tr>
</thead>
<tbody>
<tr>
<td>• A person talks about death, threatens of committing or discusses suicide.</td>
</tr>
<tr>
<td>• Discusses different methods of suicide.</td>
</tr>
<tr>
<td>• A person mentions suicidal ideation.</td>
</tr>
<tr>
<td>• A person attempts any act of deliberate self harm.</td>
</tr>
<tr>
<td>• A person is seen making goodbye gestures or communications, writing of will or other acts suggestive of a suicidal plan.</td>
</tr>
<tr>
<td>• A person has suffered recent major loss of life or property.</td>
</tr>
<tr>
<td>• Hopelessness.</td>
</tr>
<tr>
<td>• Severe agitation/anxiety</td>
</tr>
</tbody>
</table>
Suicidal risk: anyone can attempt suicide however a few risk factors pre-dispose an individual to attempt / commit suicide. The presence of a psychiatric disorder however is the single most important and common risk factor for suicidal behavior.

Fig.No. 1 enlists the risk factors increasing the risk of suicide:

Suicidal Risk-Assessment: this is an integral part of management of such patients. This establishes the severity of the situation and the likelihood of future death by suicide.

Box No. 4 –Types of suicidal risk

- **Low suicidal risk**: such patients have less severe psychiatric illnesses, better social support, fewer attempts, have employed attempted less lethal means of suicide like superficial cut marks on wrist.
- **Moderate suicidal risk**: presence of psychiatric disorder, multiple attempts, attempts in last few days, low-moderate lethality attempts like pills, alcoholism +/-
- **High suicidal risk**: severe psychiatric illness, attempt in last few hours, lethal attempt like hanging/gunshot/pesticide, poor social support

Two important terms in suicide:

1. Intentionality: this refers to the intent to commit suicide. Patients with high intentionality make elaborate plans and try to ensure that the attempt of suicide leads to definite death.
2. Lethality: this refers to the means adopted to commit suicide. High lethality of suicide is indicative in the use of firearms, hanging or use of pesticide as means.
Fig No 2- factors associated with high suicidal risk.

Suicidal Risk is high if

- Lethal attempt
- Psychotic symptoms
- Suicidal intent
- Accessible weapon
- Alcohol use
- Suicidal plan

Management of a Suicidal patient

- A suicidal patient either thinks attempting suicide or has already attempted suicide.
- The goal of intervention is to prevent completed suicide.
- Suicide is preventable if intervention is done timely.
- Assessment of suicidal risk is important.
Following simple steps must always be followed:

**Steps in Management of Suicide**

**Step 1**  
Assess the physical condition of the patient. (check vitals, pallor, cyanosis, higher mental functions, local injury/wound etc.)

**Step 2**  
If found medically stable then calm down patient, and do a risk assessment as per the above points mentioned.

**Step 3**  
Evaluate for underlying psychiatric illness by taking history and mental status examination.

**Step 4**  
Once suicidal risk is established it is best to admit the patient. Proceed by instructing for 24 hr strict vigilance by hospital staff and family members. No potentially harmful object should be near the patient and all medication given should be supervised.

**Prevention of Suicide**

Suicide is preventable condition. Early recognition of risk factors for suicide. Early diagnosis and prompt treatment of mental disorders like depression, schizophrenia, bipolar disorder and substance abuse. Not ignoring warning signals such as talks, threats, thoughts or previous attempts of deliberate self harm. Directly asking patient about death wishes, suicidal ideation and intentions helps in assessing the risk. 24 hr strict vigilance is advised till the underlying psychiatric illness responds to treatment.

**When to Refer a suicidal patient?**

- All suicidal patients require psychiatric evaluation once medically stable.
- Hospitalization in psychiatric ward is recommended for all patients who have made lethal attempts of deliberate self harm or who exhibit high risk of suicide.
- Early psychiatric intervention can prevent suicide.
Violent patient

Introduction:

Violence is associated with many psychiatric disorders. Violence may include physical assault/ threat or breaking of property and verbal abuse. Serious threat may be posed to others and the treating team by a violent patient .Violence can occur in any psychiatric disorder however is more common in patients of psychosis, mania, schizophrenia or substance intoxication .Organic causes like neurological insult can also present with violence. These patients are sufferers themselves first and perpetrators of violence only later. Also care should be taken not to violate the human rights of any patient by forceful restraint or injection unless as last resort.

Causes of violence in medical practice:

Box No. 4

Some medical conditions that can present with violence

- Ictal or post ictal states
- Head injuries
- Frontal & temporal lobe pathologies
- Dyselectrolemias
- Delirium and dementias
- Renal / hepatic failure
- Endocrine disorders etc
Psychiatric factors that underlie violent behavior include:

- Manic hyper excitement and agitation
- Exacerbation of psychosis
- Paranoid delusions and fear
- Substance intoxication or withdrawal
- Delirium
- Catatonic hyperexcitement
- Mental retardation with behavioral problems

Management of violent behavior in clinical settings

The management of a violent patient can be challenging. It requires patience, vigilance, team effort and preparedness. Following box enlists steps in the approach to a violent patient.

Box No. 5:

Approach to a violent patient includes

- Ensuring the safety of others including oneself
- Restrain or seclusion may be required
- To rule out organicity and look for treatable medical cause
- Medication oral or parenteral to control agitation
- Psychiatric assessment by history and MSE to establish underlying diagnosis
- Treat underlying psychiatric disorder
Special precautions in dealing with a violent patient:

- Do not approach a violent patient alone. Other staff should be present.
- Ensure the removal of potentially harmful objects with the patient or in the vicinity.
- Assess for the possibility of possession of weapon by the patient.
- In case of uncontrollable violence a suitable escape door must be present.
- Do not intimidate, argue or pose any threat to the patient.
- Approach the patient in a benign, non-threatening and calm manner.
- Maintain at least 3 to 6 feet distance from the patient.
- If patient agrees to speak with you sit him down and ask him about his problem. Listen to him and let him know you are there only to help him and he need not fear.

Steps in Management of violence in clinical settings:

1) Try to talk to the patient if possible and calm him with your words.
2) Urge or convince him to take oral medication.
3) If he declines your request consider injectable drugs to calm him down.
4) Restrain or seclusion may be required in very severe cases. However keep a concern for the human rights of the patient.

Box No. 6: – psychotropic drugs used in management of violence

<table>
<thead>
<tr>
<th>MEDICATION AND DOSAGE</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lorazepam , 2-4mg, PO or I/M or diazepam 5-10mg iv/ im</td>
<td>effective particularly in non psychotic</td>
</tr>
<tr>
<td>Olanzapine ,10 mg, PO or I/M</td>
<td>indicated in psychosis</td>
</tr>
<tr>
<td>Haloperidol 5-10mg + Promethazine 25-50mg IM</td>
<td>very effective in rapid effect in rapid control of psychotic agitation</td>
</tr>
</tbody>
</table>
When to refer a violent patient

• All suspected cases of organicity should be investigated and referred to concerned speciality.

• All others should be stabilized and referred to psychiatrist for evaluation and management.

• The medical condition must be examined and stabilized before a psychiatric referral.

• Very severe cases need inpatient management.

Other psychiatric emergencies

Substance withdrawal particularly complicated alcohol withdrawal is an emergency where patient may have seizures or in rare instance go into delirium tremens (DT). 20% of untreated cases of DT die hence prevention and timely intervention is crucial. Patients dependent on opioids may also have very distressing withdrawal symptoms but they are less likely to cause serious consequences like alcohol withdrawal. Management is with benzodiazepines and thiamine oral or in injection in alcohol withdrawal.

Panic attacks are discrete episodes of severe anxiety associated with autonomic symptoms like breathlessness, palpitations, and heaviness of head and sense of impending doom. Such patients frequent casualty settings often thinking they're having a heart attack. Use of benzodiazepines is indicated. Patients should be referred thereafter to psychiatrist.

Drug induced acute dystonias, oculogyric crisis, extrapyramidal symptoms often present as psychiatric emergencies. Such patients are usually on an antipsychotic (mostly typical antipsychotics like haloperidol). IM/IV Promethazine 25-50 mg is treatment of choice.
ALCOHOL USE AND DRUG USE DISORDERS

INTRODUCTION

Almost all cultures have used psychoactive substances to facilitate social interaction, to alter consciousness, to heal. Our society’s expanded chemical manipulation simply represents large technical capacity, more wealth, leisure and individual choice. Among many ills which have plagued the human society today, the drug abuse is of greatest social relevance.

To begin with, let us understand what a drug is. World Health Organization (WHO) defines a drug as any substance that, when taken into the living organism, may modify one or more of its functions. This definition conceptualizes ‘drug’ in a very broad way, including not only the medicines but also the other pharmacologically active agents. A psychoactive drug is one that is capable of altering mental functioning.

Drug-addiction is a chronic, often relapsing brain disease characterized by compulsive drug seeking and use despite harmful consequences to the user and the people around. Repeated drug exposure leads to changes in structure and function of the brain. Initial decision of drug-intake may be voluntary but repeated drug exposures affect person’s self-control and ability to make sound decisions.

There are four important patterns of drug use disorders, which may overlap each other. These are as follows:

1. Acute Intoxication
2. Withdrawal State
3. Dependence Syndromes
4. Harmful Use

1. Acute Intoxication is a transient condition following the administration of alcohol or other psychoactive substance, resulting in disturbances in level of consciousness, cognition, perception, affect or behaviour, or other psycho-physiological functions and responses. This is usually associated with high blood levels of the drug. Sometimes even a low dose of the drug or alcohol is sufficient to cause intoxication. This may occur either due to low threshold (i.e. chronic renal failure) or due to idiosyncratic sensitivity. As the time passes, the intensity of intoxication lessens and the symptoms eventually disappear in the absence of further use of the substance. The recovery is generally complete except in cases where tissue damage or other complications have occurred.
2. **Withdrawal State** comprises a cluster of symptoms that are specific to the drug used and develops on total or partial withdrawal of the drug usually after repeated and/or high-dose use. Withdrawal syndrome is generally short lasting ranging from few hours to few days. Withdrawal symptoms are relieved if the drug is taken. The withdrawal states are further classified as:

(i) Uncomplicated

(ii) With convulsions

(iii) With delirium

3. **Dependence syndrome** is a condition, which generally develops on regular and continued use of a drug. When a person becomes dependent on a particular drug, he has a compulsive need to obtain and use the drug, each time requiring a higher dose of the drug to get the same effect because of development of tolerance. In case the drug is not available or available in lesser quantity, withdrawal symptoms develop. This behaviour is detrimental to personal and social life of the individual. According to the International Classification of Diseases (ICD-10), the dependence syndrome is a cluster of physiological, behavioural, cognitive phenomena in which the use of a substance or a class of substances taken on a much higher priority for a given individual than other behaviours that once had greater value. The dependence syndrome is characterized by a strong, often over-powering desire to take a psychoactive drug (may or may not have been prescribed medically). The definite diagnosis of dependence should usually be made only if at least three of the following have been experienced or exhibited at some time during the previous year;

(i) A strong desire or sense of compulsion to take the substance.

(ii) Difficulties in controlling substance-taking behaviour in terms of its onset, termination or levels of use.

(iii) A physiological withdrawal state occurs when the substance use has ceased or reduced,

(iv) Evidence of tolerance is seen in dependent users. In order to achieve the effects of the drug obtained with low doses initially, he person has to take the higher doses.

(v) Progressive neglect of alternative pleasures or interests because of substance use occurs and the individual spends more time in procuring and using the psychoactive drug.

(vi) There is persistence with the substance despite of harmful effects caused by the drug on various body organs

4. **Harmful Use** of a drug can be classified as follows:

(i) Continuous drug use, despite the awareness of harmful medical and/ or social effect of the drug being used.

(ii) A pattern of physically hazardous use of drug (i.e. driving during intoxication)

The diagnosis of ‘Harmful use requires that the actual damage is caused to the physical or the mental health of the user. If the patient qualifies for the diagnosis of dependence syndrome then harmful use is not diagnosed. ICD-10 includes other syndromes associated with psychoactive
substance use such as psychotic disorder, amnesic syndrome, and residual and late-onset psychotic disorder. The major dependence producing drugs are as shown in Table I.

### TABLE I: PSYCHOACTIVE SUBSTANCES

1. Alcohol  
2. Opioids i.e. opium, heroin  
3. Cannabinoids, i.e. Cannabis, Marihuana  
4. Cocaine  
5. Amphetamines and other sympathomimetic drugs  
6. Hallucinogens i.e. Lysergic acid diethylamide (LSD), Phencyclidine (PCP)  
7. Sedatives and hypnotics i.e. barbiturates  
8. Inhalants i.e. volatile solvents  
9. Nicotine, and  
10. Other Stimulants i.e. Caffeine

---

**AETIOLOGY OF SUBSTANCE USE DISORDER IS A COMPLEX INTERPLAY OF MULTIPLE FACTORS. BIOLOGICAL, PSYCHOLOGICAL AND SOCIAL FACTORS ARE IMPLICATED AND SHOWN IN TABLE II.**

### TABLE II: AETIOLOGICAL FACTORS IN SUBSTANCE USE DISORDERS

1. Biological Factors  
   (i) Genetic Vulnerability  
   (ii) Co-morbid psychiatric disorder or personality disorder  
   (iii) Co-morbid medical disorders  
   (iv) Reinforcing effects of the drugs  
   (v) Withdrawal effects and craving  
   (vi) Biochemical factors  
2. Psychological Factors  
   (i) Curiosity, need for novelty seeking  
   (ii) General rebelliousness and social non-conformity  
   (iii) Early initiation of alcohol and tobacco  
   (iv) Poor impulse control  
   (v) Sensation-seeking (high)  
   (vi) Low self-esteem (anomie)  
   (vii) Concerns regarding personal autonomy  
   (viii) Poor stress management skills  
   (ix) Childhood trauma or loss  
   (x) Relief from fatigue or boredom  
   (xi) Escape from reality  
   (xii) Lack of interest in conventional goals  
   (xiii) Psychological distress  
3. Social Factors  
   (i) Peer pressure (often more important than the parental factor  
   (ii) Role model: imitating the ego-ideal.
ALCOHOL DEPENDENCE

Complications
Alcohol dependence has its effect not only on the consumer’s body but also on the social and personal life of the individual leading to several complications both medical and social. These are as follows:

1. Acute Intoxication
After initial excitation for a brief period, there is generalized Central Nervous System depression with alcohol use. With increasing intoxication, there is increased reaction time, slowed thinking and distractibility and poor motor control. Later, dysarthria, ataxia and in-co-ordination occur. There is progressive loss of self control with frank disinhibited behaviour. The duration of intoxication depends on the amount and the rapidity with which alcohol is ingested. The signs of intoxication are obvious with blood alcohol levels of 150-200 mg%. Increasing drowsiness followed by coma and respiratory depression develop at the blood level of 300-450 mg%. When the blood levels reach between 400-800 mg%, death is likely to occur (Table III).

TABLE III: BLOOD ALCOHOL LEVELS AND BEHAVIOUR

<table>
<thead>
<tr>
<th>Blood Alcohol Concentration</th>
<th>Behavioural Correlated</th>
</tr>
</thead>
<tbody>
<tr>
<td>25-100 mg%</td>
<td>Excitement</td>
</tr>
<tr>
<td>80 mg%</td>
<td>Is the legal limit for driving in UK while in India this limit is</td>
</tr>
<tr>
<td>100-200 mg%</td>
<td>Serious intoxication, slurred speech, in-co-ordination, nystagmus.</td>
</tr>
<tr>
<td>300-350 mg%</td>
<td>Hypothermia, dysarthria, cold sweats</td>
</tr>
<tr>
<td>350-400 mg%</td>
<td>Coma</td>
</tr>
<tr>
<td></td>
<td>Death may occur</td>
</tr>
</tbody>
</table>
Sometimes a small dose of alcohol may produce active intoxication in some persons. It is an idiosyncratic reaction to alcohol and not related to blood levels. This condition is known as *pathological intoxication*. Sometimes an individual develops complete amnesia of the acute intoxication known as *blackouts*.

2. **Withdrawal Syndrome**

In a dependent alcoholic when the blood levels decrease withdrawal symptoms appear. The most common with withdrawal syndrome is a hangover on the next morning. Mild tremors, nausea, vomiting, weakness, irritability, insomnia and anxiety are other common withdrawal symptoms. Sometimes withdrawal syndrome is more severe characterized by one of the following three: alcoholic hallucinosis, alcoholic seizures, and delirium tremens.

(i) **Alcoholic hallucinosis**: During partial or complete abstinence, a dependent alcoholic experiences hallucinations (usually auditory). The hallucinations are generally accusatory or threatening in nature. It occurs in about 2% of the cases. These hallucinations persist after the withdrawal syndrome is over, and classically occur in clear consciousness. Usually recovery occurs within one month and the duration is very rarely more than six months.

(ii) **Alcoholic seizures** (rum fits): Generalized tonic clonic seizures occur in about 10% of alcohol dependent patients, usually 12-48 hours after a heavy bout of drinking. Often, these patients have been drinking alcohol in large amounts on a regular basis for many years. Multiple seizures (2-6 at one time) are more common than single seizures. Sometimes status epilepticus may be precipitated. In about 30% of the cases, delirium tremens follows.

(iii) **Delirium tremens** (DT): It is the most severe alcohol withdrawal syndrome. It occurs usually within 2-4 days of complete or significant abstinence from heavy alcohol drinking in about 5% of patients, as compared to acute tremulousness which occurs in about 34% of the cases. The course of delirium tremens is short and recovery generally occurs within 37 days. This is an acute organic brain syndrome with the following characteristic clinical features:

(a) Clouding of consciousness with disorientation in time and place.
(b) Poor attention span and distractibility
(c) Visual (and also auditory) hallucinations and illusions, which are often vivid and very frightening. Tactile hallucinations of insects crawling over the body may occur.
(d) Marked autonomic disturbance with tachycardia, fever, sweating, hypertension and papillary dilatation are the usual features encountered.
(e) Psychomotor agitation and ataxia may be present.
(f) Insomnia, with a reversal of sleep-wake pattern can be seen.
(g) Dehydration with electrolyte imbalance is present.

Death may occur due to cardiovascular collapse, infection hyperthermia or self-inflicted injury. At times, medical illness like pneumonia, fractures’ liver disease and pulmonary tuberculosis may complicate the clinical picture.

3. Neuropsychiatric complications of chronic alcohol use
   (i) Wernicke’s encephalopathy: This is an acute reaction occurring in response to severe deficiency of thiamine, commonest cause being chronic alcohol intake. The onset of this disorder characteristically occurs after a period of persistent vomiting. The important clinical features are:
      (a) Ocular signs: Course nystagmus and ophthalmoplegia, with bilateral external rectus muscle paralysis occur early. In addition, papillary irregularities, retinal hemorrhages and papilledema can occur, causing an impairment of vision.
      (b) Higher mental function disturbance: Disorientation, confusion, recent memory disturbances, poor attention span and distractibility are the common impairments of higher mental functions. Other early symptoms seen are apathy and ataxia. Peripheral neuropathy and serious malnutrition are generally co-existent. Neuropathological findings show neuronal degeneration and hemorrhage in thalamus, hypothalamus, mammillary bodies and midbrain.
   (ii) Korsakoff’s psychosis: Korsakoff’s psychosis often follows Wernicke’s encephalopathy and to-gether they are referred as the Wernicke-Korsakoff Syndrome. Clinical manifestations of Korsakoff’s psychosis include an organic amnestic syndrome, characterized by gross memory disturbances with confabulation and impaired insight. Neuropathological findings show widespread lesion and the most consistent changes are seen in bilateral dorsomedial nuclei of the thalamus and mammillary bodies. The changes are also seen in periventricular and periequiductal grey matter, cerebellum and parts of brainstem. The cause is usually severe, untreated thiamine deficiency secondary to chronic alcohol use.
   (iii) Marchiafave-Bignami disease: This disorder is probably caused by alcohol-related deficiency with pathological changes in the form of wide-spread demyelination of corpus callosum, optic tract and cerebellar peduncles. Clinical manifestations include disorientation, epilepsy, ataxia, dysarthria,
hallucinations, spastic limb paralysis, and deterioration of personality and intellectual functions. This is a rare disorder.

(iv) Other Complications of Alcohol Use include
(a) Alcoholoc dementia
(b) Cerebellar degeneration
(c) Peripheral neuropathy
(d) Central Pontine myelinosis
(e) Optic atrophy (particularly with methyl alcohol)

Treatment:
Following steps should be considered before starting treatment for alcohol dependence:

(iv) Look for the possibility of a physical disorder.
(v) Look for the possibility of a psychiatric diagnosis.
(vi) Patient’s motivation to undergo treatment should be assessed.
(vii) Assessment of the social support the patient enjoys should be done.
(viii) Personality characteristics of the patient should be assessed in details.
(ix) Level of current and past occupational and socials functioning should be assessed.

The treatment comprises two broad components i.e. detoxification and treatment of alcohol dependence.

1. Detoxification
Treatment of alcohol withdrawal symptoms is detoxification. Symptoms that are produced due to non-availability or lesser availability of alcohol to a dependent alcohol user are withdrawal symptoms and the constellation of symptoms form withdrawal syndrome. The usual duration of uncomplicated withdrawal syndrome is 7-14 days. The aim of detoxification is the symptomatic management of the emergent withdrawal symptoms.

Benzodiazepines are the drugs of choice for symptomatic detoxification. Chlordiazepoxide is generally used in the dosage range of 80-200 mg per day in three to four divided doses. Another drug used is diazepam, its dose being 40-80 mg per day in divided doses. The typical dose of chlordiazepoxide in moderate alcohol dependence should be as follows:

- 1st day, 20 mg four times
- 2nd day 15 mg four times
- 3rd day 10 mg four times
- 4th day 5 mg four times
- 5th day 5 mg two times and then stop.

In severe dependence, higher doses are required for longer period (up to 10 days. In some countries drugs like chlormethiazole (1-2 g/day) and carbamazepine (600-1600 mg/day) are also used for detoxification. These drugs follow a standard dose regime
with dosage steadily decreasing and stopping on 10th day. Nutritional deficiency is generally associated with alcohol dependence therefore, vitamins should be given.

2. **Treatment of alcohol dependence** After the detoxification process is over further management is done with the aim to prevent the patient from restarting alcohol consumption. Several methods are available to choose that suit best to the patient. Some of these important methods are as follows:
   (i) Behaviour therapy
   (ii) Psychotherapy
   (iii) Group therapy
   (iv) Deterrent therapy
   (v) Other medications
   (vi) Psychosocial rehabilitation

**OPIOID USE DISORDER**

The natural alkaloids of opium and their synthetic preparations are highly dependence producing drugs (Table IV). The dried exude obtained from unripe seed capsules of *Papaver somniferum* is a highly dependence producing and is abused for centuries. In the last few decades, the use of opioids has tremendously increased all over the world. India being the transit point for illicit drug trade between golden triangle (Burma-Laos-Thailand) and golden crescent (Iran-Afghanistan-Pakistan) is among the worst affected countries. Addition of heroin to Indian streets some two decades ago has caused devastating effects on the adolescents and youth of the country.

**TABLE IV: OPIOID DERIVATIVES**

<table>
<thead>
<tr>
<th>1. Natural Alkaloids of opium</th>
</tr>
</thead>
<tbody>
<tr>
<td>(i) Morphine</td>
</tr>
<tr>
<td>(ii) Codeine</td>
</tr>
<tr>
<td>(iii) Thebaine</td>
</tr>
<tr>
<td>(iv) Noscapine</td>
</tr>
<tr>
<td>(v) Papaverine</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>2. Synthetic Compounds</th>
</tr>
</thead>
<tbody>
<tr>
<td>(i) heroin</td>
</tr>
<tr>
<td>(ii) Nalorphine</td>
</tr>
<tr>
<td>(iii) Hydromorphone</td>
</tr>
<tr>
<td>(iv) Methadone</td>
</tr>
<tr>
<td>(v) Dextropropoxyphene</td>
</tr>
<tr>
<td>(vi) Meperidine (Pethidine)</td>
</tr>
<tr>
<td>(vii) Cyclazocine</td>
</tr>
<tr>
<td>(viii) Levallophan</td>
</tr>
<tr>
<td>(ix) Diphenoxylate</td>
</tr>
</tbody>
</table>
Morphine and heroin are the most potent dependence producing derivatives; they bind to \textit{mu} opioid receptors. The other opioid receptors are \textit{kappa} (for pentazocin), \textit{delta} (for a type of encephalin), \textit{sigma} (for phencyclidine), \textit{epsilon} and \textit{lambda}. Heroin (diacetyl-morphine) is two times more potent than morphine in injectable form. Apart from the parental mode of administration, heroin can also be smoked or chased (\textit{chasing the dragon}), often in impure form (popularly known as \textit{smack} or \textit{brown sugar} in India. Due to its higher addictive potential than morphine, heroin causes dependence after a short period of exposure. Tolerance to heroin occurs rapidly and can be increased to up to more than 100 times the first dose needed to produce an effect.

**Acute Intoxication**

Acute intoxication due to opioids is characterized by apathy, bradycardia, hypotension, respiratory depression, sub-normal core body temperature and pin-point pupils. Subsequently, reflexes become delayed, pulse becomes thready and coma may occur in case of a large overdose. In severe intoxication, mydriasis may occur due to hypoxia.

**Withdrawal Syndrome**

Withdrawal symptoms generally appear within 12-24 hours, reach their peak within 24-72 hours and subside within 7-10 days of the last dose of opioids. He characteristic symptoms include lacrimation, rhinorrhea, papillary dilatation, sweating, diarrhea, yawning, tachycardia, mild hypertension, onsomnia, raised body temperature, muscle cramps, generalized bodyache, severe anxiety, piloerectio, nausea, vomiting and anorexia. There can be marked individual differences in manifestation of withdrawal symptoms. Withdrawal syndrome with heroin is more severe as compared to the one seen with morphine.

**Complications**

Chronic opioid use may lead to several complications. The important ones are listed here. One may experience one or more of the following complications:

(i) \textit{Complications due to illicit use} are quite common. Illicit drug is usually contaminated with some toxic additives. Complications generally seen are, parkinsonism, degeneration of globus pallidus, peripheral neuropathy, amblyopia, transverse myelitis.

(ii) \textit{Complications due to intravenous use} are quite common if the needles are exchanged. The user gets exposed to the risk of various infections. The complications generally seen are, AIDS, skin infections, thrombophlebitis, pulmonary embolism, septicemia, viral hepatitis, tetanus, endocarditis.

(iii) \textit{Drug pedaling and criminal activities} may become a part of chronic users’ life leading to various social and legal complications. Production, manufacture, import, export, sale, purchase and even use of opioids is illegal and is liable to be penalized with severest of punishment (under Narcotic Drugs and Psychotropic substances Act 1985 in India). Chronic use also leads to financial and occupational difficulties leading to marital discord and other social problems.
Treatment
Before starting treatment correct diagnosis should be established on the basis of detailed history of drug intake, the examination and laboratory tests. These tests provide an evidence of drug use. These tests include, Naloxone challenge test to precipitate withdrawal symptoms and urinary opioid testing with radio-immunoassay (RIA), free radical assay technique (FRAT), thin layer chromatography (TLC), gas liquid chromatography (GLC), high pressure liquid chromatography (HPLC) or enzyme-multiplied immuno-assay technique (EMIT). After confirming the diagnosis, the treatment can proceed on three lines:

1. Treatment of overdose
2. Detoxification
3. Maintenance therapy

1. Treatment of opioid overdose
Narcotic antagonists (i.e. naloxone, naltrexone) are generally used to treat opioid overdose. Intravenous injection of 2 mg Naloxone followed by repeated injection in 5-10 minutes, causes reversal of overdose. Since naloxone has a short half-life, repeated doses are required every 1-2 hour. General supportive care should be provided along with.

2. Detoxification is the process in which an opioid dependent person is “freed” from opioids. This is usually done by abruptly stopping the opioid, and managing the emergent withdrawal symptoms. Detoxification of opioid dependents is highly successful, relatively cheaper, applicable on a very large scale, associated virtually with no morbidity or mortality and acceptable to almost all patients. Following methods are available for the management of the withdrawal symptoms:

(i) *Use of substitution* drugs such as methadone is quite common though this drug is not available in India, to control withdrawal symptoms. Methadone is relatively less addicting, has longer half-life, decreases possible criminal behaviour and has much milder withdrawal symptoms when stopped. The aim is to gradually taper the patient from methadone. Relapse rates are quite high when methadone is stopped. It is also argued that one dependence is replaced by another.

(ii) *Clonidine* is an alfa² agonist that acts by inhibiting norepinephrine release at the presynaptic alfa² receptor. The usual dose of clonidine is 0.3-1.2 mg. Per day, and it is tapered off within 10-14 days. It is started after stopping the opioid. Clonidine causes excessive sedation and postural hypertension, therefore the treatment is ideally started in the indoor setting. Regular blood pressure monitoring should be done.

(iii) *Naltrexone* in combination with clonidine is used for the treatment of opioid dependence. Naltrexone is a orally available narcotic antagonist blocking the action of opioids in a dependent person and thus causing withdrawal symptoms. These withdrawal symptoms are managed with addition of clonidine for a period of 10-14 days and then stopping it. Subsequently the patient continues on naltrexone alone. Now if
the person takes an opioid, there are no pleasurable experiences as the opioid receptors are blocked by naltrexone. Therefore, this method can be regarded as the combination of detoxification and maintenance treatment. The usual dose of naltrexone is 100 mg orally, administered every alternate day.

(iv) **Other drugs** are also used as detoxification agents. These are:

(a) LAAM (levo-alpha-acetyl-methadol) is not in wide use for opioid dependence and its use as a long-term treatment agent began in late 1960s. Its analgesic effect and delayed action and longer duration of action were first noted in animals in 1948. With subcutaneous injection of 10-30 mg LAAM analgesic effect is produced after 4-6 hours lasting for 48-72 hours. Intravenous administration also produces similar results, while with oral administration effect appears much quicker (within 1-2 hours) and persists for 72 hours.

(b) Propoxyphene

(c) Diphenoxylate

(d) Buprenorphine is a long-acting partial mu-agonist and is commonly used for both detoxification and maintenance treatment.

(e) Lofexidine is a alpha2 agonist like clonidine

3. **Maintenance therapy**

After the patient is detoxified, the next step is to put the patient on maintenance therapy. The patient is maintained on one of the following regimes:

(i) Methadone maintenance (Agonist substitution therapy)

(ii) Opioid antagonists

(iii) Other methods

(iv) Psychosocial rehabilitation

**CANNABIS USE DISORDER**

Cannabis is derived from hemp plant *Cannabis sativa* and different parts of the plant yield the products popularly known as grass, hash or hashish, marihuana, charas, bhang etc. The plant carries more than 400 identifiable chemicals of which about 50 are cannabinoids, the most active being

<table>
<thead>
<tr>
<th>Cannabis preparation</th>
<th>Part of the plant from where it is obtained</th>
<th>THC content (%)</th>
<th>Potency as compared to Bhang</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hashish/Charas</td>
<td>Resinous exudates from the flowering tops of cultivated plants</td>
<td>8-14%</td>
<td>10</td>
</tr>
<tr>
<td>Ganja</td>
<td>Small leaves and brackets of inflorescence</td>
<td>1-2%</td>
<td>2</td>
</tr>
</tbody>
</table>
of highly cultivated plants

<table>
<thead>
<tr>
<th>Bhang</th>
<th>Dried leaves, flowering shoots and cut tops of uncultivated plants</th>
<th>1%</th>
<th>1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hash oil</td>
<td>Lipid soluble plant extract</td>
<td>15-40%</td>
<td>25</td>
</tr>
</tbody>
</table>

**COCAINE USE DISORDER**

Cocaine is an alkaloid derived from the coca bush, *Erythroxylum coca*, found in Bolivia and Peru. It was isolated by Albert Neimann in 1860 and was used y Karl Koller, a Friend of Sigmund Freud in 1884 as the first effective local anaesthetic agent. In the last few decades, cocaine has gained popularity as a street drug, *crack* and can be administered in the body orally, intranasally, by smoking or parenterally, depending upon the preparation available. Commonest forms used are, cocaine hydrochloride and free base alkaloid. Both intravenous use and free base inhalation produce a ‘rush’ of pleasurable sensations.

Cocaine is central stimulant, which inhibits the reuptake of dopamine, along with the reuptake of norepinephrine and serotonin. In animals, cocaine is the most powerful reinforcer of the drug-taking behaviour. Cocaine is sometimes used in combination with opiates like heroin (‘speed ball’) or at times amphetamines. Cocaine has started making its presence felt in India’s bigger cities.

Acute effects are due to central stimulant and sympathomymetic effects as euphoria, confidence, increased energy, increased heart rate and blood pressure, dilated pupils, constriction of peripheral blood vessels and rise in body temperature and metabolic rate. In an intoxicated state, the user presents with papillary dilatation, tachycardia, hypertension, sweating and nausea or vomiting. A hypomanic picture with increased psychomotor activity, grandiosity, elation of mood, hypervigilance and increased speech output may be present. Later, judgment is impaired and there is impairment of social and occupational; functioning.

In various trials it has been found that the effects of cocaine on heart, blood pressure respiratory rate, and mood increase as the dose is raised from 4 mg. To 30 mg. Major effects are observed with the dose 16 mg and above. In higher doses cocaine can cause depression of the medullary centres and death from cardiac, and more often respiratory arrest. Because cocaine causes increased energy and confidence and can produce irritability and paranoia, it may lead to physical aggression and crime.
Chronic effects are generally not marked if cocaine is used for 2-3 times a week for recreational purposes. Taken daily in fairly large amounts, it can disrupt eating and sleeping habits, produce minor psychological disturbances including irritability and difficulty in concentration, and create a serious psychological dependence. Perceptual disturbances (especially pseudohallucinations), paranoid thinking and rarely psychosis also occur in chronic users of cocaine. A runny and clogged nose is common to be seen and that can be treated with nasal decongestant sprays. Less often, the nose can become inflamed, swollen or ulcerated. Rarely, perforated nasal septum is also reported.

Dependence potential varies with the route of administration. Intravenous cocaine use is most powerful drug-reinforcer.

**Withdrawal syndrome**

Cocaine does not produce physical dependence in the sense that alcohol and heroin does but sometimes- mild withdrawal symptoms such as anxiety and depression arise. Physical dependence is very mild if it is there but the psychological dependence is very strong.

**TABLE VI: COMPLICATIONS DUE TO COCAINE USE**

<table>
<thead>
<tr>
<th>The complications of chronic cocaine withdrawal include:</th>
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<tbody>
<tr>
<td>1. Acute anxiety reaction</td>
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<tr>
<td>2. Uncontrolled compulsive behaviour</td>
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<tr>
<td>3. Psychotic episode (with persecutory delusions and yactile and other hallucinations)</td>
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<tr>
<td>4. Delirium and delusional disorder</td>
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<tr>
<td>5. seizures (especially with high dose)</td>
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<tr>
<td>6. Respiratory depression</td>
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<tr>
<td>7. Cardiac arrhythmias</td>
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<tr>
<td>8. Coronary artery occlusion</td>
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<tr>
<td>9. Myocardial infarction</td>
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<tr>
<td>10. Lung damage</td>
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<tr>
<td>11. Gastrointestinal necrosis</td>
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<tr>
<td>12. Fetal anoxia</td>
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<tr>
<td>13. Nasal septum perforation</td>
</tr>
</tbody>
</table>

**INHALANTS**

Volatile solvents have the capacity to intoxicate those who deliberately inhale them. Hundreds of intoxicating volatile products are available in the homes as well as market place much within the reach of adolescents and young people. Use of inhalants is found more common among school going adolescents and inhalation of these agents is a peer-originated and peer-perpetuated activity. There is a wide range of inhalants commonly abused:
(i) Airplane glue  
(ii) Fingernail polish remover  
(iii) Gasoline  
(iv) Paint thinner  
(v) Liquid shoe polish  
(vi) Plastic cement  
(vii) Cleaning fluid  
(viii) Wax strippers  
(ix) Petrol  
(x) Kerosene oil

**Causes**

(i) Indulgence in inhalant use behavior is generally caused by unsuccessful and unrewarding school experiences.  
(ii) Personality deficiencies are reported to be important predisposing factors in confirmed inhalant abusers.  
(iii) Youngsters overwhelmed with anxiety, depression, or both; border-line or over schizophrenia; and those with character disorders employ inhalants in effect at self-treatment for their intrapsychic and interpersonal distress.  
(iv) Social disorganization within the community also contribute to this practice

**Consequences**

(i) Psychiatric features of acute intoxication resemble those of alcohol intoxication except for its brief duration. The period of relative stimulation and of disinhibited behaviour is also similar to that of alcohol. This can result in accidental injury or death and the releasing of aggressive impulses against one’s self or others. Cognitive deficit may occur with extensive and prolonged inhalant abuse. Psychological maturation gets arrested and aberrant behaviour is not uncommon. School-dropout is a usual consequence of inhalant abuse.  
(ii) Physical symptoms may occur due to the toxic effects of the inhalants. Some of the volatile solvents are the known poisons. Carbon tetrachloride is so toxic that it has been removed from the commercial trade, and benzene’s use is limited for the same reason. Hexane and leaded gasoline can cause a serious polyneritis, and the latter is capable of producing encephalopathy. Toluene is involved in dysfunction of kidney, nervous system and bone marrow. Metallic spray paints and other aerosols may have dangers caused by secondary ingredients rather than by solvent themselves. Sudden sniffing deaths have been reported. Ventricular fibrillation and other arrhythmias occur.

**BENZODIAZEPINES AND OTHER SEDATIVE-HYPNOTIC USE DISORDER**

These drugs are generally used in the treatment of anxiety and insomnia. Currently, these are the most often prescribed drugs. Chlordiazepoxide was discovered by Sternbach in 1957 and since then benzodiazepines have replaced other sedative-hypnotic drugs. Benzodiazepines produce their effects by acting on benzodiazepine receptors (GABA-Benzodiazepine receptor complex), thereby indirectly increasing the action of GABA, the chief inhibitory neurotransmitter in the human brain.
Benzodiazepine (or other sedative-hypnotic) use disorder can either be iatrogenic or originating with illicit drug use. Dependence, both psychological and physical, can occur and tolerance is usually moderate.

**TOBACCO**

Tobacco is a plant product derived mainly from *Nicotiana tabacum* and *Nicotiana rustica* grown all over the world and used by people in almost all countries. Tobacco plant is a native of Americas and was brought by the European navigators on their discovery of Americas in 1492. Tobacco leaves contain an active alkaloid, which is highly toxic and develops resistance to its own action and hence highly addictive. Tobacco is used in smokeless form and it is smoked as cigarette, beedi etc. Withdrawal symptoms include restlessness, nervousness, anxiety, insomnia, increased weight. For tobacco-cessation, pharmacological and behavioural techniques are used. First line treatment includes Bupropion (150 mg per day for 3 days and then 150 mg twice a day for 6-7 weeks). Nicotine replacement therapy in the form of nicotine patches, nicotine spray, nicotine lozenges is available. Second-line treatment includes clonidine and amitriptyline. Behaviour therapy and counseling are used along with pharmacotherapy or independently.