Psychiatric disorders occur frequently in HIV-infected patients but the reported prevalence rates differ considerably, depending on the stage of infection and study population. Fact is, though, that there are multiple factors that can have an impact on comorbid psychiatric illness: psychiatric disorders, e.g. substance abuse, can be an independent risk factor for HIV infection. Furthermore, there are the neuropathological effects of the virus itself, and there is evidence that the infection of microglia leads to neuronal damage due to the excretion of neurotoxins. Additionally, opportunistic infections and some of the antiretroviral drugs may cause psychiatric symptoms.

Apart from the affection of the patient’s well-being, psychiatric disorders may lead to problems in antiretroviral therapy: adherence to antiretroviral medication becomes poorer. Therefore, early diagnosis and therapy of psychiatric disorders are of vital importance for HIV-positive individuals (Angelino 2001).

Major depression

Major depression is the most frequently occurring psychiatric disorder in HIV patients. Reports on prevalence rates differ substantially and reach up to 40% (Angelino 2001). Major depression is a severe illness with serious complications: up to 15-20% of all patients with recurrent depressive episodes commit suicide. Further common complications are physical, social or role model function impairment (Low-Beer 2000).

Major depression interferes with all aspects of being and may have a severe impact on quality of life. It is characterized by depressed mood, decreased energy and loss of interest. Patients tend to be unable to experience joy or satisfaction in activities that would usually generate these feelings; they may feel ill, lack energy and experience a sense of doom. Also feelings of guilt, a lack of self-esteem and self-reproach are frequent (Angelino 2001). Additionally, neurovegetative symptoms such as loss of appetite and sleep disturbances with so-called early morning waking or fatigue are common. Furthermore, depressed patients describe somatic symptoms such as pain or vertigo. Often the severity of symptoms changes during the day with greater severity in the morning and relief in the evening. Poor concentration and cognitive impairment, the so-called pseudodementia in depression may also occur. The individual presentation of these symptoms varies notably and may therefore make diagnosis difficult.

Two simple questions, though, may provide valuable hints:

1. During the past month have you often been bothered by feeling down, depressed or hopeless?
2. During the past month have you often been bothered by little interest or pleasure in doing things?
These two questions are being recommended by the U.S. Preventive Services Task Force for screening for depression in primary care. If at least one of the two questions is confirmed by the patient, further diagnostic testing is recommended (Pignone 2002). This screening can be improved by simply inquiring whether help is needed. Asking “is this something with which you would like help?” improves the specificity of general practitioners diagnosis for major depression significantly (Arroll 2005).

The following criteria of ICD-10 should be explored when making a diagnosis of depression:

_a) Pervasive low mood (see above)
_b) Loss of interest and enjoyment (see above)
_c) Reduced energy, diminished activity
_d) Disturbed or increased sleep
_e) Diminished or increased appetite
_f) Poor concentration and attention
_g) Poor self-esteem and self-confidence
_h) Ideas of guilt and unworthiness
_i) Psychomotor retardation or agitation
_j) Ideas or acts of self-harm or suicide

Therapy is indicated if symptoms last for more than two weeks and when at least two of the first three symptoms in addition to at least one of the other symptoms are reported by the patient.

All of these symptoms might occur as a reaction to a stressful life event or sad circumstances. In these cases treatment is not immediately necessary. If the symptoms persist for an unreasonable period of time – more than a couple weeks – a depressive episode might have been triggered. This should then be treated accordingly (Ebert 2001). Aggressive treatment is also obviously necessary in suicidality. HIV-positive patients are more at risk than the general population. The highest rate of suicidal thoughts and attempts occur approximately one to two years after diagnosis of HIV infection. Altogether, though, the rate of suicide among HIV patients has dropped recently – probably due to the improvement of therapy since the beginning of the HAART era (Einsiedel 2001).

**Treatment**

Treatment of depression is based on two principles: medication and psychotherapy. Since we cannot discuss different aspects of psychotherapy in this article, we will focus on pharmacological treatment. In general, treatment of depressed HIV-infected patients does not differ from that of other patients. It is shown in various studies, that antidepressant medication is efficacious in treating depression among depressed, HIV-positive individuals (Himmelhoch 2005). Medication should therefore always be part of a therapeutic regimen. It should consist of acute phase therapy, maintenance therapy and prophylaxis of a relapse of depression. The goal of treatment should be the complete remission of depressive symptoms. After allevia-
tion, treatment should be continued for at least six months. At the end of treatment, medication should be reduced slowly over a period of weeks.

Once antidepressant medication has been initiated, it may take two weeks for patients to experience a benefit. Side effects, however, might occur earlier, and patients should be informed about this. A non-response to treatment is considered when – given a standard dose of medication or therapeutic serum levels have been attained – there is no relevant benefit for the patient after four to six weeks (Benkert 2003).

At such time, a switch to an antidepressant of another class should be considered. Another period of two to four weeks latency for the therapeutic effect has to be taken into account. Alternatively, an augmentation strategy – added medication with e.g. lithium or thyroid preparations – could be started, since effects might be seen earlier. Sometimes the combination of two antidepressants might bring relief. These strategies should only be provided by experienced therapists. Without thorough experience in treating psychiatric disorders, one should concentrate on three to four antidepressant drugs. In this way, side effects and therapeutic benefits can be more easily observed.

The choice of the appropriate antidepressant can be based on the side effect profile, e.g. sedating vs. activating. Previous therapies are important too: a drug that previously had beneficial results in a patient will be effective in this patient again (Ebert 2001).

**Selective serotonin (5-HT) re-uptake inhibitors**

So-called serotonin (5-HT) re-uptake inhibitors (SSRI) are considered to be first-line medication in depressed HIV-positive patients since they are effective and well tolerated. Starting with low doses reduces the probability of adverse effects.

Recently, there have been reports on SSRI medication precipitating suicide, especially in children and adolescents. When looking at available data though, these findings are not consistent and are not easily transferable to adults. In most countries, population suicide rates have fallen in the last years even though significantly more antidepressants - and especially SSRIs - have been prescribed. Furthermore, it is difficult to show effects of medication on suicide since suicide is rare, even among depressed patients, and it is therefore difficult, especially in short clinical trials, to assess the risks of medication-related suicides statistically. However, long-term studies are required to gain further information on benefits and risks of antidepressant medication (Gunnel 2004).

Overall, the risk of suicide for adults does not seem to be increased by medication with SSRIs. This is for instance supported by a recent Swedish database study, examining nearly 15000 suicides that found no increased risk for the treatment of depressed individuals with SSRIs (Isacsson 2005). Nonetheless, doctors should closely monitor patients with psychiatric disorders, regardless of their medication, for suicide risk, and, if indicated, ask for suicidal thoughts or self-harm in order to react promptly.
Table 1: Selective Serotonin (5-HT) Re-uptake Inhibitors (SSRI) *

<table>
<thead>
<tr>
<th>Drug (Trade name™)</th>
<th>Dosage / day (generally once daily administration)</th>
<th>a) Interactions with HAART</th>
<th>b) Evaluation / comments</th>
<th>c) Selected side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Citalopram (Cipramil™, Sepram™)</td>
<td>20 mg in the morning, therapeutic dose is 20-60 mg</td>
<td>a) Lopinavir/r, ritonavir increase citalopram levels</td>
<td>b) effective, well tolerated, non-sedating antidepressant</td>
<td>c) Initially diarrhea, nausea, decreased sexual arousal / erection</td>
</tr>
<tr>
<td>Fluoxetine (e.g. Fluctin™, Prozac™)</td>
<td>10 mg in the morning for 2-3 days, then 20 mg</td>
<td>a) Increased levels of amprenavir, delavirdine, efavirenz, indinavir, lopinavir/r, nevirapine, ritonavir and saquinavir, Nevirapine decreases fluoxetine levels</td>
<td>b) Activating; most clinical trials conducted with fluoxetine</td>
<td>c) see above</td>
</tr>
<tr>
<td>Fluvoxamine (Fevarin™, Fluvoxamin-neuraxpharm™)</td>
<td>50 mg in the morning, after 3-4 days increase dose to 100-200 mg</td>
<td>a) Increased levels of amprenavir, delavirdine, efavirenz, indinavir, lopinavir/r, nevirapine, ritonavir and saquinavir, Nevirapine decreases fluoxetine levels</td>
<td>b) Potent inhibitor of CYP1A2</td>
<td>c) see above</td>
</tr>
<tr>
<td>Paroxetine (Seroxat™, Tagonis™)</td>
<td>10 mg in the morning for 2-3 days, therapeutic dose is 20 mg</td>
<td>a) Lopinavir/r, ritonavir increase paroxetine levels</td>
<td>b) Somewhat sedating, administration at bedtime if possible</td>
<td>c) see above</td>
</tr>
<tr>
<td>Sertraline (Gladem™, Zoloft™)</td>
<td>25-50 mg in the morning, lowest effective dose 50 mg, maximum 150 mg</td>
<td>a) Lopinavir/r, ritonavir increase sertraline levels</td>
<td>b) Non-sedating. In agitation, akathisia, or insomnia, combination with benzodiazepine possible – applicable for all SSRIs</td>
<td>c) see above</td>
</tr>
</tbody>
</table>

* Note: SSRIs should not be combined with monoamin oxidase inhibitors (MAOI) e.g. Molcedemid (Aurorix™). Adjustment of dosage is required in renal or hepatic disorder. (Angelino 2001, Benkert 2001, Einsiedel 2001)
Tricyclic antidepressants

Tricyclic antidepressants (TCAs) – named after their chemical structure which contains three rings – are effective and, in HIV patients, well studied agents. However, side effects are more frequent in this class of antidepressants. Their anticholinergic effects need to be pointed out: they are contraindicated in patients with urinary retention and closed-angle glaucoma and they should be avoided in patients with bundle branch blocks. Furthermore, TCAs are easier to under- or overdose than SSRIs. Serum levels should therefore be obtained if possible.

<table>
<thead>
<tr>
<th>Drug (Trade name™)</th>
<th>Dosage/day</th>
<th>a) Interactions with HAART</th>
<th>b) Evaluation/comments</th>
<th>c) Selected side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amitriptyline (e. g. Saroten™, Laroxyl™, Novoprotect™, Amineurin™)</td>
<td>Initially 2-3 x 25 mg usual therapeutic dose 3 x 50 mg or 2 x 75 mg</td>
<td>a) Lopinavir/r, ritonavir increase amitriptyline levels</td>
<td>b) Promotes sleep. Weight gain, constipation – might be desired side effects</td>
<td>c) Delirious syndrome when fast dose increase</td>
</tr>
<tr>
<td>Clomipramine (Anafranil™, Hydiphen™)</td>
<td>2-3 x 25 mg for three days usual therapeutic dose 3 x 50 mg or 3 x 75 mg</td>
<td>a) Lopinavir/r, ritonavir increase clomipramine levels</td>
<td>b) Initially possible agitation, combination with benzodiazepine possible, also see above</td>
<td>c) Effective in chronic pain</td>
</tr>
<tr>
<td>Doxepin (Aponal™, Sinquan™)</td>
<td>Initially 3 x 25 mg usual therapeutic dose 3 x 50 mg or 3 x 75 mg</td>
<td>a) Lopinavir/r, ritonavir increase doxepin levels</td>
<td>b) see above</td>
<td>c) Often orthostasis</td>
</tr>
<tr>
<td>Imipramine (Tofranil™, Pryleugan™)</td>
<td>2-3 x 25 mg for three days usual therapeutic dose 3 x 50 mg or 3 x 75 mg</td>
<td>a) Lopinavir/r, ritonavir increase imipramine levels</td>
<td>b) see above</td>
<td>c) Especially at the start of therapy anticholinergic adverse effects</td>
</tr>
</tbody>
</table>

For further reading see Angelino 2001, Benkert 2001, Einsiedel 2001
Other drugs / therapies

There are numerous other antidepressants but at the time being there is not much data on their use in HIV-infected patients. These include the noradrenergic and serotonergic drug mirtazapine (unlike SSRIs and tricyclic agents, there are so far no reports on sexual dysfunction with this drug) and the combined serotonin-noradrenaline re-uptake inhibitor venlafaxine. The selective noradrenaline re-uptake inhibitor reboxetine seems to be interesting in the therapy of HIV-infected patients since it is not metabolized via cytochrome P450 (CYP450) (Carvalhal 2003).

Table 3: Other antidepressants

<table>
<thead>
<tr>
<th>Drug (Trade name)</th>
<th>Dosage / day</th>
<th>a) Interactions with HAART</th>
<th>b) Evaluation / comments</th>
<th>c) Selected side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mirtazapine (Remeron™)</td>
<td>Initially 15 mg at bedtime usual therapeutic dose 30-45 mg</td>
<td>a) not known</td>
<td>b) Sedating, promotes sleep, weight gain no sexual dysfunction</td>
<td>c) Cave!: not in leukopenia!</td>
</tr>
<tr>
<td>Reboxetine (Edronax™)</td>
<td>Initially 2 to 4 mg maintenance therapy 8 mg to 12 mg</td>
<td>a) not known</td>
<td>b) not sedating</td>
<td>c) Dry mouth, insomnia, sweating, tremor and urinary retention. Cave!: Dose reduction (2 x 2 mg) in renal or hepatic insufficiency</td>
</tr>
<tr>
<td>Venlafaxine (Trevilor™)</td>
<td>Initially 37.5 mg in the morning administer twice daily maintenance therapy 75 to 375 mg/day</td>
<td>a) Lopinavir-ritonavir, ritonavir increase venlafaxine levels</td>
<td>b) Extended release formulation with lesser side effects. Effective in anxiety</td>
<td>c) Initially high rates of gastrointestinal side effects. RR ↑, allergic skin reactions, delayed ejaculation</td>
</tr>
</tbody>
</table>

For further reading see Angelino 2001, Benkert 2003

New formulations of existing antidepressants are being developed: intravenous formulations with a faster onset of antidepressant action or a once-weekly administered SSRI. (Norman 2004). Furthermore, single enantiomers have been introduced in several countries, e.g. the S-enantiomer of the SSRI citalopram, escitalopram. It is more than twice as potent at inhibiting serotonin uptake and is supposed to maintain therapeutic efficacy at a lower effective dosage. Pharmacokinetic interaction with ritonavir – a CYP3A4 substrate and prototype CYP3A4 inhibitor – which may potentially affect plasma concentrations of escitalopram, was not clinically significant (Gutierrez 2003). None of these agents, however, will be a sovereign remedy, and one should, especially when experience in psychiatric care is limited, only use a few drugs and know them well instead of trying all available substances.

In addition to the above, herbal medicines are also in use, even though there is an ongoing discussion about their effectiveness. There were great expectations espe-
cially about St. John’s wort – a herbal substance without serious adverse effects – when clinical trials demonstrated an antidepressant effect in mild to moderate depression (Linde 1996). Unfortunately hopes have fallen somewhat since St. John’s wort did not show an advantage above placebo in further clinical trials (Hypericum Depression Trial Study Group 2002). Remarkably enough, though, the SSRI in this trial was not very effective either and merely showed a positive trend above placebo in effectiveness.

In addition to the above, there are more therapeutic options aside from medication, e.g. controlled sleep withdrawal, where the patient has to stay awake throughout the night. Following this procedure, there is a significant reduction of symptoms the next day in about one half of treated patients—but only until the next night’s sleep. Repeated sleep withdrawal, though, might reduce the duration of a depressive episode. Phototherapy, especially in seasonal depression, and electroconvulsive therapy carried out in specialized centers for non-responding patients, are therapeutic options too. There are no data on these therapies in HIV patients. Evidence does exist, however, from small clinical trials for a therapeutic effect of exercise in HIV patients (Neidig 2003). Three times a week jogging for half an hour is a good antidepressant and a therapeutic chance that is possibly not tried often enough.

**Psychotic disorders**

*Psychotic* means the occurrence of delusions or prominent hallucinations, and typically the patient has no insight into their pathologic character. The prevalence of psychotic disorders in individuals with HIV or AIDS is rather unclear: rates vary between 0.2 and 15 % (Sewell 1996). Basically, psychotic disorders can be classified into two different forms:

**Primary psychotic disorders**

Psychosis that occurs independently of infection with HIV is to be seen as a comorbid condition. Diseases such as schizophrenia, schizophreniform disorder and brief psychotic disorder can be classified into this group. Typical symptoms are delusions, hallucinations, disorganized speech (e.g. frequent derailment or incoherence) or grossly disorganized or catatonic behavior. Etiopathogenetically, a biopsychosocial concept, the vulnerability-stress-coping model, is assumed. It is thought that genetic and psychosocial factors determine a predisposition or an increased vulnerability for psychotic decompensation.

Therefore, an infection with a neuropathological virus such as HIV could trigger a pre-existing psychosis (Einsiedel 2001).

**Secondary psychotic disorders**

Characteristic symptoms of a secondary psychotic disorder are prominent hallucinations or delusions. They are caused by an organic disorder of the central nervous system (CNS) as a consequence of a general medical condition. In HIV patients this could, for example, be an opportunistic infection, cerebral lymphoma or HIV encephalopathy. In addition to that, psychotic symptoms can be caused by medications or drug-drug interactions e.g. in HAART (Foster 2003). Therefore an exact
The occurring delusional themes are numerous, including somatic delusions, delusions of grandeur, religious delusions, and, most frequently, paranoia or persecutory delusions. Diseases that affect subcortical structures or the temporal lobes are more frequently associated with delusions than others. In hallucinations, every sensory quality (auditory, visual, olfactory, gustatory or tactile) might be affected.

Patients with a previously undiagnosed general medical condition, such as HIV infection, might develop an acute psychiatric condition due, for example, to HIV encephalopathy, brain damage from an opportunistic CNS infection such as toxoplasmosis, neoplasms involving the CNS, or metabolic dysfunction. In all acute psychotic disorders, a magnetic resonance image of the brain (more sensitive than computed tomography) and examination of cerebral spinal fluid should therefore be carried out as soon as possible. HIV infection does not show any specific psychopathological findings (Röttgers 2000).

**Treatment**

While in organic psychosis, the causative general medical condition must be treated first, in primary psychosis, according to its multifactorial etiology, therapy should consist of a combination of pharmacological, psychotherapeutic, psychoeducational and sociopsychiatric intervention.

Symptomatic treatment with neuroleptics is initially the most important line of treatment in the acute phase of primary psychotic disorders. In principle, the pharmacological treatment of HIV patients does not differ much from that of other populations, but it should be started at low doses and titrated cautiously (Farber 2002), since a dysfunction of the blood brain barrier and consequently a higher rate of medication side effects is to be expected: start low, go slow!

In acute psychotic disorder, regardless of the etiology, the use of a conventional antipsychotic agent, e.g. haloperidol 5 mg PO or IM, is usually successful. For additional sedation in cases with more severe agitation, comedication with a benzodiazepine is possible. When aggressive behavior is present, diazepam 5 to 10 mg PO or IM is a good choice; if fear or anxiety is the leading symptom, lorazepam up to 2.5 mg is indicated. In the further course of treatment, change to an atypical antipsychotic agent (see below) is recommended.

In less acute symptomatic psychotic disorders and in primary comorbid psychosis the use of atypical antipsychotic agents is again the treatment of choice, due to various reasons: atypical antipsychotic agents cause significantly less extrapyramidal symptoms (EPS) and tardive dyskinesia (TD) than typical antipsychotic drugs. Furthermore, they might provide an advantage in non-responding patients and in the treatment of negative symptoms: asociality, the withdrawal from relationships; avolition, the loss of initiative and drive; affective flattening or inappropriateness; alogia, a poverty of speech production and content; anhedonia, difficulty experiencing pleasure. These are often the most debilitating symptoms in psychotic disorders. Because of the lower risk of developing EPS and TD – for which HIV-infected patients are more susceptible than others – treatment with atypical antipsychotic agents might improve adherence to psychopharmacological treatment
too. In case of insufficient effectiveness, a different atypical antipsychotic agent should be selected after approximately four weeks.

Table 4: Atypical antipsychotic agents

<table>
<thead>
<tr>
<th>Drug (Trade name)</th>
<th>Dosage/day</th>
<th>a) Interactions with HAART</th>
<th>b) Evaluation/comments</th>
<th>c) Selected side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amisulpirid (Solian™)</td>
<td>twice daily</td>
<td>a) No interaction to be expected</td>
<td>b) Nearly complete renal elimination, poses advantage in patients with liver damage</td>
<td>c) EPS in doses &gt; 400 mg/d possible, usually not severe</td>
</tr>
<tr>
<td>positive symptoms: 400-800 mg</td>
<td>negative symptoms: 50-300 mg</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>maintenance therapy: 200-400 mg</td>
<td>Clozapine (Leponex™)</td>
<td>prescribing doctor needs to register at manufacturers</td>
<td>a) Because of risk of agranulocytosis (1-2 %) in HIV patients not recommended</td>
<td>b) Atypical antipsychotic agent with significance for non-responding schizophrenia and in patients with non-tolerable EPS</td>
</tr>
<tr>
<td>start with 6.25-12.5 mg, increase every 1-2 days by 25 mg to max. 600 mg, maintenance therapy: 100-400 mg</td>
<td>Olanzapine (Zyprexa™)</td>
<td>starting dose 5 mg h.s. maintenance 5-20 mg when sedation during daytime is wanted: two to three doses/day</td>
<td>a) No interaction with PIs</td>
<td>b) Good antipsychotic effect. Few EPS when &lt; 20 mg. Trials with HIV patients available. Side effects, weight gain (depending on dosage) and/or sedation might be favorable.</td>
</tr>
<tr>
<td>Quetiapine (Seroquel™)</td>
<td>start with 25 mg slow titration to 300 to 450 mg divided into two doses/day</td>
<td>a) Contraindication in combination with ritonavir, macrolide antibiotics and ketoconazole.</td>
<td>b) No trials with HIV patients published.</td>
<td>c) Common (&gt;10 %) sedation, drowsiness. Occasionally orthostasis, liver enzymes↑, weight gain. Cave!: Leukopenia</td>
</tr>
<tr>
<td>Risperidone (Risperdal™)</td>
<td>slow titration over one week start with 0.5-2 mg maintenance dose: 4-6 mg divided into two doses/day in renal or hepatic insufficiency do not exceed 4 mg/day</td>
<td>a) NRTIs increase risperidone plasma level.</td>
<td>b) Good antipsychotic effectiveness. Dose dependent EPS: seldom when ≤ 6 mg. Trials with HIV patients published. No influence on blood count, no increase in seizures. First atypical antipsychotic agent available in long acting formulation (twice weekly).</td>
<td>c) Orthostasis, especially in the beginning and at high doses – titrate slowly!</td>
</tr>
</tbody>
</table>
Table 4: Atypical antipsychotic agents

<table>
<thead>
<tr>
<th>Drug (Trade name)</th>
<th>Dosage/day</th>
<th>a) Interactions with HAART</th>
<th>b) Evaluation/comments</th>
<th>c) Selected side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ziprasidone (Zeldox™)</td>
<td>start with 2 x 20 mg Maximum dose is 2 x 80 mg IM administration possible.</td>
<td>a) Not examined</td>
<td>b) So far no trials with HIV population. Contraindicated in patients with long QT interval, cardiac arrhythmias, myocardial infarction. EPS rates not higher than in placebo. Only minimal weight gain.</td>
<td>c) Cave: QTc prolongation! &gt; 1 %: drowsiness, hypotension, sedation</td>
</tr>
</tbody>
</table>

Acute treatment in psychiatric emergency

Most important: de-escalation by “talking down” – this includes measures such as staying in contact with the patient, taking him seriously and adopting a non-confrontational position. Should the use of restraints be necessary, stay calm but act firmly. Always leave the patient the chance to correct inappropriate behavior and always use the least possible restrictive method of restraint.

Table 5: Psychiatric emergency (Benkert 2003; Currier 2004)

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Psychopharmacological treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Agitation in acute psychosis</td>
<td>Haloperidol 5-10 mg PO or IM, may be repeated after 30 min, maximal 50 mg in the first 24 hrs. Cave: EPS – then 2.5-5 mg (1/2-1 Amp.) biperidene (Akinethon™) IV or IM. plus</td>
</tr>
<tr>
<td>Agitation and aggression in mania</td>
<td>oral or IV application of 2 mg lorazepam, when panic is predominant; maximum dose 10 mg / day (inpatient) or diazepam, when stronger sedation is needed; in aggressive patients: 10 mg PO, IM or slowly IV. Repetition after 30 min possible. Maximum dose 40-60 mg parenteral or 60-80 mg oral (inpatient). Cave: hypotension, respiratory depression alternatively oral treatment with 2 mg of risperidone plus 2 mg of lorazepam (Currier 2004)</td>
</tr>
<tr>
<td>Acute intoxication with psychoactive drug</td>
<td>treatment of general medical condition if necessary antipsychotic agent e.g. melperone 50-100 mg for sedation or haloperidol (especially in psychotic symptoms) 2-5 mg PO or IM.</td>
</tr>
<tr>
<td>Delirium due to general medical condition (e.g. infection, exsiccosis, electrolyte metabolism disorder)</td>
<td>change or reduce causative substance in accordance to severity of symptoms, if necessary antipsychotic agent e.g. melperone 50-100 mg for sedation or haloperidol 2-5 mg PO or IM in hospitalized patients if necessary clomethiazole 2 capsules every 2 hours, maximum dose 20 capsules/day. Cave: respiratory depression, hypersecretion; strictly for inpatients only!</td>
</tr>
<tr>
<td>Drug-induced delirium (e.g. antidepressants, antibiotics, rarely efavirenz or others)</td>
<td></td>
</tr>
</tbody>
</table>
References


