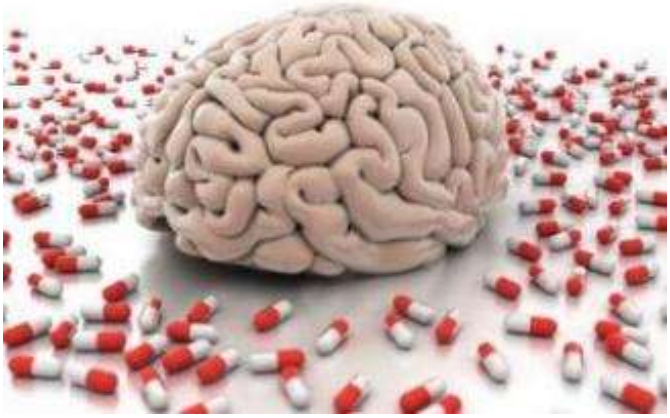

Tardive Dyskinesia

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Tardive dyskinesia, or TD, is a disorder associated with the use of certain types of medications which act upon the nervous system, primarily those used to treat psychiatric disorders. The term *tardive* means delayed onset. The prefix *dys* means difficult, bad, faulty or abnormal and the term *kinesia* refers to movement. The symptoms of tardive dyskinesia include abnormal involuntary movements of the face, lips, tongue, jaw, eyelids, trunk and upper and lower extremities and can range from very mild to life-defining. Swallowing, speech, ambulation, functional living skills and even breathing can all be impacted by this disorder. Undiagnosed tardive dyskinesia is often the source of complex swallowing disorders, primarily the inability to protect the airway due to a reduced or absent gag reflex.

Symptoms may appear anywhere between 3 months and several years after exposure to the offending agents, with the average time of onset being four years. Some individuals do not demonstrate signs of TD until they are being tapered off of the causative medications. Contact does NOT need to be continuous but may be cumulative over a prolonged period of time.

Tardive dyskinesia was first identified in the 1950s following the introduction of the first generation of antipsychotic medications. Introduction of newer psychiatric medications, including SSRI's, was thought to be a solution to the problem of TD, but unfortunately this has not proven to be the case.

"Several studies have recently been conducted comparing the prevalence rate of tardive dyskinesia with second generation, or more modern, antipsychotic drugs to that of first generation drugs. The newer antipsychotics appear to have a substantially reduced potential for causing tardive dyskinesia. However, some studies express concern that the prevalence rate has decreased far less than expected, cautioning against the overestimation of the safety of modern antipsychotics.[41][47]"
http://en.wikipedia.org/wiki/Tardive_dyskinesia

Other medications not used for psychiatric purposes have also been shown to cause TD, including Reglan (metoclopramide) and Phenergen (promethazine), which are used primarily to treat disorders of the GI tract. Depakote (divalproex sodium), used for both anti-epileptic and psychiatric purposes, has also been shown to be a causative agent.

Some individuals are at higher risk than others for developing tardive dyskinesia. These include females, the elderly, those with developmental disabilities or organic brain injuries, cigarette smokers and individuals who have shown previous sensitivities to psychiatric medications.

"Tardive dyskinesia not only may be painful and disfiguring, but it can also predict poor outcome in schizophrenia. Although many treatments have been tried, none have proven completely efficacious. The best treatment even today is prevention." Industrial Psychiatry Journal; 2010 July-December;

Screening tests for tardive dyskinesia include the AIMS and DISCUS. All individuals taking medications with the potential to cause tardive dyskinesia should be formally screened for signs of TD before the medication is initiated and then at appropriate intervals thereafter, normally every six months. Experience has shown that the professionals demonstrating the highest proficiency in identifying tardive dyskinesia symptoms are pharmacists and nutritionists. Additionally, patients and /or their caretakers should receive information about potential side-effects so these may be reported at the earliest possible time. Advocacy is

critical in this area to ensure the best possible outcomes for individuals taking these medications.

Treatment for TD symptoms includes removal, if possible, of the offending agents. For those who show withdrawal signs of TD, the causative medication is usually continued. Other treatments include a number of medications, which reduce the effects on the central nervous system. Cogentin is quite commonly used for treatment of TD symptoms, though the American Psychiatric Association now recommends against the use of this particular medication due to its negative side effects. Vitamins E and B6 have been successfully used to mediate the unpleasant effects of TD. Prevention of symptoms through the use of the fewest possible medications at the lowest effective dosage and the use of non-medication interventions continues to be the best solution for avoiding tardive dyskinesia.

Medscape, Tardive Dyskinesia, last updated Feb. 2012

HRST: Identifying TD Risk and Preventative Actions

Those who use the HRST are undoubtedly familiar with the long list of medications flagged as having a potential association with tardive dyskinesia. And that list continues to grow. We have now identified Depakote and Phenergen (used for nausea and vomiting) as well as all of their generic relatives, as potential causative agents. All medications included in this list, regardless of the reason for their use, will automatically force a score of 4 under the Psychotropic Medications section of the HRST. In the case of Reglan (metoclopramide) and Phenergen (promethazine), a person may have a score of 4 under this section, even if they have never had a psychiatric issue.

So why do we do this? There is one very simple reason. The population most frequently served by the HRST, those with developmental and intellectual disabilities, are also among the most likely to experience this particular side-effect.

Remember from HRST training that the primary reason for scoring is to trigger the considerations that assist with managing potential areas of health risk. Any time an individual receives a score of 4 in the Psychotropic Medications section of the HRST two very important considerations are triggered. One which prompts the prescribing professional to do AIMS or DISCUS screening at appropriate intervals and another which prompts training of those who work or live with the individual to be aware of potential symptoms and to report these to the appropriate professional for further action.