GLYCINE Supplementation ---
as it relates to the insomnia associated with
Dysaerobic, Sympathetic, and Glucogenic Imbalances
as well as excess Histamine and Prostaglandin E2

- Glycine supplementation at bedtime (3 grams) in human volunteers with frequent unsatisfactory sleep ….

  --- improved subjective sleep quality
  --- improved sleep efficacy (sleep time to in bedtime ratio)
  --- shortened polysomnography latency both to sleep onset and to slow wave sleep without changes in the sleep architecture
  --- lessened daytime sleepiness
  --- improved performance of memory recognition tasks.

  This improvement in sleep quality and in mental performance the next day was achieved by an entirely different mechanism and with none of the CNS depressant effects of traditional drugs such as benzodiazepines.

  Patients who suffer from insomnia associated with either a Sympathetic, a Glucogenic, or a Dysaerobic Imbalance, or from excess Histamine or Prostaglandin E2, generally benefit from supplementation with glycine at bedtime. The glycine activates the glycinergic receptors in the brain, thus ensuring deeper more restorative sleep, and awaking the next morning more fully refreshed. 3 grams (6, 500 mg capsules) shortly before bed.

- Various neuronal populations are related to 2 biological rhythms --- the diphasic circadian rhythm of wake/sleep, and the periodic cycles of non-REM/REM sleep. Hypothalamic nuclei are the sources of circadian rhythm and sleep onset control. The control of periodic non-REM/REM cycling is within the pons.

- Glycinergic postsynaptic inhibition is responsible for the atonia of REM sleep --- During REM sleep, only receptors on alpha motoneurons in the trigeminal motor nucleus are excited. These receptors have been identified as glycinergic.

- REM sleep behavior disorder (RBD) (= uncontrolled and often violent movements during sleep) is a neurological disease characterized by loss of normal REM motor inhibition and subsequent dream enactment. RBD is clinically relevant because it predicts neurodegenerative disease onset (e.g., Parkinson's disease) and is clinically problematic because it disrupts sleep and results in patient injuries and hospitalization. The potential for injury to the patient and the patient's bed partner is as high as 96%.

Development of RBD may be one of the first manifestations of Parkinson's disease or other Parkinsonian syndromes. Abnormal inhibitory transmission underlies this disorder. Lab mice with deficient glycine transmission duplicate the cardinal features of RBD. These mice with impaired glycine receptor function exhibit REM motor behaviors, non-REM muscle twitches, sleep disruption, and EEG slowing --- the designing RBD features.

Both clonazepam and melatonin are effective in treating human RBD. Although clonazepam is an effective first-line agent in RBD, melatonin alone or as an adjunct can be equally effective. Particularly for those who experience intolerable side effects with clonazepam, or for those in whom clonazepam precipitates or aggravates obstructive sleep apnea, melatonin is the preferred choice. 0.5 mg of melatonin taken at bedtime is effective in many cases; 1.0 mg of melatonin is effective in most cases. Persistent benefit with melatonin supplementation beyond 1 year of therapy occurs in most, but not all, patients.

[See our MELATONIN Article.]