Abstract
A bibliographic search has been performed in MEDLINE using cortisol and panic as key-words, occurring in the title and/or in the abstract. Human studies were selected, with no time limit. The following publications were excluded: review articles, case reports, panic attacks in disorders other than panic disorder, and studies on changes that occurred in-between panic attacks. The results showed that real-life panic attacks as well as those induced by selective panicogenic agents such as lactate and carbon dioxide do not activate the hypothalamic-pituitary-adrenal (HPA) axis. Agonists of the colecystokinin receptor B, such as the colecystokinin-4 peptide and pentagastrin, increase stress hormones regardless of the occurrence of a panic attack and thus, seem to activate the HPA axis directly. The benzodiazepine antagonist flumazenil does not increase stress hormones, but this agent does not reliably induce panic attacks. Pharmacological agents that increased anxiety in both normal subjects and panic patients raised stress hormone levels; among them are the α2-adrenergic antagonist yohimbine, the serotonergic agents 1-(m-chlorophenyl) piperazine (mCPP) and fenfluramine, as well as the psychostimulant agent caffeine. Therefore, the panic attack does not seem to activate the HPA axis, in contrast to anticipatory anxiety.

Keywords
stress hormones, panic attack, anxiety.