tency with either a categorical view of panic disorder as a stable clinical entity or panic disorder as one facet only of a 'general neurotic syndrome'.

Result: PSE profile at baseline was virtually identical with that at follow up. Few patients had 'changed' in PSE syndrome diagnosis after 5 to 6 years.

Conclusion: These data, although not conclusive, are supportive of the concept of DSM III-R Panic Disorder as a stable clinical entity and are correspondingly difficult to reconcile with the view that Panic Disorder is but one facet only of a general neurotic syndrome.

"FEAR FIGHTER," BEHAVIOURAL SELF-HELP FOR AGORAPHOBIA ON COMPUTER MULTIMEDIA — A NEW SYSTEM

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Computer aids in medicine have been available for many years mostly to do assessment or to collect data. Computer aids for treatment are still uncommon. A behavioural self care system has been computerised and subjected to prepilot testing.

10 agoraphobic patients who were either receiving treatment or had done so previously were asked to comment on Fear Fighter and compare it to their experience of face to face behaviour therapy. All 10 patients felt that Fear Fighter was user friendly, even those who had not used a computer before. Although they could not comment on Fear Fighter's efficacy as they were not using it as a self treatment tool, all 10 patients felt keenly that "Fear Fighter" could be used for self-treatment and could have helped them. The use of multimedia, in particular female voice-over accompanying text, was seen as a great advantage in making the computer system feel friendly.

THE BENZODIAZEPINE ANTAGONIST FLUMAZENIL IS ANXIOLYTIC IN HIGH STATE ANXIETY AND ANXIOGENIC IN LOW STATE ANXIETY IN A STUDY OF 23 FEMALE ANIMAL PHOBICS

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Objectives: Flumazenil (Ro 15–1788) initially was believed to be a pure benzodiazepine receptor antagonist, exerting no intrinsic effects. Later in several studies evidence for agonistic or inverse agonistic effects of flumazenil was found. In humans flumazenil has been shown to be anxiogenic, e.g. [1], or anxiolytic [2]. On the other hand it has been asserted that "flumazenil exhibits virtually zero intrinsic efficacy" [3]. File and Hitchcott [4] explained these contradictions by suggesting that flumazenil normalizes the benzodiazepine receptor, returning it to a baseline state. The theory states that flumazenil effects depend on the anxiety level: When anxiety is high, flumazenil reduces it; when anxiety is low, flumazenil increases it. Our study tested these assumptions.

Methods: 23 female spider phobics were diagnosed as having simple phobia according to the criteria of DSM-III-R. They were about to receive exposure therapy and were given either an infusion of 2 mg flumazenil in 0.9% saline solution at a rate of 0.2 mg per minute or saline solution 0.9% alone under randomized double-blind conditions. Immediately after the infusion patients were asked to rate their pre-infusion state anxiety and their maximum state anxiety (if increased) or minimum state anxiety (if decreased) during the infusion on a scale from 0 to 10.

Results: Mean anxiety ratings were not significantly changed by the infusion in either group. While retrospective ratings of state anxiety before the infusion and ratings of anxiety experienced during the infusion were positively correlated for placebo subjects ($r = 0.68$, $p < 0.02$), they were negatively correlated in the flumazenil group ($r = -0.73$, $p < 0.02$): phobics with high ratings of pre-infusion state anxiety showed low ratings of anxiety while receiving flumazenil and vice versa.

Conclusion: The findings of our study support the theory suggested by File and Hitchcott.

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DECREASED PERIPHERIAL-TYPE BENZODIAZEPINE RECEPTOR IN POSTTRAUMATIC STRESS DISORDER

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Peripheral-type benzodiazepine receptors (PBR) have been located in various peripheral organs and in non-neural tissue in the brain. PBR is involved in steroidogenesis and is sensitive to stress and hormonal changes. In a previous study we demonstrated that PBR is decreased in patients with generalized anxiety disorder (GAD) but not in obsessive-compulsive patients (OCD). In the present study we assessed [^{3}H] PK 11195 binding characteristics in posttraumatic stress disorder (PTSD). Eighteen PTSD patients and 7% age-and sex-matched controls participated in the study. All subjects underwent a psyciatric interview using the Structured Clinical Interview for DSM-III-R-Patient Version. Symptom severity was assessed using: DSM-III-R Scale for PTSD, Impact of Event Scale, Beck Depression Inventory, State-Trait Anxiety Inventory. All psychological measures were significantly higher in PTSD patients when compared to control subjects ($p < 0.001$). Decreeased platelf PBR density (−62%; $p < 0.001$) was observed in the PTSD patients when compared to controls. The results in the present study are in accordance with the finding in GAD patients, but differ from those obtained in OCD patients. It is possible that the reduction in PBR density is an adaptive response in order to avoid chronic hypercortisolemia secondary to stress.

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