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Opinion Article

A Study of Anxiety Disorders and Neurological Diseases

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DESCRIPTION

The 1999 annual meeting of the Anxiety Disorders Association of America provided an outstanding venue and abundant intellectual stimulation for satellite scientific conference focused on issues of development and vulnerability. It is within living professional memory that mental states in clinical medicine were understood as radically demarcated from their underlying neuroanatomy, neurochemistry, and neuropsychology. The behavioral disorders were relegated to the realm of the "functional," whereas all the rest of clinical medicine was navigated to the "organic" subcontinents of neurology, cardiology, and general medicine. The bridging insights of neuropsychiatry over the past 20 years have greatly improved our insights about the interactive nature of neurology and psychiatry. While there may be many paths to the functional states that are diagnosed as anxiety and mood disorders, this symposium explored in some detail the model that such disorders result from a combination of genetic vulnerability and adverse early life experience, with this double impact producing profound, long-lasting alterations in stress responsiveness along multiple dimensions. In such a model, this resulting sustained deregulation constitutes, or is integral to, a primary pathophysiological substrate for anxiety disorder. The anxiety disorders may be the most common of the psychiatric disorders. Lifetime prevalence rates for DSM-III-R-diagnosable anxiety disorders are as high as 30.5% for women and 19.2% for men. These disorders occur through out the life span, from childhood to later years.

They include a variety of distressing symptoms including nervousness, sleeplessness, hypochondriasis, and many somatic symptoms. They tend to persist, and they are difficult to diagnose and treat. The majority of the presentations addressed issues arising in the vast and complex arena of the phenomena that must be called on to link the molecular and cellular mechanisms of development, stress-related regulation, genetic vulnerability, and behavioral substrates to the everyday experience of anxiety disorders and their treatment. The observation that persons suffering from certain neurological diseases develop certain behavioral syndromes at incidence rates greater than expected has been a most useful insight for neuropsychiatry. Such observations have substantially shaped our theoretical understanding of the affective disorders, obsessive compulsive disorder, and the psychoses. Another major area of basic neuroscience progress has been functional and structural studies of the hippocampus. A focus for several of the symposium presentations was the integral role of the hippocampus in modulating stress responses and, in turn, it's possible role as a crucial target for the long-term impact of stress on brain circuitry and behavior. The anxiety disorders, however, have not received analogous clinical neuropsychiatric investigation. We do have certain neurobiological insights concerning the neuroanatomy and neurochemistry which is of importance to the clinical expression of generalized or episodic anxiety however; these insights derive primarily from animal experiments, or from functional imaging in humans. Disease-based neuropsychiatry has yet to make its contributions to our understanding of the anxiety disorders. It is also interesting that this neuroendocrine structural perspective, with possible hippocampal degeneration resulting from early and/or sustained stress, brings these dimensions back into focus after a decades-long concentration on brain neurotransmitter deregulation, especially of the monoamine transmitters, as the focal point for hypotheses about pathophysiology. Both episodic and generalized anxiety syndromes have been reported to occur in PD populations at elevated rates compared with normal and disease controls. Some of these patients clearly seem to have onset of the anxiety symptomatology prior to first motor symptoms of PD, suggesting some underlying, shared neurobiological vulnerability to PD and anxiety. Another major development in basic neuroscience, which may well have a substantial impact on how the possible neural substrates of both mood and anxiety disorders are conceived, is a shift in how the processes of neuronal plasticity and sustained neurogenesis are viewed. It is not difficult for us to conceptualize neuroanatomical structural circuits that might sub serve the reported clinical link between anxiety disorders and extrapyramidal movement disorders. From the work of Mogenson and colleagues in the 1970s the nucleus accumbens has been described as an interactive neural relay, modulating striatal motor system output by ventral tegmental and temporal

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lobe limbic inputs. The impact of these new perspectives on thinking about anxiety disorders is enhanced because a major site for such phenomena is the hippocampus, which, as noted above, is already receiving intense scrutiny as a potential site for profound involvement in, and exhibiting consequences of, altered stress responses.

For example, recent observation indicates that the dentate gyrus of the hippocampus is a site of neurogenesis well into adulthood, making it a likely target for mediating the effects of experience on subsequent brain structural changes. The shell of the accumbens is closely linked to or continuous with the anterior extension of the amygdala. Together, these structures provide circuitry linkage between the extrapyramidal motor system and limbic structures, which are activated in stress response and fear. Brain imaging studies in a few psychiatric patients with anxiety have noted temporal lobe and the basal ganglion metabolic changes. The basic observations about this type of lifelong possible substrate for the integration of experience into neuronal circuitry are exciting, but investigation of whether and how this substrate might actually mediate such functional effects is just beginning. In any case, the data available to date indicate that this system may well constitute an exquisitely sensitive index of the structural impact of sustained stress on the brain. Much of the data presented at the symposium has implications for both anxiety and mood disorders, and for their comorbidity. It is not surprising that the neuroscience literature regarding the brain circuitries that are the likely players in such disorders is directed toward extensively overlapping neural elements that cannot be cleanly separated.