Int. J. Prenatal and Perinatal Psychology and Medicine Vol. 12 (2000) No. 4, 569–577

Prenatal Learning of Emotionality and Its Unlearning through Meditation: A Possible Neurobiological Explanation

Erika Gsell

Rome, Italy

Keywords: high emotionality, hyperarousability, prenatal sensitization, foetal trauma, meditation

Abstract: High levels of emotionality have been found to be positively correlated with somatic symptoms, low control over emotional behavior, socially maladaptive or problematic behavior, inability to cope with stress or tendency to engage in emotion-oriented coping. There is agreement between researchers that high levels of emotionality are characterized by cortical and subcortical hyperarousability. In this regard, the following hypotheses are posited: hyperarousability might be caused by prenatal sensitization, i.e. synaptic morphological changes due to repeated overstimulation in utero as a consequence of maternal stress and fetal traumas; and these changes might be reversed by long-term, regular meditation inducing a synaptic desensitization.

Zusammenfassung: *Pränatales Erlernen von Emotionalität und deren Korrektur durch Medidation: Eine mögliche neurobiologische Erklärung.* Es konnte gezeigt werden, daß hohe Emotionalität mit somatischen Symptomen, mit schlechter Kontrolle von emotionalem Verhalten, mit unangepaßtem oder problematischem Sozialverhalten sowie mit mangelhafter oder fehlender Streßbewältigungsfähigkeit einhergeht. Zwischen Forschern besteht Übereinstimmung darin, daß ein hoher Grad an Emotionalität durch kortikale und subkortikale Übererregbarkeit charakterisiert ist. Deshalb werden folgende Hypothesen aufgestellt: Die Ursache dieser Übererregbarkeit könnten pränatale morphologische Veränderungen an den Synapsen (Sensibilisierung) darstellen. Diese sind auf eine vielfache Überstimulation in utero infolge von mütterlichem Streß und fötalen Traumata zurückzuführen. Diese Veränderungen können durch eine regelmäßige, langfristige und damit zur synaptischen Desensibilisierung führenden Meditation rückgängig gemacht werden.

*

Correspondence to: Erika Gsell, Via d. Fratte di Trastevere 48, 00153 Rome, Italy

Foreword

Since my earliest childhood, I have suffered from high levels of emotionality that persisted after many years of intense psychotherapy but progressively diminished through regular Buddhist practice. Recently, I started looking for a possible neurobiological explanation for my experience. The present paper is the result of my research. It makes no claim to being a scientifically faultless work but should rather be considered as a basis for discussion.

High Levels of Emotionality: Effects and Neurobiological Basis

Emotionality is an important aspect of temperament [1]. It determines the sensitivity to emotional stimulation and the level of affective reaction towards it (Remark 1). High levels of emotionality have been found to be positively correlated with somatic symptoms, low control over emotional behavior, socially maladaptive or problematic behavior, inability to cope with stress or tendency to engage in emotion-oriented coping (Remark 2). [2–9]

Several authors have linked intense emotional responsiveness with higher levels of cerebral arousability. Jung [10] attributed the extreme emotional sensitivity of the introverted type to external stimuli to the "intensity of the primary and the prolongment of the secondary cerebral function" (Remark 3), which produce – on the psychic level – the persistence of affective vibrations, that render the person incapable of being rapidly submitted to new stimuli (Remark 4)

The concept of introversion was further elaborated by Eysenck [12]. Although he principally postulated the existence of two clearly distinct types of excitation – cortical arousal, associated with introversion, and autonomic activation, associated with emotionality (Remark 5) – he conceded that activation and arousal tend to become synonymous "when strong emotions are involved, frequently and for long periods" and that this distinction tends to break down when highly emotional people are involved, i.e. people for whom even quite mild stimuli are emotionally activating. In a later work [13] he again stated that the apparent diversity of multiple arousal mechanisms "may not prevent the systems from operating in a relatively unified fashion."

Recent neuroscientific and neuropsychological research has confirmed that, despite the multidimensionality of the arousal concept [14], a certain degree of interrelation between the different cerebral systems exists. Austin [15] points out that "the brain has reverberating circuits and therefore rhythmic firings in one part of the brain go on to influence the excitability of other nerve cells some distance away, and are then influenced in return". Robinson [16] has shown that cerebral arousability is a primary and direct determinant of differences in emotionality (Remark 5), and Stelmack [17] has demonstrated that introverts display enhanced responsiveness on measures of autonomic as well as electrocortical activity. Selva [18] stresses that many limbic structures are associated more with memory and learning processes than with emotional life and Damasio's research [19] has clearly shown that emotions involve the cortical as well as subcortical activation: if the brain mechanisms for primary emotions can be found in the subcortical structures, especially in the amygdala, the mechanisms that implement secondary emotions are based in the neocortex, particularly in the frontal lobes.

Prenatal Learning of Emotionality/Arousability?

Whereas the above-mentioned research appears to prove with enough evidence that high levels of emotionality are characterized by high levels of cortical and subcortical arousability, it is much more difficult to time and determine their causes.

Emotionality, as well as other aspects of temperament, is observable at birth: some newborns are extremely excitable, irritable and vulnerable to stimuli while others are more tranquil and less reactive.

According to the extreme biological position, which links specific genes to different behavioral traits [20], introversion and emotionality are present at birth and are thus hereditary. In this sense, Kagan et al. [21] suggest that "most of the children we call inhibited (Remark 6) belong to a qualitatively distinct category of infants who were born with a lower threshold for limbic-hypothalamic arousal to unexpected changes in the environment or novel events that cannot be assimilated easily . . . ".

"Present at birth" does not, however, necessarily mean hereditary. As Winnicott [22] pointed out as far back as 1949, ". . . a severe (psychological) birth trauma can cause a condition which I will call congenital, but not inherited, paranoia . . . one finds clinically an apparent mental defect, this in spite of the original normal brain tissue development."

In the meantime, the hypothesis that temperament is influenced by intrauterine and perinatal factors has been confirmed. Reliable scientific studies have clearly demonstrated that human fetuses are able to learn and memorize stimuli [23, 24]. Evidence of a functioning fetal memory is provided by studies using three different learning paradigms: habituation, classical conditioning and exposure learning (Remark 7).

Neurobiological research [25–27] has demonstrated that, due to the plasticity of neurons at the cortical and subcortical levels, the synaptic connections of the brain are not irreversibly determined by genotypic instructions, but are influenced by experiential sensory inputs (Remark 8). Synaptic activity does not only modify synaptic functions but induces structural changes [28, 29].

Van de Carr [30, 31] and Blum [32, 33] point out that stimuli present in the development period of maximum plasticity of the brain structures (Remark 9) will result in very basic developmental effects and therefore in most cases irreversibly affect the later stages of human cognitive and emotional growth (Remark 10). Consequently, they highlight the need for sufficient and appropriate stimulation (Remark 11).

Whereas Van de Carr and Blum emphasize the danger of understimulation and sensory deprivation, I would like to point out the risk of overstimulation and excess of input and put forth the question: could emotionality, in the sense of hyperarousabilty, not possibly be a consequence of prenatal sensitization (Remark 12) due to maternal stress and fetal traumas?

In his extensive review of the research in the field of the neurobiology of early trauma [34], Lloyd de Mause has shown that fetal traumas can lead to persistent hyperarousal, due to the overstimulation of synapses that result in hypersensitivity and imbalances in important neurotransmitters, such as catecholamines. Early trauma, he further points out, is encoded in the emotional memory system that is

based, as we have seen before, in the amygdala and the prefrontal cortex. Maternal stress in pregnancy has proved to have other long-term-neurodevelopmental effects: in offspring, neurodevelopmental deviance, adverse developmental and psychiatric outcomes [35]; in adulthood, increased basal cortisol levels and raised adrenocorticotropin responses during stress [36] and long-term changes in the expression of neuropeptide receptors, in the form of decreased mu opiate receptors (Remark 13) in selected brain areas such as striatum and amigdala [37].

Unlearning through Meditation

Is the overstimulation of synapses – with the morphological changes which occur (cf. Remarks 11 and 12) – an irreversible process? From a psychological point of view, the question would be: can high levels of emotionality be reduced? As I pointed out in the foreword, I have personally experienced the reduction of high levels of emotionality as a consequence not of psychotherapy but of regular Buddhist practice (Remark 14). In this part of my work I will try to explain, inferring from the strictly related field of anxiety disorders, why and how this is possible.

Anxiety disorders and high emotionality appear to be tightly connected: as a trait or state (Remark 15), anxiety is characterised (Remark 16) primarily by autonomic arousal [39]. Furthermore, and as Goleman [40] points out, the key symptom of chronic anxiety is a prolonged phase of recovery from stress: the person is unable to interrupt the reaction and remains in an aroused state even after the event is over. This recalls the theory of the protracted secondary function of introverts as postulated by Jung (Remark 3).

From a neurobiological point of view, as Ledoux [41] explains, the psychotherapeutic (in particular psychoanalytic) treatment of anxiety disorders aims at creating a synaptic potentiation in brain pathways that control the amygdala, by explicit knowledge through cortical areas involved in conscious awareness. However, he further points out, "as the connections from the cortical areas to the amygdala are far weaker than the connections from the amygdala to the cortex, it is easy for emotional information to invade our conscious thoughts but so hard for us to gain conscious control over our emotions". As Goleman stresses (ibid.), meditation, on the contrary, is not an attempt to control the mechanisms that underly mental processes, but aims to change them; the behavioral and personality change is a consequence of the changes that occur through the voluntary self-regulation of mental states.

Research evidence [40, 42–46] is generally consistent with the hypothesis that meditation (Remark 17) reduces state and trait anxiety, and increases the ability to withstand stress and to recover more rapidly from stress and stressful stimulation, as measured by a variety of autonomic indices, including GSR (Remark 18).

There is agreement between researchers that long-term, regular meditation (Remark 19) in all its forms (Remark 20) most probably produces its effects through the reduction of somatic arousal and activation (Remark 21), due to the decrease in oxygen consumption and carbon dioxide elimination associated with the reduction of rate and volume of respiration [44–48].

As Austin [49] points out, breathing more quietly and prolonging the phase of expiration quiets the brain through the slowing down of the firing activity of many

nerve cells (Remark 22). For example, nerve cells of the central nucleus of the amygdala and hippocampus fire measurably less whenever breathing is quieter and when expiration is prolonged [50, 51]. This reduced firing in the amygdala and hippocampus could itself contribute to physiological calming [52].

I hypothesize that long-term, regular meditation reduces hyperarousability by reversing – through desensitization – the morphological changes which had occurred in the synapses as a consequence of overstimulation in the very first stages of development. This is the mechanism through which it may occur: whereas repeated excessive stimulation, with its corresponding neurotransmitter density, alters (through sensitization) the presynaptic receptors (terminals), creating more active and larger zones (cf. Remark 12), the quieting down of neuronal firing activity, linked to a decreased flow of neurotransmitters, slowly desensitizes, i.e. reduces the diameter and activity of the same presynaptic receptors, inducing a physiological condition of normal arousability.

This neurobiological mechanism can be compared to the one involved in antidepressant therapies (Remark 23), in which clinical improvement is obtained after several weeks of treatment, because a prolonged administration of antidepressants produces a decrease in number and a desensitization of the presynaptic and postsynaptic receptors that are needed for the full expression of the pharmacological activity in terms of neurotransmitter levels [54, 55].

Remarks

1 There is no widely accepted agreement on the definition of emotionality. Some authors [2, 4, 5, 8, 9] prefer the term "affect intensity" to define the strength with which individuals experience their emotion, because they consider emotionality to be related to the regular experience of negative emotion. On the contrary, for other authors [3, 6, 7, 18] – and in line with Eysenck and his school [12, 13] – emotionality is a more general dimension which produces the amplification of all emotional responses. It is in this latter sense that I will use the term throughout my paper.

2 Semmer [11] points out that if one defines coping as an "attempt to do something about a stressful situation", it is doubtful whether emotion-oriented coping can really be called coping or may not be better regarded as "distress proneness vis-a-vis a potentially stressful condition".

3 Jung refers to "Die zerebrale Sekundärfunktion" written by Otto Gross in 1902. Gross defines cerebral "secondary function" as the process of regeneration which follows the "primary function", i.e. the firing of the cell with its chemical discharge. The secondary function will take more or less time according to the intensity of the previous loss of energy. Particularly strong psychic processes, having a strong affective tone, will involve a particularly high loss of energy and therefore the regeneration phase of the cell will last longer, i.e. the secondary function will be protracted.

4 Jung points out that for this reason, introverts try to protect themselves from external stimuli, appear inhibited and absorbed in their own thoughts, feel more at ease in their interior psychic world than in exterior life, and tend to misanthropy and solitude [10].

5 In his first works, Eysenck used the term neuroticism instead of emotionality. Although some authors (e.g. Robinson) employ the term neuroticism even today, for the sake of clearness I use the term emotionality throughout this paper.

6 Kagan et al. State, interestingly, "... it is important to differentiate between those children and adolescents who are quiet and restrained in unfamiliar social situations because of the influence of temperamental factors and those who behave this way because of environmental experiences alone. We suspect that the contemporary construct of introversion (referring to Eysenck), usually applied to adults, contains both types. Finally we note that these data support Jung's claim (cf. Psychological Types), which Freud rejected, that temperamental factors contribute to the development of social anxiety and avoidance and to the symptoms of panic and agoraphobia that had been classified earlier in the century as components of hysteria".

7 In classical conditioning, a previously neutral stimulus, following a number of pairings with a response eliciting stimulus, elicits a reaction when presented alone; in exposure learning, familiar and unfamiliar stimuli elicit a differential response; in habituation, a decrease in response follows repeated presentation of the same stimulus [24].

8 Edelman has identified and explored the two key levels at which biological brains establish neuronal connections: the developmental processes during embryogenesis that establish general neuronal connectivity patterns (for the primary repertoire) and the neuronal group selection based on experience for the secondary repertoire (The Rembered Present pp. 59–88 Italian edition)

9 Blum [32, 33] calls it the protodevelopmental period. It includes in this definition 36 postnatal months as well as eight to ten months of prenatal life.

10 Van de Carr [30] asks: "Can such things as how fast an individual assimilates and responds to sensory data be modified during this time?" (p. 481)

11 Blum [32, 33] explains in detail how, through an external stimulation, a morphological change of a synapse occurs: "Through external stimulation electromagnetic action potentials are generated within the neural network. When an action potential arrives at the presynaptic terminal, it causes the fusion of vesicles to the presynaptic membrane, the release of neurotransmitters, and a flux of ions across the postsynaptic membrane. In conjunction with the release of transmitters there is the appearance of neural cell adhesion molecules (NCAM) at the surface of the presynpatic cell and a transient flux of free extracellular CA(++) into the synaptic cleft. These two events increase the adhesion of both cell types leading finally to a decrease in the synaptic cleft width and an increase of the diameter of the presynaptic membrane surface. Such a synapse, which has been morphologically modified by neuronal activity, will be more efficient due to an increase in the ratio of the presynaptic surface area to the synaptic cleft volume, and in this way, due to the corresponding increase in neurotransmitter density. Such an activity-dependent modification of a synapse can serve as some kind of memory, comparable to long-term potentiation and presynaptic facilitation".

12 Sensitization is an alteration of presynaptic terminals as a consequence of repeated excessive stimulation: there are more active and larger zones on the terminal end, allowing neurotransmitters to pulse freely through the critical synapses [29].

13 Insel [37] stresses that, as opiate receptors (particularly mu receptors) and opiate peptides develop early in ontogeny, stress-induced increases in endogenous opiates could lead to a lasting homologous down regulation for these abundant receptors in the fetal brain or – as an alternative explanation – that opiate receptors are not decreased per se, but that either more cells or more processes with opiate receptors are eliminated during prenatal stress.

14 As I have more extensively explained elsewhere [38], the Buddhist practice I refer to consists primarily in the chanting of the mantra Nam-myoho-renge-kyo, which means: "I dedicate my life to the mystical law of the Lotus-Sutra".

15 Trait anxiety refers to the general tendency to be anxious, as distinguished from state anxiety, i.e. the degree of anxiety at a particular moment [42].

16 However, anxiety as a trait or state cannnot be explained by autonomic arousal alone; it is its occurence in seemingly harmless situations that makes them distressful to the person [39].
17 Although Eppley et al. [42] "strongly recommend that researchers not treat meditation as a generic independent variable but attend and report the specific kind of meditation that they are using", among other authors [40, 44–46, 48] a consensus exists regarding the efficacy of all meditation techniques in lowering the anxiety-level and in helping govern stress.
18 In TM practitioners, low levels of spontaneous GSR (galvanic skin response) were observed. GSR is the resistance of the skin to an electric current. It reflects the level of anxiety and the lability or stress level of an individual. Lower frequencies of spontaneous GSR (i.e. a rise in resistance) reflect greater relaxation and emotional stability [43, 44].
19 Physiologically, the state produced by meditation has been found to be distinct from commonly encountered states of consciousness such as wakefulness, relaxation, sleep or dreaming, and from altered states of consciousness such as hypnosis or autosuggestion [44–48].
20 Austin [49] describes meditation with the following words: "Sitting quietly for 20 minutes once or twice a day, taking a passive attitude and using a simple concentration device. This usually implies focusing on the breathing, repeating some simple word which is in keeping with one's belief system, or chanting a Sutra (Remark 14)".
21 Some authors [44, 46] have specifically pointed out the decreased secretion of norepinephrine due to the reduced activity of the major sympathetic network.
22 This phenomenon, Austin [49] points out, has been observed to an even higher degree in cats, in which each prolonged expiration inhibits not only many of the single inspiratory nerve cells in the medulla, which are influenced by respiration, but other nerve cells as well [53].
23 I am grateful to Dr. Stefano Cavallari for having drawn my attention to and supplied me with literature on this point, thus providing me with ... the missing link in the chain of my evidence ...

References

[1] Lazarus RS (1991) Emotion and Adaptation. Oxford University Press, pp. 297–348
[2] Larsen RJ, Diener E (1987) Affect Intensity as an Individual Difference Characteristic: A Review. J of Research in Personality 21: 1–39
[3] Korotkov D, Hannah E (1994) Extraversion and Emotionality as Proposed Superordinate Stress Moderators: A Prospective Analysis. Personality and Individual Differences 16(5): 787–792
[4] Flett GL et al. (1989) Affect Intensity and Self-Control of Emotional Behaviour. Personality and Individual Differences 10(1): 1–5
[5] Kardum I (1999) Affect intensity and frequency: their relation to mean level and variability of positive and negative affect and Eysenck's personality traits. Personality and Individual Differences 26: 33–47
[6] Belsky J et al. (1996) Infant Positive and Negative Emotionality: One Dimension or Two? Developmental Psychology 32(2): 289–298
[7] Eisenberg N et al. (1998) Shyness and Children's Emotionality, Regulation, and Coping: Contemporaneous, Longitudinal and Across-Context Relations. Child Development 69(3) 767–790

[8] Catanzaro SI (1997) Mood Regulation Expectancies, Affect Intensity, Dispositional Coping, and Depressive Symptoms: A Conceptual Analysis and Empirical Reanalysis. Personality and Individual Differences 23(6): 1065–1069
[9] Flett GL et al. (1996) Affect Intensity, Coping Styles, Mood Regulation Expectancies and Depressive Symptoms. Personality and Individual Differences 20(2): 221–228
[10] Jung CG (1925) Psychologische Typen. Rascher, Zürich Leipzig Stuttgart, pp 383–404
[11] Semmer N (1997) Individual Differences, Work Stress, and Health. In: Schabracq MJ, Winnubst JA, Cooper CL (eds.) Handbook of Work and Health Psychology. Wiley, Chichester
[12] Eysenck HJ (1967) The Biological Basis of Personality. American Lecture Series Nr. 689. Charles C. Thomas, Springfield, Ill., p 233
[13] Eysenck HJ (1990) Biological dimensions of personality. In: Pervin L (ed.) Handbook of Personality, theory and research. Guilford Press, New York, pp 244–276
[14] Frijda NH (1990) Emozioni. Società editrice il Mulino, Bologna, pp 245–252
[15] Austin JH (1998) Zen and the brain: toward an understanding of meditation and consciousness. The MIT Press, Cambridge (Mass.) London, p 87
[16] Robinson LR (1998) Sex differences in brain activity, personality and intelligence: a test of arousability theory. Personality and Individual Differences 25: 1133–1152
[17] Stelmack RM (1990) Biological Bases of Extraversion:Psychophysiological Evidence. Journal of Personality 58(1): 293–311
[18] Martinez-Selva JM (1997) Psicologia y psicobiologia de las differencias individuales desde la perspectiva de Eysenck. Anales de Psicologia 13(2): 111–117
[19] Damasio AR (1994) Descartes' Error: Emotion, Reason, and the Human Brain. Avon Books, New York, chap. 7
[20] Hamer DH, Copeland P (1998) Living With Our Genes: Why They Matter More Than You Think. Doubleday, New York
[21] Kagan J et al (1988) Biological Bases of Childhood Shyness. Science 240: 167–171
[22] Winnicott DW (1992) Birth Memories, Birth Trauma, and Anxiety. Int J Prenatal and Perinatal Studies 4(1/2): 17–33
[23] Riese ML (1990) Neonatal Temperament in Monozygotic and Dizigotic Twin Pairs. Child Development 61: 1230–1237
[24] Hepper PG (1997) Memory in utero? Developmental Medicine and Child Neurology 39: 343–346
[25] Levi-Montalcini R (1998) L'asso nella manica a brandelli. Baldini & Castoldi, Milano
[26] Edelman G (1987) Neural Darwinism: the theory of neuronal group selection. Oxford University Press, Oxford, pp 183–203
[27] Edelman G (1991) Il presente ricordato. Rizzoli, Milano (The Remembered Present – A Biological Theory of Consciousness. Basic Books, New York 1989)
[28] Selkoe DJ (1992) L'invecchiamento cerebrale. Le Scienze, Milano (In: Rita Levi-Montalcini L'asso nella manica a brandelli. Baldini & Castoldi, Milano, 1998, p 54)
[29] Austin JH (1998) Zen and the brain: toward an understanding of meditation and consciousness. The MIT Press, Cambridge (Mass.) London, pp 225–227
[30] Van de Carr FR (1998) The Factors in Prenatal and Perinatal Sensory Input. Int J Prenatal and Perinatal Psychology and Medicine 10(4) 477–482
[31] Van de Carr FR (1998) Educating Parents to Educate Their Children. Int J Prenatal and Perinatal Psychology and Medicine 10(3): 313–322
[32] Blum T (1998) Human Proto-Development: Very Early Auditory Stimulation. Int J Prenatal and Perinatal Psychology and Medicine 10(4): 457–476
[33] Blum T (1991) Early Prenatal Perception and Adequate Uditive Stimulation. Int J Prenatal and Perinatal Psychology and Medicine 3(3/4): 283–296

[34] de Mause L (1995) Childhood and History. (Chap. 3 Restaging Early Traumas in War and Social Violence, www.psychohistory.com/htm/09_trauma.html)
[35] Van Os J, Selten JP (1998) Prenatal exposure to maternal stress and subsequent schizophrenia – The May 1940 Invasion of the Netherlands. British J Psychiatry 172: 324–326
[36] Glover V (1997) Maternal Stress or Anxiety in Pregnancy and Emotional Development of the Child. British J Psychiatry 171: 185–186
[37] Insel TR et al. (1990) Prenatal Stress has long-term effects on brain opiate receptors. Brain Research 511: 93–97
[38] Gsell E et al. (1999) Prenatal Psychology, Analytical Psychology and Buddhism. Int J Perinatal and Prenatal Psychology and Medicine 11(2): 207–225
[39] Zuckerman M (1990) Still Another Failure of the Arousal Theory: A Critique of Personality, Situation and Physiological Arousability by G. Stemmler and E. Meinhard. Personality and Individual Differences 11(3): 309–312
[40] Goleman D (1997) La forza della meditazione. Rizzoli, Milano
[41] Ledoux J (1999) The Emotional Brain. Phoenix (Orion Books), London, pp 263–265
[42] Eppley KR et al. (1989) Differential Effects of Relaxation Techniques on Trait-Anxiety: a Meta-Analysis. J Clinical Psychology 45(6): 957–974
[43] Orme-Johnson DW (1973) Autonomic Stability and Trascendental Meditation. Psychosomatic Medicine 35(4): 341–349
[44] Wallace RK, Benson H (1972) The Physiology of Meditation. Scientific American 226: 84–90
[45] Jevning R et al. (1992) The Physiology of Meditation: A Review. A Wakeful Hypometabolic Integrated Response. Neuroscience and Biobehavioral Reviews 16: 415–424
[46] Dilbeck MC, Orme-Johnson W (1987) Physiological Differences Between Trascendental Meditation and Rest. American Psychologist 42: 879–881
[47] Badawi K et al. (1984) Electrophysiologic Characteristics of Respiratory Suspension Periods Occuring During the Practice of the Trascendental Meditation Program. Psychosomatic Medicine 46(3): 267–276
[48] Wallace RK (1970) Physiological Effects of Trascendental Meditation. Science 167: 1751–1754
[49] Austin JH (1998) Zen and the brain: toward an understanding of meditation and consciousness. The MIT Press, Cambridge (Mass.) London, pp 57–99
[50] Zhang J et al. (1986) Respiratory modulation of neuronal discharge in the central nucleus of the amygdala during sleep and waking states. Experimental Neurology 91: 193–207
[51] Frysinger R, Harper R (1989) Cardiac and respiratory correlations with unit discharge in human amygdala and hippocampus. Electroencephalography and Clinical Neurophysiology 72: 463–470
[52] Zhang J et al. (1986) Cryogenic blockade of the central nucleus of the amygdala attenuates aversively conditioned blood pressure and respiratory responses. Brain Research 386: 136–145
[53] Orem J, Netick A (1986) Behavioral control of breathing in the cat. Brain Research 366: 238–253
[54] Bellantuono C, Tansella M (1994) Gli psicofarmaci nella pratica terapeutica. Il Pensiero Scientifico Editore, Roma
[55] Briley M, Moret C (1993) Neurobiological mechanisms involved in antidepressant therapies. Clinical Neuropharmacology 16(5): 387–400