

# How Cognitive Neuroscience interacts with Psychiatric Forensic Examination: Conceptual Clarification and Methodological Assessment

*Luca Casartelli*



PhD Candidate at the University of Geneva, Switzerland, fellow at the Institute for Biomedical Ethics (Geneva University Medical School)

**N**euroethics deals with the ethical, political, juridical and social implications of neurophysiologic experimental data and their clinical applications<sup>1</sup>. Relevant neurophysiologic studies focus on cerebral anatomical lesions, functional abnormalities, neuro-biochemical imbalances, behavioural genetics, and affective or emotional disorders. Specifically, the role of cerebral studies in psychiatric forensic examination is currently under debate<sup>2</sup>. Western Penal Codes admit that when a subject is unable to control his conative, cognitive, affective and physical capacities, insanity may be pleaded<sup>3</sup>. The main argument in favour of the use of experimental data to support the classical psychiatric forensic approach is based on the fact that *some* neuroscientific findings can shed light on the defendant's mental capacity. However, we propose that not all experimental data should be used in legal issues without a clarification of their probative value in the forensic theatre.

## *The Three Paradigms: a Conceptual Distinction*

In Neuroethics, the relationship between neuroscience and law is often defiled by a conceptual misunderstanding. Indeed, there is a misleading overlap between the philosophical concept of free will, the juridical notion of liability, and the neuroscientific evidence concerning the subject's auto-determination capability. The same (ab)use of the three paradigms just mentioned is often present in folk psychology's idea of human responsibility. To disentangle the conceptual confusion, we will briefly describe the three paradigms.

1. The Philosophical debate on free will classically distinguishes *determinist* and *indeterminist* positions about the universe. The determinist position supposes that the state of the universe is essentially a function of physical laws and conditions present at the beginning of the universe, while an indeterminist position denies it<sup>4</sup>. A determinist supporting the existence of free will in a deterministic world is called a "*compatibilist*" or a "*soft determinist*", but if he denies the existence of free will, then he is called a "*hard determinist*". An indeterminist who agrees with free will existence is named a "*libertarian*". Many interesting arguments have been employed to endorse such philosophical alternatives<sup>5</sup>.

2. The juridical notion of liability is defined in many ways in Western Penal Codes, but it essentially focuses on two aspects: the subject's cognitive capacities and the subject's volitional capability<sup>6</sup>. It is widely accepted that the admissibility of a scientific proof that establishes a defendant's charged mental insanity cannot be decided without the implementation of rigorous and shared criteria, even if the same scientific evidence may be extremely probative for one purpose and completely not pertinent for another<sup>7</sup>. To exemplify, in order to judge the proof of admissibility in the US Code, two "Federal Rules of Evidence" are usually quoted: FRE 401: "Relevant evidence" means evidence having any tendency to make the existence of any fact that is of consequence to the determination of the action more probable or less probable than it would be without the evidence.

FRE 403: Although relevant, evidence may be excluded if its probative value is substantially outweighed by the danger of unfair prejudice, confusion of the issues, or misleading the jury, or by considerations of undue delay, waste of time, or needless presentation of cumulative evidence<sup>8</sup>.

Considering the example of brain imaging, American Courts have to confront with three main questions<sup>9</sup>: can we use neuroimaging as a probative tool in the attribution of criminal responsibility? Are brain images prejudicial rather than misleading or confusing to the jury and Courts? Can the use of neuroimaging be more dangerous than probative? No definitive conclusion can be drawn about this issue at present, and the current approach is, therefore, to evaluate each case separately.

### 3. Neuroscientific evidence concerning the subject's auto-determination

Neuroscientific evidence concerning the subject's auto-determination capability contains the implicit proposal of a specific theory of action, based on the subject's awareness of being himself the author of the action<sup>10</sup>. Following the classical work by Libet and colleagues<sup>11</sup>, many studies have investigated the activation of specific motor areas in the brain and sometimes criticized the methodological underpinning of Libet's approach<sup>12</sup>. Early studies underlined the role of *readiness potentials* generated in the supplementary motor area (SMA)<sup>13</sup>, while more recent research has compared the neurological correlates of voluntary action with the supposed instant when the intention to make a spontaneous movement arises to the subject's consciousness<sup>14</sup>. Two interesting cortical networks have been found which show that voluntary action follows essentially a parietal motor circuit (early sensory cortices-S1, parietal cortex, lateral part of the premotor cortex, primary motor Cortex-M1), then a circuit where M1 receives a broad class of inputs from pre-supplementary motor area (pre-

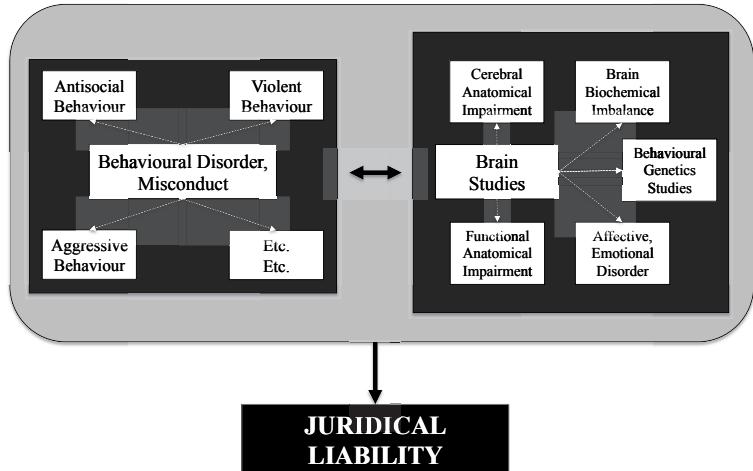
SMA), SMA, which in turn receives inputs from the basal ganglia and the prefrontal cortex<sup>15</sup>. We suggest that, although the neuroscientific data from Libet and colleagues are unable by themselves to solve the question concerning the philosophical concept of free will and its existence or contribute to the notion of juridical liability, they can still be conveniently used to sustain an argument in favour (or against, depending on the underpinning theory) free will existence. Manifestly, the shallow interconnection among the three heterogeneous paradigms such as the philosophical concept of free will, the juridical notion of liability and the action awareness based on neuroscientific data, turn out to be dangerously insufficient when we try to relate neuroscience and law. Therefore, we prefer to propose a clear distinction among these paradigms, while enclosing

a multivariate approach that makes use of the three paradigms synergistically to shed light on the neuroethical relationship concerning "neuroscience and law".

### The explanatory role of neuroscience in Courts: a new perspective

Another, more recent perspective supporting the explanatory role of neuroscience in the psychiatric forensic examination can also be delineated. Recent work seems to indicate a more convincing correlation between the results of brain studies and behavioural disorders. To make a profitable use of these experimental data in legal issues, we have first to scientifically clarify the correlation among misconduct (for example: aggressive, impulsive or violent behaviour) and brain anatomical lesions, functional abnormalities, neurobiochemical imbalances, genetic susceptibility or affective disorders. Following this initial work, we should try and establish a relation with juridical liability:

## *A recent perspective seems to indicate a more convincing correlation between the results of brain studies and behavioural disorders*



Based on our present experience and investigation, we believe that neuroscientific studies do not change the core of the notion of juridical liability. However, these studies can still be useful to obtain a more profound empirical comprehension and clarification on the real mental capabilities of the charged. Neuroscientific studies do not nullify the concept of juridical liability but can really *help* and *scientifically sustain* a judgment on the mental capacity of the charged. Clearly, many distinctions and assessments have to be considered. To clarify this hypothesis and for the sake of argument we propose four views on the same topic.

*An Experimental Approach: four different views*  
We consider four views to support our research hypothesis.

#### 1) Traumatic Brain Injury (TBI)

TBI is considered the typical situation that leads to brain anatomical deficits. Starting from the renowned case of Phineas Gage<sup>16</sup>, many recent studies focus on the behavioural consequences of TBI<sup>17</sup>. In fact, a growing body of evidence shows the existence of a correlation between TBI and executive dysfunctions: a study by Colantonio and colleagues regarding TBI in a forensic psychiatry population has shown that a history of TBI was present in 23% of 394 eligible patients' records<sup>18</sup>. Briefly, alcohol and/or substance abuse disorders and antisocial personality disorders/psychopathy are more frequent in charged subjects with a history of TBI. Obviously, this data underline the existence of a correlation but does not definitely depict the causal direction; in brief,

they cannot categorically determine when TBI is the consequence of alcohol and/or substance abuse disorders or vice-versa. To conclude: even if further studies are needed, brain anatomical deficits, assessed by neuroimaging techniques such as Magnetic Resonance Imaging (MRI) or Computed Tomography (CT), are surely an important element to be seriously considered when to plead mental states of the charged, also considering the high proportion of prison inmates found to suffer of the consequences of TBI<sup>19</sup>.

#### 2) Brain Functional Abnormalities: Aggressive, Impulsive and Violent Behaviour

Based on the cognitive neuroscience literature and a revisited form of the Raine's Model<sup>20</sup> stressing the role of environmental factors, we can try and correlate functional brain abnormalities with behavioural phenotypes. A recent study focusing on aggressive and impulsive behaviour describes an association between the activation of specific areas of the prefrontal cortex (dorsolateral, dorsomedial, ventromedial, orbital medial, ventrolateral) and the dorsal anterior cingulate cortex with the alteration of emotion regulation and impulse control<sup>21</sup>. Phenomenological experience of aggressive impulses has been associated with activity of hypothalamic structures, the amygdala and insula; decision making and socio-emotional information processing are related to the activity of the right anterior cingulate cortex, ventromedial and orbital medial prefrontal cortex, and anterior insula cortex<sup>22</sup>. Other studies inquired functional correlations of human aggressiveness and impulsiveness<sup>23</sup>.

The underlying concept is that a functional cerebral abnormality can be correlated to misconduct and behavioural disorders, such as aggressive, antisocial, impulsive and violent behaviour. It is important to remember that “correlation” does not necessarily mean “deterministic causation”. Thus, we are not able to definitely assess if functional cerebral deficits are causative or, differently, the consequence of misconduct<sup>24</sup>. In summary: brain functional abnormalities described in the literature may be useful to enlighten the neural underpinnings of phenomenological experience of the charged, for example explaining *how* the defendant “shapes” his aggressive behaviour or *how* the subject manifests an impulse control deficit. Therefore, we claim that the use of these data in Courts has to be carefully considered since their probative value has not been sufficiently documented or proven.

### *3) Behavioural Genetics: the MAOA Case*

Even if many authors have referred to the possibility of using behavioural genetics studies in legal cases<sup>25</sup>, very few of such cases concerning the use of genetics in Courts<sup>26</sup> have been reported. Furthermore, these cases are extremely controversial. A classical one in the Neuroethics literature is the MAOA gene case in Caspi's and colleagues' work<sup>27</sup>. Briefly, the classical model claims that the MAOA gene encodes for the MAOA enzyme; the MAOA enzyme degrades neurotransmitters like norepinephrine, dopamine, and serotonin. High levels of the MAOA enzyme are associated with a low activity of the neurotransmitters. Conversely, low levels of the enzyme are correlated with a high activity of the neurotransmitters, that in turn is associated with antisocial and violent behaviour when specific environmental factors are co-present (e.g.: childhood maltreatment). Consequently, environmental factors correlated to the MAOA enzyme activity have been considered predictive of violent and antisocial behaviour<sup>28</sup>. Even if this study has been widely cited, these data have not been always replicated, and therefore they appear rather controversial. Furthermore, conceptual and methodological

biases undermine evidence reliability<sup>29</sup>. In conclusion, considering that the probative value of behavioural genetics studies for legal issues appears ambiguous, we claim that at present there is no sufficient or trustworthy evidence to support the use of these studies in psychiatric forensic evaluation.

### *4) Affective and Emotional Disorder: Bilateral Amygdala Damage*

The Italian Penal Code apparently does not allow that an emotional or affective state be used to plead insanity<sup>30</sup>; this means, for example, that even if I fly into a temper, I am not excused if I kill or batter my neighbour; obviously, the Code *ratio* tries to prevent every attempt from pleading insanity for daily quarrel or honour killing. Nevertheless, current neuropsychiatry and neuropsychological research present a certain number of evidences supporting the idea that emotional or affective disorders underpin specific psychiatric conditions<sup>31</sup>. Recently, an interesting case concerning bilateral amygdala damage, that induces modifications in the phenomenological experience of fear, has been reported<sup>32</sup>. Bilateral amygdala damage is extremely rare, even if we can find some studies in the literature<sup>33</sup>. Without denying the risk of the inappropriate use or frank abuse of emotional and affective disorders to plead insanity, we argue that neuropsychiatric and neuropsychological research may still help to define some pathological emotional and affective disorder, potentially meaningful at least in some legal issues, and should therefore be pursued.

### *Conclusive Considerations*

In conclusion, we sustain that neuroscientific research – specifically in neuropsychiatric and neuropsychological areas – may be relevant in legal issues, especially in helping to define the mental states of the charged. Clearly, we cannot always assume that the neurological and neuroscientific arguments are sufficient to plead insanity. We have to consider, from time to time, the probative value of different data, without improperly overlap

heterogeneous conceptual paradigms. Furthermore, we support a multivariate approach including both the classical psychiatric model and the neuroscientific evidence in the psychiatric forensic evaluation. Considering the implications of psychiatric forensic evaluation, we defend a prudential attitude, although prudence must not become blindness towards new neuroscientific possibilities. To appropriately use neuroscientific evidences we need a neurocognitive model capable to explain and categorize empirical data better. Further empirical, methodological and conceptual studies may provide more consistent agreement and open new ways to consider the relation between neuroscience and law<sup>34</sup>.

#### NOTES

- <sup>1</sup> B. BAERTSCHI, *La neuroéthique: ce que les neurosciences font à nos conceptions morales*, La Découverte, Paris 2009; W. GLANNON, «Diminishing and Enhancing Free Will», in *AJOB Neuroscience*, 2(3) (2011), 15–26; E. RACINE, *Pragmatic Neuroethics: Improving Treatment and Understanding of the Mind Brain*, MIT-Press, Cambridge-MA 2010; A. ROSKIES, «Neuroethics for the New Millennium», in *Neuron*, 35 (2002), 21–23; A. ROSKIES, «Neuroimaging and Inferential Distance», in *Neuroethics*, 1 (2008), 19–30.
- <sup>2</sup> T. BROWN et E. MURPHY, «Through a Scanner Darkly: Functional Neuroimaging as Evidence of a Criminal Defendant's Past Mental States», in *Stanford Law Review*, 62/4 (2010); H. T. GREELY, «Law and the Revolution in Neuroscience: An Early Look at the Field», in *Akron L. Rev.*, 42 (2009), 687–715.
- <sup>3</sup> W. GLANNON, «Diminishing and Enhancing Free Will», op. cit.
- <sup>4</sup> A. ROSKIES, «Neuroscientific Challenges to Free Will and Responsibility», in *Trends in Cognitive Sciences*, 10/9 (2006).
- <sup>5</sup> W. GLANNON, «Neuroscience, Free Will and Responsibility», in *Journal of Ethics in Mental Health*, 4/2 (2009), 1–6; E. NAHMIAS et al., «Is Incompatibilism Intuitive?», in *Philosophy and Phenomenological Research*, 73/1 (2006); E. NAHMIAS, «Scientific Challenges to Free Will», in T. O'CONNOR et C. SANDIS (eds.), *A Companion to the Philosophy of Action*, Wiley-Blackwell, Oxford-UK 2010.
- <sup>6</sup> S.J. MORSE, «Genetics and Criminal Responsibility», in *Trends in Cognitive Sciences*, 15/9 (2011), 378–380.
- <sup>7</sup> T. BROWN et E. MURPHY, «Through a Scanner Darkly: Functional Neuroimaging as Evidence of a Criminal Defendant's Past Mental States», op. cit.
- <sup>8</sup> FEDERAL RULES OF EVIDENCE, USA 2011, <http://federalevidence.com/rules-of-evidence> (Date of access: 31/08/2011).
- <sup>9</sup> W. SINNOTT-ARMSTRONG et al., «Brain Images as Legal Evidence», in *Episteme*, 5 (2008), 359–373.
- <sup>10</sup> B. LIBET et al., «Time of Conscious Intention to Act in Relation to Onset of Cerebral Activity (Readiness-Potential): the Uncounscious Initiation of a Freely Voluntary Act», in *Brain*, 106 (1985), 623–642; see also ID., «Consciousness, Free Action, and the Brain», in *Journal of Consciousness Studies*, 8 (2001), 59–65; ID., *Mind Time: The Temporal Factor in Consciousness*, Harvard University Press, Cambridge-MA 2004; ID., «Do We Have Free Will?», in W. SINNOTT-ARMSTRONG and L. NADEL (eds.), *Conscious Will and Responsibility: a Tribute to Benjamin Libet*, Oxford University Press, New York 2010, Chapter I.
- <sup>11</sup> B. LIBET et al., «Time of Conscious Intention to Act in Relation to Onset of Cerebral Activity (Readiness-Potential): the Uncounscious Initiation of a Freely Voluntary Act», op. cit., 623–642.
- <sup>12</sup> H. C. LAU et al., «On Measuring the Perceived Onsets of Spontaneous Actions», in *Journal of Neuroscience*, 26 (2006), 7265–7271; H.C. LAU et al., «Manipulating the Experienced Onset of Intention after Action Execution», in *Journal of Cognitive Neuroscience*, 19 (2007), 81–90; W. SINNOTT-ARMSTRONG and L. NADEL (eds.), *Conscious Will and Responsibility: a Tribute to Benjamin Libet*, op. cit.
- <sup>13</sup> B. LIBET et al., «Time of Conscious Intention to Act in Relation to Onset of Cerebral Activity (Readiness-Potential): the Uncounscious Initiation of a Freely Voluntary Act», op. cit., 623–642.
- <sup>14</sup> P. HAGGARD, «Conscious Intention and motor Cognition», in *Trends in Cognitive Sciences*, 9 (2005), 290–295; ID., «Human Volition: Towards a Neuroscience of Wills», in *Nature Review Neuroscience*, 9/12 (2008), 934–46; B. LIBET et al., «Preparation- or Intention-to-Act, in Relation to Pre-Event Potentials Recorded at the Vertex», in *Electroencephalography Clinical Neurophysiology*, 56/4 (1983), 367–72; C. S. SOON et al., «Unconscious Determinants of Free Decisions in the Human Brain», in *Nature Neuroscience*, 11/5 (2008), 543–545.
- <sup>15</sup> P. HAGGARD, «Human Volition: Towards a Neuroscience of Will», op. cit., 934–46.
- <sup>16</sup> H. DAMASIO et al., «The Return Of Phineas Gage: Clues about the Brain from the Skull of a Famous Patient», in *Science*, 264/5162 (1994), 1102–1105; J. GRAFMAN et al., «Frontal Lobe Injuries, Violence and Aggression: a Report of the Vietnam Head Injury Study», in *Neurology*, 46 (1996), 1231–1238; J. VOLVAKA et al., «Neurological, Neuropsychological, and Electrophysiological Correlates of Violent Behavior», in *Neurobiology of Violence*, American Psychiatric Press, Washington, DC (1995), 77–122.
- <sup>17</sup> S.W. ANDERSON et al., «Impairments of Emotion and Real-World Complex Behaviour Following Childhood or Adult-Onset Damage to Ventromedial Prefrontal Cortex», in *Journal of International Neuropsychology Society*, 12 (2006), 224–235; D. MOBBS et al., «Law, Responsibility and the Brain», in *PLoS Biology*,

- 5/4 (2007), 693–700; L. ROCHAT et al., «Executive Disorders and Perceived Socio-Emotional Changes after Traumatic Brain Injury», in *Journal of Neuropsychology*, 3/2 (2009), 213–27; L. ROCHAT et al., «Assessment of Impulsivity after Moderate to Severe Traumatic Brain Injury», in *Neuropsychological Rehabilitation*, 20/5 (2010), 778–797; L. ROCHAT et al., «How Impulsivity Relates to Compulsive Buying and the Burden Perceived by Caregivers after Moderate-to-Severe Traumatic Brain Injury», in *Psychopathology*, 44/3 (2011), 158–164.
- <sup>18</sup> A. COLANTONIO et al., «Brain Injury in a Forensic Psychiatry Population», in *Brain Injury*, 21 (2007), 1353–1360.
- <sup>19</sup> PW. SCHOFIELD et al., «Traumatic Brain Injury among Australian Prisoners: Rates, Recurrence and Sequelae», *Brain Injury*, 20/5 (2006), 499–506; B. SLAUGHTER et al., «Traumatic Brain Injury in a County Jail Population: Prevalence, Neuropsychological Functioning and Psychiatric Disorders», *Brain Injury*, 17/9 (2003), 731–41.
- <sup>20</sup> A. RAINÉ, «From Genes to Brain to Antisocial Behavior», in *Current Directions in Psychological Science*, 17/5 (2008), 323–328.
- <sup>21</sup> E. F. COCCARO et al., «Corticolimbic Function in Impulsive Aggressive Behaviour», in *Biological Psychiatry*, 69/12 (2011), 1153–1159.
- <sup>22</sup> *Ibidem*.
- <sup>23</sup> P. PIETRINI et V. BAMBINI, «Homo Ferox: The Contribution of Functional Brain Studies to Understanding the Neural Bases of Aggressive and Criminal Behavior», in *International Journal of Law and Psychiatry*, 32 (2009), 259–265; A. RAINÉ et Y. YOUNG, «Neural Foundations to Moral Reasoning and Antisocial Behavior», in *Scan*, 1 (2006), 203–213; A. SIEGEL et J. VICTOROFF, «Understanding Human Aggression: New Insights from Neuroscience», in *International Journal of Law and Psychiatry*, 32 (2009), 209–215; J. L. SIEVER, «Neurobiology of Aggression and Violence», in *American Journal of Psychiatry*, 165/4 (2008), 429–442.
- <sup>24</sup> D. MOBBS et al., «Law, Responsibility and the Brain», op. cit., 693–700; A. ROSKIES, «Are Neuroimages like Photographs of the Brain?», in *Philosophy of Science*, 74 (2007), 860–872; Id., «Neuroimaging and Inferential Distance», op. cit., 19–30; W. SINNOT-ARMSTRONG et al., «Brain Images as Legal Evidence», op. cit., 359–373.
- <sup>25</sup> D. MOBBS et al., «Law, Responsibility and the Brain», op. cit., 693–700; S.J. MORSE, «The Non-Problem of Free Will in Forensic Psychiatry and Psychology», in *Behavioral Sciences & the Law*, 25 (2007), 203–220; Id., «Genetics and Criminal Responsibility», op. cit., 378–380; O.D. JONES, «Behavioral Genetics and Criminal Law», in *Journal of Law Contemporary Problems*, 69 (2006), 81–100.
- <sup>26</sup> M. LEVITT et N. MANSON, «My Genes Made Me Do It? The Implications of Behavioural Genetics for Responsibility and Blame», in *Health Care Analysis*, 15 (2007), 33–40; S.J. MORSE, «Genetics and Criminal Responsibility», op. cit., 15/9 (2011), 378–380.
- <sup>27</sup> A. CASPI et al., «Role of Genotype in the Cycle of Violence in Maltreated Children», in *Science*, 297 (2002), 851–854.
- <sup>28</sup> *Ibidem*.
- <sup>29</sup> D. FOLEY et al., «Childhood Adversity, Monoamine Oxidase A Genotype, and Risk for Conduct Disorder», in *Arch Gen Psychiatry*, 61 (2004), 738–744; B. HABERSTICK et al., «Monoamine Oxidase A (MAOA) and Antisocial Behaviors in the Presence of Childhood and Adolescent Maltreatment», in *Am J Med Genet B Neuropsychiatr Genet*, 135b (2005), 59–64; K. NILSSON et al., «Role of Monoamine Oxidase A Genotype and Psychosocial Factors in Male Adolescent Criminal Activity», in *Biol Psychiatry*, 59 (2005), 121–127; S. YOUNG et al., «Interaction between MAO-A Genotype and Maltreatment in Risk for Conduct Disorder: Failure to Confirm in Adolescent Patients», in *Am J Psychiatry*, 163 (2005), 1019–1025; M. RETI et al., «Monoamine Oxidase A Regulates Antisocial Personality in Whites with no History of Physical Abuse», in *Comprehensive Psychiatry*, 52 (2011), 188–194.
- <sup>30</sup> Art. 90, *Cod. Pen. It.*
- <sup>31</sup> T. BAYNE et E. PACHERIE, «Bottom-Up or Top-Down? Campbell's Rationalist Account of Monotheistic Delusions, in Philosophy», in *Psychiatry, & Psychology*, 11/1 (2004), 1–11; T. BAYNE et E. PACHERIE, «Experience, Belief, and the Interpretive Fold», in *Philosophy, Psychiatry, & Psychology*, 11/1 (2004), 81–86; T. BAYNE et E. PACHERIE, «In Defence of the Doxastic Conception of Delusions», in *Mind & Language*, 20/2 (2005), 163–188.
- <sup>32</sup> J. FEINSTEIN et al., «The Human Amygdala and the Induction and Experience of Fear», in *Current Biology*, 21 (2011), 34–38.
- <sup>33</sup> S.K. SCOTT et al., «Impaired Auditory Recognition of Fear and Anger Following Bilateral Amygdala Lesions», in *Nature*, 385 (1997), 254–257; D. TRANEL et al., «Altered Experience of Emotion Following Bilateral Amygdala Damage», in *Cognitive Neuropsychiatry*, 11/3 (2006), 219–232.
- <sup>34</sup> Acknowledgments. This work was supported by a grant from the *Fondation pour des Bourses d'Etudes Italo-Suisse, Lausanne*. I am in debt to Adriana Gini and Andrea Coppadoro for their insightful comments on an earlier version of this paper. The content of this work has been presented at the “Neuroscience and Law” workshop organised by the Neurobioethics Study and Research Group of the Pontifical Athenaeum *Regina Apostolorum*, Rome, September 23<sup>rd</sup>, 2011.