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EDITORIAL

Does being a genius require a bigger and better brain that the rest of us; is artistic creativity the product of elite cerebration; or are both born not of superiority but, rather, degeneracy and abnormality? These were issues that much preoccupied scientists and eugenicists of the late 19th century; and they remain with us today even if cloaked in more restrained and politically correct language. In *L'uomo di genio in rapporto alla psichiatria* (1888: English translation, *The Man of Genius,* 1891), Cesare Lombroso (1835–1909), professor of legal medicine at the University of Turin, argues that genius is a form of degeneration:

> 'just as giants pay a heavy ransom for their stature in sterility and relative muscular and mental weakness, so the giants of thought expiate their intellectual force in degeneration and psychoses. It is thus that the signs of degeneration are found more frequently in men of genius than even in the insane . . . [the problem is that] moral insanity and epilepsy . . . so often found in association with genius are among the forms of mental alienation which are most difficult to verify . . . although quite evident to the alienist.'

Professor Lombroso supports his thesis with a catalogue of (mainly unsupported) references to the physiognomy, behaviour and medical afflictions of men (and a few women) each of whom conjured acts of genius in the arts and sciences. The neurosis of genius is often precocious, as indeed are men of genius in terms of early acquisition of intellectual prowess. Proverbially the 'man who has genius at five is mad at fifteen'. Lombroso rehearses the literature on the co-morbidity of extreme intelligence and madness from Plato's (423–347 BC) *Phaedo* to Maxime du Camp (1822–94) whose *Souvenirs littéraires* (1882–83) exposes the seeds of insanity in many contemporary French writers, notably Gustave Flaubert (1821–80). Moving on, Professor Lombroso aligns genius with physical and character traits: small stature, rickets, pallor (*pulchrum sublimum virorum florem*), emaciation, stammering, left-handedness, sterility, delayed physical development, misoneism ('rejecting the discoveries of others'), vagabondage, failed marriage and 'indifference to the attractions of women', and defects of the cranium and brain. Whilst latent in many potential men of genius, the epiphany may require release through an event that 'irritates the cerebral cortex'. We learn of individuals who were dull until genius blossomed after a blow to the head, and of others whose skulls were deformed. In general, the genius does have a bigger than average head: (Francesco) Petrarca (1304–74) at 1602 cc; (Aurelius Ambrosius) Saint Ambrose (*c* 340–397) with a 1792 cc cranium; (Alessandro Giuseppe Antonio Anastasio) Volta (1745–1827) coming in at 1860 cc, and the list topped by (Ivan Sergueïevitch) Tourgueniev (1818–83) sporting a cranial vault of 2012 cc. Furthermore, the brain within is often conspicuous in men of genius for variations of its cerebral convolutions, especially the third frontal, well illustrated by the brain of the mathematician (Johann Carl Friedrich) Gauss (1777–1855). Genius involves impulsivity, often acting in defiance of reason; *eureka* moments that may occur during somnambulism or unconscious inspiration; and the 'shock of a sudden unexpected sensation' with regional cerebral blood flow abruptly augmented at the expense of the general circulation. But after moments of creativity the man of genius may descend into the troughs of despair and inferiority. Is it mildy amusing that (John) Abernethy (1764–1831) was consulted by a patient with melancholy and advised him to go and hear the actor and comedian (Joseph) Grimaldi (1778–1837) in order to cure his ailment through laughter rather than medicines? 'But I *am* Grimaldi' came back the reply. The man of genius has moments of supreme brilliance but may lapse periodically into stupidity: Lombroso quotes Victor Hugo (1802–85): 'she did not know Latin but understood it very well (*Les misérables*, 1862); 'the wealth of a nation depends on its prosperity [Louis-Napoléon Bonaparte (1808–73)]; or 'when one has crossed the bounds, there are no limits left [(François) Ponsard (1814–67)]. Their thoughts are monotypic being focused on one exclusive problem characterized by intuition that runs ahead of understanding, with a fondness for clever words and newly coined phrases; but, equally, blind alleys of incomprehension on other topics and absurd moments of absent-mindedness.

Lombroso proceeds by cataloguing the men of genius who suffered tics and other movement disorders, epilepsy, alcoholism and other addictions, melancholy leading to suicide, delusions of grandeur, and *folie du doute* with obsessional behaviour and overt insanity: 'when (Dr Samuel) Johnston (1709–84) walked the streets of London he was compelled to touch every post he passed; if he omitted one he had to return' . . . 'Napoleon could not pass through a street even when leading an army without counting and adding up the rows of windows'. Professor Lombroso moves on to discuss the influence of the weather and climatic conditions on genius. The most favourable season for aesthetic, astronomical, chemical, physical and mathematical creativity is springtime with a progressive decline during the summer and a burst of creative energy in the autumn before the nadir of winter. On the influence of race, sex and heredity on genius, Professor Lomroso highlights the disproportionate achievements of Jewish people, the paucity of female genius, the advantage of being

the child of older parents, and the dynasties in which genius passes down the family line—concluding that the natural history of such pedigrees is for genius to co-exist with epilepsy and criminality 'till the line ends in idiocy and sterility'.

After noting the now familiar link between genius and insanity in painters and musicians, and the evidence for monomania in their works, Lombroso concludes that the pathology of art lies in an exaggerated predilection for symbols, attention to minuteness of detail, excessive prominence of one colour, the choice of licentious subjects, and an exaggerated degree of originality. In literature, the trait is characterized by punning and plays-upon-words (see *Brain* 2013; 146: 685–7). In science, the man of genius rushes forward regardless of danger, faces with eagerness the greatest difficulties, seizes the strangest connections, grasps the newest and most salient points, and shows originality carried to the point of absurdity.

In short, the man of genius has a large head and precocious intellect but suffers from a psychosis that has several characteristics: no firmness of character ('he bends with the wind that blows'); self-consciousness that appreciates itself and has no monkish humility; a propensity for stimulants and alcohol; anomalies of the reproductive functions, that is, sexual ambiguities, excesses and perversions; affective insensibility or lack of moral sense; the need endlessly to travel; erratic behaviour in terms of career and course of study; dependence on moments of inspiration outside which the genius is shackled; preoccupation with religious doubts; concern with his (her) own *ego*; the tendency for delusions, illogical deductions, absurd contradictions and grotesque and inhuman fantasies in the works; importance attached to dreams; and extreme alternation between 'erethism and atony, inspiration and exhaustion'.

This exhaustive and exhausting analysis leads Lombroso to consider that the creative power of genius is a degeneration belonging to the family of epilepsy. 'Such conclusions may seem strange to persons unacquainted with the way in which the region of epilepsy has been extended in modern times, so that many cases of headache . . . or loss of memory . . . are now recognised as forms of epilepsy, though in disguise; their manifestation . . . causing the disappearance of every trace of the pre-existing epilepsy'. He goes on to explain that aberrant cerebral activity leads to active and violent unconcsiousness manifesting in the genius as creativity and in the epileptic as 'motory agitation'. The epileptoid nature of genius is not an accidental phenomenon but a true *morbus totius substantiae*. 'Between the physiology of the man of genius and the pathology of the insane there are points of coincidence; there is even actual continuity'. This axis is well articulated by (Fyodor Mikhailovich) Dostoyevsky (1821–81) in *Bésy* [1872: English translation as *The possessed* by Constance Garnett (1861–1946) in 1916); and *Idiot* [1868–9: English translation as *The idiot*, by F(red) Whishaw (1854–1934), 1887]. Lombroso concludes that

> 'the frequency of genius amongst lunatics and of mad-men among men of genius, explains the fact that the destiny of nations has often been in the hands of the insane; and shows how the latter have been able to contribute so much to the progress of mankind. Nature has intended to teach us respect for the supreme misfortunes of insanity; and also to preserve us from being dazzled by the brilliancy of those men of genius who might well be compared, not to the planets which keep their appointed orbits, but to falling stars, lost and dispersed over the crust of the earth.'

Albert Einstein (born 1879) died on 18 April 1955, aged 76 years from rupture of an aortic aneurysm. Thomas S(tolz) Harvey (1912–2007) carried out the autopsy on the day of Einstein's death, removed his brain (weighing 1230 g), photographed it, cut blocks for preparation of histological slides from each section, and subsequently distributed the material to various investigators. Most of Einstein's brain tissue is now lost. The National Museum of Health and Medicine (Silver Spring, Maryland, USA) has recently acquired material relating to Einstein from the estate of Dr Harvey. In this issue of *Brain*, Dean Falk, Frederick Lepore and Adrianne Noe describe the macroscopic appearances of the Einstein brain based on 14 previously unpublished photographs taken by Dr Harvey during the autopsy (see page 1304; and see cover). Dean Falk and his colleagues point out that although the evolution of human cognition is associated with a general increase in brain size, variation in the connections of different regions may force non-random expansion of selected parts, due to the 'tension-based theory of gyrus formation'. The ensuing variation and asymmetry in gyral and sulcal patterns and hemispheric architecture may also reflect advantages conferred on the individual in terms of cognitive performance. The gyral architecture of Einstein's cerebrum is compared with that of two collections of 'normal' brains. The 14 photographs are accompanied by line drawings with selected areas coloured on which are documented differences in the size and organization of individual gyri, as assigned by the authors. Einstein did not have an particularly big brain; nor is its apparent asymmetry with expansion of the right frontal and left occipital lobes especially unusual for a right-handed male. But the gyral patterns are distinct. Naturally the focus is on Einstein's frontal lobes, which show bilateral expansion of the mid-frontal association cortex and asymmetric anomalies and enlargements of other frontal and prefrontal gyri and sulcal patterns (especially those parts of the motor cortex representing the face, tongue and Broca's area). Taking a strictly localizationist stance, these features are seen as evidence in support of enhanced ability in free-thought experiments such as Einstein 'imagining himself riding alongside a light-beam or being enclosed in an elevator accelerating up through space'. Selective enlargements of the left inferior parietal lobule and the right superior parietal lobule are 'consistent with enhanced visuospatial and mathematical abilities'. The inferior temporal gyri are greatly over-developed. The cingulate gyri are relatively convoluted especially on the left, as are much of the medial surfaces of Einstein's cerebral cortices. In short, and in so far as gross anatomy can illuminate the matter, Einstein did have a better brain than those who still struggle conceptually with $E = MC^2$. The authors hope that their description of the regional peculiarities of Einstein's brain together with availability of the roadmap to the blocks that may still exist will allow the histological features of the areas that made him distinct, from a cerebral perspective, now to be better aligned.

Gustav Klimpt (1861–1918), Oscar Kokoschka (1886–1980) and Egon Schiele (1890–1918)—creative members of the early 20th century Viennese circle of artists—provide ammunition for Lombroso's concept of the pathology of art. In 'The fantastic

organ', Karl Friston reviews '*The age of insight: the quest to understand the unconscious in art and mind, and brain, from Vienna 1900 to the present*' by Eric Kandel (2012: see page 1328). By way of Hermann von Helmholtz (1821–94), we visit the louche world of old Vienna and the formulations of Eric Kandel on what art can teach of brain function; and via the bottom-up analyses of David Marr (1945–80) to Friston's over-arching view of the brain as the organ of prediction. Kandel uses 'innovative thinking and insights in science and art' and modern ideas on perception and aesthetics to caricature the brain as an inference machine that tests its fantasies and hypotheses against sensory reality. Depicted through art, the work of Klimpt, Kokoscka and Schiele expressed unconscious inference dependent on activity of mirror neurons; and, in entering the private theatre of another's mind in order to explore its nature, mood and emotion, these artists were early exponents of the modern concept of theory of mind. Science and art reveal the mutual connections between things, and distil causal structure through inferring these relationships. The way the brain works through its multiple circuits and connections, is to make predictions based on external and internal information received and align these through appropriate responses. Perception resolves the errors and ambiguities of plausible but competing sensory hypotheses, and matches predictions to sampling. Action is broadly similar and involves active inference: when prediction errors cannot be settled by altering them to match exteroceptive sampling, the situation is acted upon so that the exteroceptive, interoceptive and proprioceptive predictions are now optimally aligned. The concepts of prediction coding and viewing the brain as an inferential organ are Helmholszian in their top-down principles of organization. Professor Friston considers the 'Marrian' bottom-up assembly of primitive elements into a complete percept to be 'an ugly hiatus in what should have been a seamless progression of ideas . . . of the brain as an inference machine'. The reading of the sensory runes and acting upon them, and knowledge of what the brain itself is doing at any one time, is (in part) the work of mirror neurons. Karl Friston ends his review with a series of rhetorical statements that expose the unanswered questions raised by the concept of the brain as a system for assessing prediction errors and, through neuromodulation, responding to the challenges that process poses. Professor Friston is Wellcome Principal Research Fellow and Scientific Director of the Wellcome Trust Centre for Neuroimaging at the Institute of Neurology, Queen Square (London); the inventor of statistical parametric mapping, voxel-based morphometry and dynamic causal modelling, and much else in brain sciences besides; and a respected and diligent reviewer to whom this journal often turns for sage advice.

Impulsivity and drug dependence lie in the realm of impaired active inference and failed neuromodulation; Professor Lombroso would also have us believe that each contributes to the pathology of art and the stuff of genius. Amongst papers in the current issue, Joyce van der Vegt and investigators from Hvidovre (Denmark), Nijmegen (The Netherlands) and Lübeck, Kiel and Hamburg (Germany) study patients with recent onset Parkinson's disease in order to disambiguate abnormalities of reward processing and impulse control disorders related to loss of dopaminergic neurons from the effects of exposure to dopaminergic medication (page 1192); using games that provide monetary gain and loss through gambling in drug naive cases, they show much reduced functional MRI activity in meso-cortico-limbic dopaminergic regions indicating that these clinical features are intrinsic to the disease not its treatment. Xi Luo and a team from New Haven and Providence (USA) study cognitive function in cocaine addicts prospectively and show that females with reduced activation in the thalamus and dorsal anterior cingulate cortex during tasks that involve error processing relapse more easily (page 1231). Livia Curcio and colleagues from Rome, Varese and Milan (Italy) demonstrate that D(extro)-serine is a co-agonist for *N*-methyl-D-aspartate receptor potentiation and depression of synaptic transmission; this ligand is much reduced in the nucleus accumbens of cocaine-dependent rats during early abstinence and its synthetic and degrading enzymes are under- and over-expressed, respectively, implicating the molecular machinery of D-serine in the altered mechanisms of synaptic plasticity seen in cocaine addiction (page 1216).

Genetic analysis has allowed the phenotypic spectrum of progressive myoclonus epilepsy syndromes and knowledge on their underlying mechanisms to expand considerably. Amongst four papers on epilepsy, Elisabeth Stogmann and colleagues study a large consanguineous family with cortical myoclonic tremor and epilepsy, mapping the causative gene to 1q31.3-q32.2 from which exome sequencing leads to identification of a single base pair deletion resulting in frame-shift miscoding of exon 6 in *CNTN2*, implicating a defect of neuronal excitability through loss of its product, contactin 2, with consequential failure to maintain potassium channels at juxtaparanodal regions (page 1155); next, they sequence the coding regions in 189 Caucasian patients with epilepsy but find no further mutations outside the one pedigree, occasional heterozygotes for the c503_503delG deletion being clinically unaffected. Lysa Boissé Lomax and investigators from Heidelberg, Adelaide, Melbourne and Herston (Australia), Dianalund and Odense (Denmark), Amsterdam, Nijmegen and Rotterdam (The Netherlands), Oslo (Norway), Oldenburg (Germany) and Helsinki (Finland) describe 12 examples of early onset ataxia followed by myoclonic and other forms of seizure disorder, associated with spike and wave electroencephalographic abnormalities, scoliosis and pes cavus foot deformity leading to loss of mobility and death as young adults, in association with mutations of Golgi SNAP receptor complex 2 (*GOSR2*), arising from a founder effect traced to countries bordering on the North Sea and coastal region of northern Norway (page 1146). The relationship between tremor, myoclonus and generalized epilepsy was slow to emerge in the neurological literature. In From the Archives, we review 'Dyssenergia cerebellaris myoclonica – primary atrophy of the dentate system: a contribution to the pathology and symptomatology of the cerebellum' by J Ramsay Hunt (*Brain* 1921; 44: 490–538) and 'Progressive familial myoclonic epilepsy in three families: its clinical features and pathological basis' by D.G.F. Harriman and J.H.D. Millar with an appendix on the genetic aspects by A.C. Stevenson (*Brain* 1955; 78: 325–49)

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