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Traumatic shock and electroshock: the difficult relationship between anatomic pathology and psychiatry in the early 20th century

C. Patriarca[1], C.A. Clerici[2]

[1] Division of Pathology; Asst Lariana, Ospedale Sant'Anna, Como, Italy; [2] Department of Oncology and Haemato-oncology, Università degli Studi di Milano and SSD Psicologia Clinica, Fondazione IRCCS Istituto Nazionale dei Tumori, Milano, Italy

Summary

In the conviction that a look at the past can contribute to a better understanding of the present in the field of science too, we discuss here two aspects of the relationship between early 20th century anatomic pathology and psychiatry that have received very little attention, in Italy at least. There was much debate between these two disciplines throughout the 19th century, which began to lose momentum in the early years of the 20th, with the arrival on the scene of schizophrenia (a disease histologically sine materia) in all its epidemiological relevance.

The First World War also contributed to the separation between psychiatry and pathology, which unfolded in the fruitless attempts to identify a histopathological justification for the psychological trauma known as shell shock. This condition was defined at the time as a "strange disorder" with very spectacular symptoms (memory loss, trembling, hallucinations, blindness with no apparent organic cause, dysesthesias, myoclonus, bizarre postures, hemiplegia, and more), that may have found neuropathological grounds only some hundred years later.

Among the doctors with a passed involvement in the conflict, Ugo Cerletti, the inventor of electroshock treatment, focused on the problem of schizophrenia without abandoning his efforts to identify its organic factors: if inducing a controlled electric shock, just like an experimentally-induced epileptic seizure, seems to allay the psychotic symptoms and heal the patient, then what happens inside the brain? In seeking histological proof of the clinical effects of electroconvulsive therapy ("the destruction of the pathological synapses"), and attempting to isolate molecules (that he called acroagonins) he believed to be synthesized by neurons exposed to strong electric stimulation, Cerletti extended a hand towards anatomic pathology, and took the first steps towards a neurochemical perspective. However his dedication to finding a microscopic explanation for schizophrenia – in the name of a "somatist" approach that, some years earlier, the psychiatrist Enrico Morselli had labelled "histomania" – was unable to prevent psychiatry from moving further and further away from anatomic pathology.

Introduction

Schizophrenia (Eugen Bleuer 1911) was first described as *dementia precox* by Emil Kraepelin in 1883 [1]. The condition became an epidemiologically relevant issue in the first half of the 20th century, to such a degree that the popular American Harper's magazine defined the century as "the era of schizophrenia" [2].

"Open up a few corpses" is the title of one of the chapters in Michel Foucault's famous volume, "The Birth of the Clinic" [3]. The practice of dissection, with its slow accumulation of histopathological findings at the root of neuropsychiatric signs and symptoms, right from the discovery of the luetic nature of *progressive cerebral palsy* (clinically similar to madness *sine materia*), helped to mark the nosographical boundaries of neurology, and to exclude a long list of well-known organic diseases from the field of the psychoses. We have numerous reports from authors who examined the histological evidence of neoplasms and neurodegenerative diseases collected in the 1950s and 1960s on autopsies conducted on patients who had been considered as psychiatric cases (Prof. Felice Giangaspero, neuropathologist; personal communication). In an article published in Pathologica in 1911, Ugo Cerletti, the inventor of electroshock treatment, admitted that dementia precox suffered from the lack of accepted

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Correspondenza: Carlo Patriarca, Servizio di Anatomia Patologica, Ospedale Sant'Anna di Como, via Ravona, 22020 San Fermo della Battaglia (CO), Italy - E-mail: carlo.patriarca@asst-lariana.it

microscopical descriptions. Besides, he classified other mental diseases characterized by dementia with a well known histopathological basis in neurosyphilis, trypanosomiasis (sleeping sickness), senile dementia and atherosclerotic dementia [4]. After clearing the field of known conditions and diseases of other kinds, Emil Kraepelin had little faith in an anatomo-pathological classification of psychoses, despite postulating their organic basis [5 a]. Then, in the first decades of the 20th century, the huge issue of schizophrenia came to light, a nameless ghost for the pathologist. This brought the curtain down on the conviction that "mental diseases are diseases of the brain", as Wilhelm Griesinger (a clinician and neuropathologist very influential in the second half of the 19th century) had put it [6]. In the eyes of the psychiatrist, it also marked the end of that special status of pathology in medicine that Foucault had described as "the privileges accorded to pathological anatomy." [3].

The outbreak of the First World War brought psychiatrists face-to-face with hitherto unknown situations and mindscapes [7], once again without histopathologically based solutions. The effects of mental traumatisms in wartime (shell shock) and the reports on treatment with electroconvulsive therapy (so called faradism), let a track in the background of doctors involved in the conflict as Ugo Cerletti and others.

Shell shock: a late rapprochement that came too late

As an extreme experiment on how the human mind adapts to traumatic phenomena, the war provided a tragic opportunity to test opposing theories on the pathogenesis of the soldiers' psychiatric disorders. Once the insinuation that soldiers were largely simulating their symptoms had been rejected – with some difficulty, and never completely by the world of military medicine [8 9] – two different opinions emerged. According to some, war does not make people ill, it only brings out latent psychological impairments. This was the view taken by numerous physicians in countries all over Europe, and in Germany by Alois Alzheimer [10], who lived only into the first few months of the Great War (he died in 1915). This view was also supported by the majority of Italian psychiatrists, who had inherited Lombroso's ideas [5]. According to others, people unavoidably become ill in war, as the experimental psychologist Agostino Gemelli saw on the front line in 1917. He wrote of the impoverishment of the inner life of the soldiers (what he described as the "shrinking field of consciousness of the infantryman") [11], who were useful only as unthinking launchers of an assault [12]. It was in the British scientific publications of the time, in 1915, that shell shock first became a hot topic [13]. Then Freud's studies on traumatism in wartime [14], what he called traumatic neurosis as part of his drive theory, and other studies presented at a conference of psychiatrists amply dedicated to the psychological trauma of war in Budapest in 1918 [15], anticipated modern historiography [16 17] in consolidating this psychopathological interpretation of man in wartime, or in other words of war as a pathogenic agent.

The psychological disorders of the traumatized infantryman (3 to 5% in the British army) could produce all sorts of symptoms: asthenia; amnesia; headache; vertigo; insomnia; hallucinations; nervous tics; aphasias; stammering; deafness and blindness with no apparent organic cause; tachycardia; arrhythmias; trembling; myoclonus; spastic muscle contractions or their opposite, flaccid paralysis, even to the point of hemiplegia; lack of appetite; sphincter disorders; and cutaneous paresthesias, anesthesias and hyperesthesias [18]. By the end of the war, even many of the psychiatrists who had originally taken Lombroso's approach had come to admit that wartime trauma can cause a diencephalic-mesencephalic neurovegetative lesion, with effects on the cranial nerves and systemic repercussions, though they would hasten to say that this could only happen to predisposed individuals, who they described as "constitutionally cenesthopathic" [5].

Could we claim that this also paved the way to anatomo-pathological and in particular neuropathological investigations? So it seems, although autopsies were certainly not routine practice at the front [b]. In Italy, for

[a] In Italy, where a strong impression was left by Camillo Golgi, there was a robust histopathological tradition that opposed the clinical taxonomic approach to psychological disorders. But even in Germany psychiatry did not develop in a linear manner, and Kraepelin's strict clinical prognostic classification stood in opposition to the firmly organicist approach of Karl Wernicke [5].

[b] If autopsies had been conducted more often at army hospitals it might have been possible to discover the myelotoxic effects of mustard gas almost 30 years sooner. Its effects were accidentally acknowledged only during the Second World War: Allied bombing on the port of Bari in 1943 released mustard gas from the hold of a ship that was hit, and dissections conducted on the civilians who died a few days later revealed total bone marrow aplasia. These observations also led to the first controlled studies on chemotherapy for acute leukemia [20]. It is also thanks to such studies that we know about the neuropathological effects of punctate hemorrhages

instance, dissections were being conducted for teaching purposes at the army university in San Giorgio di Nogaro, near Palmanova, behind the front lines in the north-eastern Veneto region, until this extraordinary medical school experiment was interrupted by the crushing defeat suffered at the Battle of Caporetto [19].

In his book *L'Officina della Guerra* [9], Antonio Gibelli dedicates more than one chapter to the topic of the traumatized infantrymen [c] who "not even the most ferocious discipline succeeded in controlling", concluding that specialists on every front would be wondering for years about the pathogenesis of this "strange disease" without succeeding in finding an answer. It is a state of concussion that grips a soldier who feels a cannonball whizz by, that *vent du projectile* known ever since the time of the Napoleonic wars. But what could the pathologists see in the brain of the handful cases of dead soldiers they examined who had not been exposed to gas, physical injury or direct trauma, but who had the symptoms of shell shock?

Frederick Walker Mott, the pathologist who studied the problem more than any other at the time, spoke of congestion of the meningeal and intraparenchymal vessels, and initial signs of chromatolysis of the nuclei in the motor areas of the frontal gyri, pons, and medulla oblungata [22]. These findings are rather vague and scarcely convincing, bearing in mind the delay in the fixation of the brain tissues attributable to the unavoidable logistic limitations of autopsies conducted in wartime circumstances, and the different fixing agents used (Kaiserling solution, alcohol). There was also evidence of sparse, tiny hemorrhagic petechiae in the white matter of the centrum semiovale, corpus callosum, internal capsule and subarachnoid spaces, in the absence of any external signs of trauma [27 28]. The pathologist concluded that [22]: "undoubtedly the vast majority of non-fatal cases of shell shock are more emotional in origin than commotional, and *occur especially in subjects with an inborn neurotic or neuropathic temperament*". In another study, the same author hypothesized that fatal cases had involved damage to the extracellular matrix [29], "the delicate colloidal structures (…) arresting the function of the vital centers in the medulla". About the existence of predisposed individuals, he wrote that "the moral effect of the continuous anxious tension of what may happen [under artillery bombardment], which, combined with the terror caused by the horrible sights of death and destruction around, tends to exhaust and eventually even shatter the strongest nervous system" [28].

In short, we could say that – from a histopathological standpoint – the genesis of shell shock remains unknown [30]. The review conducted by Peter Leese, Hans Binneveld and Ben Shepard on a large number of articles and monographies about *shell shock* published in England between 1915 and 1920 confirmed that efforts to find etiological explanations of this condition came to a dead end [31].

Traumatic shock experienced in times of war was classified as a clinical disorder with the introduction in the DSM-III [APA 1980] [32] for diagnosis of post-traumatic stress disorder (PTSD). This condition is characterized by intense fear, reactualization of the traumatic episode, avoidance of stimuli associated with the trauma, and increased *arousal*. Modern research approaches have found evidence of neurological changes associated with some types of trauma (including wartime trauma, but also sexual abuse by family members). For instance, imaging methods documented changes in the volume of the right hippocampus (limbic system) in Vietnam war veterans [33], and other alterations in the brain [34]. These changes are similar in some ways to those identifiable in animals submitted to prolonged stress, which are accompanied by high cortisol levels.

Although there are still many aspects to clarify, the modern conception of PTSDs essentially focuses on the involvement of procedural memory (or implicit memory), while explicit recall may even be completely lacking. In other words, patients suffer from anomalous memorization processes that tend not to regress spontaneously. These memories may be fragmented and inaccessible, or only partially accessible, for conscious recall. The condition is therefore characterized by a distortion of the meaning of perceived reality and individual subjectivity due to the effects of tumultuous emotions, and by fragments of intrusive, painful memories that are difficult to manage [35].

It is only recently, moreover, that the first histopathological data have emerged to support an organic basis for the symptoms of traumatism [36], the so-called

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caused by blister gases (yperite) or other suffocating toxic gases used in war (mixtures of chlorine and phosgene on the Italian front line) [21] that pathologists learned to identify already during the First World War, and judged responsible for arteriolar thrombosis [22]. But the most significant increase in the amount of autoptic activity, on the Western front at least, only came in the final months of 1918, coinciding with the outbreak of the Spanish flu epidemic [23].

[c] «'I'm afraid of going crazy', he told me. 'I'm going to go crazy one of these days, or I'm going to kill myself. I've got to kill myself.' I didn't know what to say. I, too, could feel the ebb and flow of waves of madness. At times I could feel my brain sloshing around inside my skull, like water inside a shaken bottle.» (from *A Soldier on the Southern Front*, by E. Lussu) [25]. For a more complete picture of the phenomenon it is worth taking a look at the other side of the front too [24], and Ernst Junger's touching descriptions of the soldiers' condition [25]. For a review on the topic, see also ref. 8.

chronic-blast traumatic brain injury (TBI). Here again, only a limited number of cases have been analyzed, on the brains of soldiers returning from military campaigns in Iraq with PTSD (and suffering from headaches, anxiety, insomnia, memory loss, depression, epileptic seizures, and chronic pain) who subsequently died of other causes, including substance abuse or suicide. The common denominator of their TBI seems to be astroglial fibrosis, revealed by immunohistochemical staining for GFAP. This involved an increase in fibrosis at the interface between the white and grey matter, in tissue adjacent to the cerebrospinal fluid, around the penetrating arteries, around the basal nuclei and limbic system – in other words, at the interface between areas of different physical density invested by the gaseous wave of the explosion. The damage can explain the symptoms [36]: headache due to tissue disruption of pia and injury to penetrating vessels, with an altered circulation of the CSF; cognitive impairments caused by damage to the "U" fibers at the interface between the grey and white matter; and memory deficits and sleep disorders due to damage to the periventricular structures of the limbic system. It is interesting that the same types of lesion were found in the brains of soldiers and victims of acute-blast TBI too, supporting the hypothesis of an early onset of this fibrotic damage (which is not seen in controls exposed to trauma not caused by explosives, as in cases of chronic traumatism, or trauma caused by contact sports or road accidents). It could be said emphatically that, a hundred years on, neurophysiology and modern pathology provide us with a new hypothesis to explain shell shock, very different from the moralistic explanations (cowardly soldiers), Lombroso's theories (genetic shortcomings in some soldiers), or purely psychoanalytical interpretations [d] of the past.

Electroshock between psychiatry and pathology

In the early 20th century world of psychiatry, there were still those who were striving for a quick fix for certain psychiatric disorders, with the aid of hypnosis, for instance [37]. During the First World War, there were even more evident signs of this drive to find rapid and effective therapies that would enable soldiers to be promptly returned to the front line [38], relying on the institution of the so-called *psychiatrie de l'avant* (prompt intervention behind the front line), and the provision of intensive treatments in city hospitals. The records of the London National Hospital report on shell shock being treated with electroconvulsive therapy (called Faradism) (Fig. 1) combined with massage, baths, heat, exercise, and suggestion (hypnosis) [31].

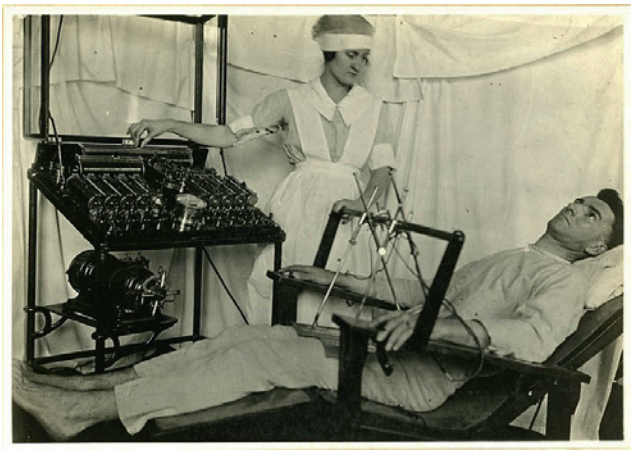

Fig. 1. Electric treatment for psychological symptoms, in psycho-neurotic cases. I World War era.

In actual fact, as the historian of medicine Giorgio Cosmacini reports (personal communication), already in the second half of the 19th century increasing use was being made in hospitals of electrotherapies that involved administering a shock or "sharp jerk" to patients with motor disorders and various other kinds of impairments [39].

It is against this background that electroconvulsive therapy (ECT) was invented by an eclectic clinician (Ugo Cerletti) who fought in the First World War and was consequently certainly able to observe the effects of the *vent du projectile* on the soldiers [40]. However, Cerletti's interest focused mainly on finding a treatment for the disease of the century, schizophrenia. At the time, it was common to treat this condition using physical means (hydrotherapy, light baths, sedatives), unless the clinician opted for a frontal lobotomy. Without arriving at such an extreme solution, severe cases were treated with insulin- and acetylcholine-induced shocks and, from 1936 onwards (with results that seemed very encouraging at the time), with the cardiazol-induced shock introduced by Lazlo von Meduna, a Hungarian scientist of international standing in close contact with Cerletti [5].

Cerletti (Fig. 2) trained in Germany as an anatomic pathologist, and held a strong belief in the concept of "somatism". For years, he studied epilepsy and its

[d] As Valeria Babini wrote [5], Freud introduced the topic of repetition compulsion, and consequently of the death drive, starting from a reflection on traumatic neurosis.

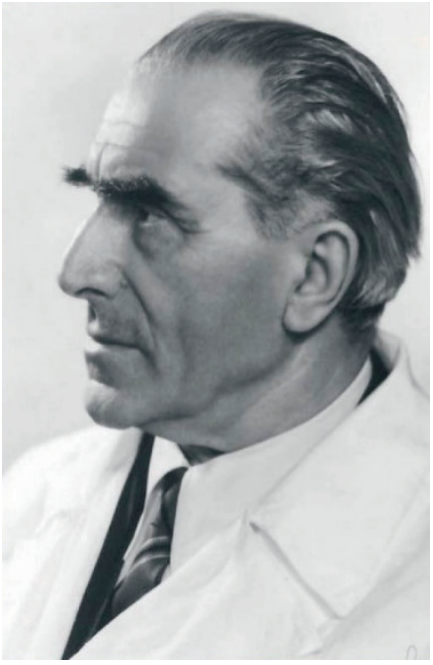

Fig. 2. Ugo Cerletti.

neuroanatomical grounds, accumulating a considerable amount of experience in research and on the wards (at the Mombello psychiatric hospital in Milan, at the Universities of Bari and Genova, and finally at the Sapienza University in Rome) [e]. His investigations began with histological studies on the brains of animals exposed to cardiazol-induced shock. Cardiazol causes an epileptic seizure, and epilepsy was a model of great interest to psychiatrists at the time. They saw a clinical, somatic (of athletic type in schizophrenia, leptosomic in epilepsy), and statistical incompatibility between epilepsy and schizophrenia. According to von Meduna, this incompatibility applied to the pathological sphere too: after analyzing histological preparations of brain tissue, he wrote about the contrast between the excessive growth of glia cells in epilepsy and "the apparent torpor of the glia system in the schizophrenic brains" [42]. By analogy, Cerletti studied the effect of electroshock on animals [43], finding it capable of producing a controlled or "fractionated" epileptic seizure of variable intensity, that the Italian scholar observed for the first time at the Testaccio slaughterhouse in Rome. He subsequently reproduced the phenomenon in animals of various species, from Komodo dragons to penguins, from porcupines to boa constrictors, which were made available by the zoo in Rome [41 44].

These experimental studies, also published in *Pathologica* in 1934 [45], continued after the introduction of ECT in clinical practice in 1938. Its clinical efficacy was so much greater than that of any other previously-attempted therapies that the diffusion of this treatment was immediate, global and destined to have a fundamental role in psychiatric treatments for more than 20 years [5]. Studies on the brains of treated animals were ambitiously aimed to discover the organic basis for mental disorders by starting from the effects of the therapy proving the most effective in humans [45]. They revealed the onset of "glial pyknosis, regressive vascular modifications, and pyknosis of the Purkinje cells", though Cerletti judged these last alterations to be partly due to artefacts. At a voltage sufficient to extinguish the most severe psychiatric symptoms, the findings in mammals, pigs, and dogs became more hazy and diffuse, and Cerletti wrote that "these changes seem reversible" [44]. This did not prevent him from hypothesizing (albeit without succeeding in documenting it clearly) the destruction of "pathological synapses" and damage to associative pathways implicated in the genesis of schizophrenia [44]. Cerletti believed that these pathways developed after the brain's ontogenesis, and were consequently more vulnerable to the insult caused by the electroshock.

Such conclusions may seem naive nowadays, in the light of modern concepts of neuroplasticity and our understanding of how the brain's structure and functions are constantly being remodeled. However it has to be said that, though he was working in a scientific world before the most important ultrastructural, biochemical, neuroendocrinological, pharmacological and genetic discoveries, Cerletti was already attenuating what he called "histological tautologies", right from his early studies. He became convinced that "the fundamental morbid core of the schizophrenic psyche" [43] lay deeper down, in the meso-diencephalic regions, and that it was strictly linked to phenomena of a biochemical, quantifiable and identifiable nature [44 f]. Cerletti never abandoned his search for the organic roots of schizophrenia, based on a "somatist" approach in

[e] For the complex cultural roots of Cerletti's medical training, see the very detailed essay by Roberta Passione [41]; and for an outline of Italian psychiatry of the time, see the volume Liberi Tutti by Valeria Babini [5].

[f] For an interesting review of neuropathological studies on schizophrenia, pooling both morphological and molecular data, see two reviews by P.J. Harrison et al. [46 47]. The same authors recommend caution in considering their interpretation, but certainly the claim [48] that "schizophrenia is the graveyard of neuropathologists" seems less pertinent today.

which he firmly believed. He consequently applied himself to attempting to isolate substances that could be synthesized by the brain during the course of ECT ("vitalizing substances of extreme defense") [41 44]. He prepared emulsions of brain tissue from treated animals, named these substances *acroagonins*, and administered them to patients. Though these attempts were destined to lead nowhere, they mark a change in the course charted by neuropathology, which moved more towards the study of neuromediators. Cerletti thus realized the need to go beyond his own invention, to assure patients the benefits of ECT without the side-effects that it carried at the time (and no longer carries today, in the patients with severe drug-resistant psychiatric disorders in whom it is still used) [49]. Cerletti's name also felt the burden of these investigations [41], particularly in the 1960s and 1970s, for inventing a treatment that had made him infamous or, at best, exposed him to a degree of *damnatio memoriae* [40].

Conclusions

"There was a time when certain psychiatrists would not have been considered proper scientists if they had not focused their best energies as researchers on the mortuary slab, the microscope, and laboratory work" [50], wrote Enrico Morselli, a fundamental figure in the history of Italian psychiatry. He was one of the most influential clinicians in the early 20th century and it was he who labelled histological studies on mental disorders as "histomania". It was true that, all too often, autopsy left psychiatrists dumbfounded [51]. Even the studies on the psychological trauma induced by explosions (an unprecedented opportunity for investigating the relationship between symptoms and supposed lesions), and the research done by Ugo Cerletti on the effects of electroshock contributed to the downfall, in the early decades of the 20th century, of a certain idea of histological malleability of the brain. Psychiatry went in other directions, albeit with some delay in Italy attributable to a diffidence blanketed in "positivism" regarding psychoanalysis, and to the advent of the Fascist autarky in the sphere of science. The path taken by psychiatry was dictated by the knowledge available at the time, which suffered from the absence of the modern neuroscience, and particularly the advances made by molecular biology and psychopharmacology, but the discipline was already oriented towards occupying its own space in the scientific world, and not biology or abstract science of the spirit. Nevertheless, the drive towards "dissecting" the psyche, and the belief in the feasibility of breaking it down into simpler elements under the effect of morphine, sleep or hypnosis, was born in minds of Freud and Charcot, also because of the anatomic and histopathological imprint on their scientific education.

Conflict of interest statement

None declared.

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