Hypothesis

Syndrome E

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The transformation of groups of previously nonviolent individuals into repetitive killers of defenceless members of society has been a recurring phenomenon throughout history. This transformation is characterised by a set of symptoms and signs suggesting a common syndrome—Syndrome E. Affected individuals show obsessive ideation, compulsive repetition, rapid desensitisation to violence, diminished affective reactivity, hyperarousal, environmental dependency, group contagion, and failure to adapt to changing stimulus-reinforcement associations. Yet memory, language, planning, and problem-solving skills remain intact. The main risk factors are male sex and age between 15 and 50. A pathophysiological model—"cognitive fracture"—is hypothesised, where hyperaroused orbitofrontal and medial prefrontal cortices tonically inhibit the amygdala and are no longer regulated by visceral and somatic homeostatic controls ordinarily supplied by subcortical systems. It is proposed that the syndrome is a product of neocortical development rather than the manifestation of a disinhibited primitive brain. Early recognition of symptoms and signs could lead to prevention through education and isolation of affected individuals.

Repeatably throughout history, groups of individuals, usually young men, have violently attacked other members of society, often with the approval of, or encouragement from, those in authority. The victims are usually defenceless and are no direct threat to their attackers. Some of the notable manifestations of the phenomenon in this century are the killing of Armenians by Turks in 1915–16, of European Jews during World War II, of Cambodians during the Pol Pot regime in the 1970s, and the ethnic killings in Rwanda in the 1990s. Civil strife, extreme conditions, and ethnic conflicts have often had a role in these events, much as poverty and lack of hygiene lead to outbreaks of infectious disease. Yet these events could not have happened without a distinct transformation in the behaviour of individuals. The uniform and repeating nature of this transformation suggests a common syndrome affecting individuals for which I propose the term Syndrome E.

Symptoms and signs

Repetitive acts of violence The hallmark of the syndrome is the perpetration of repetitive acts of violence. The individual engages in stereotyped repetition of aggressive, characterised by compulsion to spare none of the victims.1,2

Obsessive ideation Individuals are obsessed with a set of beliefs, often directed against a minority group. The combination of repetitive stereotypic acts accompanied by recurring ideation resembles the symptoms and signs of obsessive-compulsive disorder. Metaphors such as "cleansing" are often used to justify violence.3,4

Perseveration Individuals show stereotyped behaviour that perseverates in the face of changing circumstances, even when it is no longer appropriate for the situation and may jeopardise the individual’s interests.5 This behaviour reflects failure to adapt to changing stimulus-reinforcement associations.4

Diminished affective reactivity Much of the violence perpetrated by individuals is carried out, not in battlefront frenzies nor in a burst of emotion, but in a flat effect.

Hyperarousal (Rausch) The repetitive nature of violent acts often invokes a sense of Rausch (elation) according to the number of victims and the magnitude of destruction.5

Intact language, memory, and problem-solving skills Affected individuals show that the syndrome usually appear intellectually sound and remain intact in cognitive domains such as language, memory, and the ability to plan and solve problems.

Rapid habituation In the early stages of the syndrome, there is rapid habituation to acts of violence. While some report strain following homicidal activity, especially to arid children and omen, most individuals undergo desensitisation in a relatively short time.6

Compartmentalisation Individuals conduct activities calling for seemingly conflicting cognitive states. They lead a normal family life, while in parallel engaging in killing of families.6

Environmental dependency Behaviour is abnormally dependent on environment and context, which determine the borders of the behavioural compartments described above. Obedience to authority and dependence on group support are ample.

Group contagion The group environment is necessary for maintenance of the syndrome and for its propagation. Most individuals in the group respond uniformly to sets of stimuli, and the responses of individuals in the group serve as stimuli for other individuals.

Several of the signs and symptoms of Syndrome E are repeated in similar acts. The act of killing in front of a...
photographer is an ample of diminution of affective reactivity, but also suggests elation associated with the killing. Compartmentalisation is suggested by the mailing of this and other similar photographs by German soldiers along with accounts of the events to their families at home.

Risk factors
The clearest risk factors are male sex and age of between 15 and 50. There have been attempts to identify other risk factors, including certain personality traits such as authoritarian personality, Steiner’s notion of the “sleeper”, and obedience to authority. However, the evidence on this is contradictory, and there is no simple set of rules for identifying the risk of Syndrome E. However, the evidence on this is contradictory, and there is no simple set of rules for identifying the risk of Syndrome E.

Differential diagnosis
Syndrome E is a specific set of symptoms and signs. It is not a general syndrome of human aggression and should not be confused with the behaviour of individuals who carry out repetitive homicidal acts alone. These individuals constitute a very small proportion of the population, and the often have a history of antisocial personality. Neither should Syndrome E be confused with the usual manifestations of war or group combat. These conflicts often lead to atrocities, carried out by individuals in the frenzied battle. Such behaviour, however, lacks most of the features described above and is characterised by inverse symptomatology: emotional reactivity rather than dulling, and incidental, sporadic violence rather than perseveration and stematic repetition.

Pathophysiology
Prefrontal cortex
Most features of Syndrome E are found in disorders involving prefrontal cortical systems and their subcortical connections. The prefrontal cortex has three major systems: dorsolateral prefrontal cortex, orbitofrontal cortex, and medial prefrontal cortex. The dorsolateral prefrontal system appears relatively unaffected in individuals with Syndrome E since they are capable of planning, problem solving, language, and working memory. Obsessive ideation and repetitive acts of violence suggest orbitofrontal hyperkinesis, similar to that found in patients with obsessive-compulsive disorders. The motivation and elation accompanying repetitive homicidal acts suggest hyperactivity of the medial prefrontal cortex. This elation may be similar to drug-induced hyperarousal syndrome such as that induced by cocaine seen in rhesus monkeys and human beings. The medial prefrontal cortex is a major site of activation associated with compulsive self-administration of addictive drugs.

“Cognitive fracture”
In common with patients with ventromedial prefrontal dysfunction, individuals with Syndrome E cease to generate emotions appropriate to the images conjured by certain categories of situation and stimuli. Responses of the prefrontal cortex fail to arouse the autonomic, musculoskeletal, and endocrine substrate of emotion; nor are these responses regulated by somatic and visceral homeostatic controls. In Syndrome E, the prefrontal cortex becomes functionally disconnected from the deep or other neural centres that are here termed “cognitive fracture.”

Amygdala
The primary pathology underlying cognitive fracture may be in the interaction between the amygdala and the ventromedial prefrontal cortex. The amgdala has efferent connections with the orbitofrontal and medial prefrontal regions and has robust output to the hypothalamus and brain-stem nuclei, here it is presumed to regulate the autonomic, musculoskeletal, and endocrine substrates of emotions. Studies in animals suggest that the amgdala is tonically inhibited by prefrontal activation. The combination of cortical hyperarousal and diminished affective reactivity in Syndrome E may be due to hyperactivity of the orbitofrontal cortex and medial prefrontal cortices shutting off the amgdala and impairing its ability to regulate emotion and impart species-specific meaning to stimuli. Affected individuals then simultaneously perceive their victims as living and non-living, but at the same time, the mechanical repetition and compulsion, the approach a state of automation, in which intrinsic uncertainty about their own sense of living. Despite the rapidity of the behavioural change in Syndrome E, its expression is probably preceded by a longer learning process. Individuals undergo implicit emotional learning, resulting in an attitude to and their victims that is similar to phobias in patients or fear-conditioning in rats. These deep-rooted attitudes are resistant to extinction. Such extinction depends on output from the medial prefrontal cortex to the amgdala, which is impaired in Syndrome E.
Neocortical development vs subcortical disinhibition

The symptoms and signs of Syndrome E are sometimes viewed as animal-like, and attributed to a phylogenetically older “visceral brain” released from the inhibitory control of the neocortex. However, the possibility of observation in higher nonhuman primates, animals do not show the signs of Syndrome E. The symptoms and signs in Syndrome E are more characteristic of the product of neocortical development rather than of a disinhibited primitive brain. The hypothesis of cognitive fracture affecting a large number of individuals suggests vulnerability in the human neura is at the interface between the rapidly evolved prefrontal corte and the phylogenetically older subcortical nuclei.

Testing the hypothesis

It is likely that recovery from Syndrome E is accompanied by the presence of abnormalities that are indicative of unaffected members of the same group and unaffected members of other groups could be informative.

Affective reactivity

Compared with control groups, individuals with Syndrome E would be expected to show diminished changes in skin conductance, facial electromyography, and potentiation of startle reflex responses when presented with affective stimuli.13,14 The influence of the facilitation of memory for stimuli is negative emotional valence seen in unaffected individuals. Not only would the selective decrease in memory for such stimuli be the result of administration of beta-adrenergic-receptor antagonists.11,12 In common idiopathic patients with ventromedial prefrontal dysfunction, these individuals would not choose advantageous or generate anticipatory skin-conductance responses because they wonder a risk choice in gambling experiments.19

Perseveration

Individuals with Syndrome E should perform poorly on orbitofrontal tasks, such as rapid alteration of stimulus-reinforcement associations, but the should not differ from controls in performances on tests of dorsolateral frontal function, such as Wisconsin Card Sorting.4

Prefrontal hyperarousal

In disorders such as schizoaffective disorders, there is permetabolism and increased blood flow during the acute episodes but opposite changes after these episodes.20 Similarly, it could be expected that individuals with Syndrome E ill show acute hyperfrontality during outbreaks, but long-term hyperfrontality as measured by positron-emission tomography, single-photon emission computed tomography, and event-related potentials. Violent behaviour seen in youth gangs share the symptoms of Syndrome E compulsive repetition, diminished affective reactivity, hyperarousal, and group contagion. Study of gang members may be more feasible than studies involving full-blown manifestations of Syndrome E.

Prevention

Isolation of individuals with Syndrome E may offer hope of containing an outbreak, because the syndrome is ill not persistent or propagate without contact with other individuals at risk or in potential victims. Prompt diagnosis could be of paramount importance as prevention may be effective only in the early stages. The signs and symptoms of Syndrome E should be made known. Individuals in most societies know that a constellation of high fever and coughing may indicate pneumonia. In the same sense, people might become aware that Syndrome E is a sign of an emerging obsessive ideologue, hyperarousal, diminished affective reactivity, and group-dependent aggression directed at members of other subgroups who signify Syndrome E.

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References