14.3 CAUSES AND PREVENTION OF AGGRESSION FROM PSYCHOTIC INPATIENTS

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Background: Patients with schizophrenia and other psychotic disorders have an increased likelihood of engaging in violent behavior. These increased risks of dangerous and aggressive behavior, in combination with a lack of insight in their own illness, relatively often make involuntary admission of acutely disturbed psychotic patients on locked psychiatric admissions wards often inevitable. On these locked psychiatric admissions wards, aggression from psychotic patients against staff and fellow patients is a prevalent phenomenon, with the mean in the Netherlands being about 18 aggressive incidents per bed per year on locked psychiatric admissions wards.

Methods: In the lecture, a model of what causes or triggers aggressive behavior on (locked) psychiatric wards is presented. In this model, patient, ward and staff variables are integrated to explain why, and in what specific situations, psychotic patients particularly run a high risk of engaging in aggressive behavior.

Results: Based on the presented model, a number of preventive measures can be formulated. On the patient level, the administration of anti-psychotic medication is used to reduce the negative cognitive schemes and delusional thoughts that are depicted in the center of the model. A more novel intervention at the patient level may be the additional administration of nutritional supplements with (among others) high levels of omega 3 fatty acids. The results of two Dutch studies on this topic will be briefly presented in the lecture, among which a RCT on the effects of the use of nutritional supplements on aggressiveness. On the staff level, the use of short-term (daily) risk assessments by the ward nursing staff, among others by means of the six item BrØstel Violence Checklist (BVC), has been found to reduce aggressiveness and the use of coercive measures on psychiatric wards in two cluster randomized RCTs.

On the ward level, studies indicate that aggression on psychiatric wards can be reduced by preventing overcrowding on psychiatric wards, and by providing more space and privacy to the patients.

Discussion: The proposed model elucidates how certain patient, staff and ward characteristics may interact in causing aggression. The model also emphasizes that repeated inpatient aggression may be the result of a vicious circle, i.e. inpatient violence is often followed by an increase in environmental and/or communication stress on the patient, thereby heightening the risk of a repeated outburst of violence.

14.4 FOLLOW-UP TREATMENT FOR INDIVIDUALS WITH SERIOUS MENTAL ILLNESS WHO HAVE COMMITTED MAJOR CRIMES

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Background: For individuals who have a psychiatric disorder and have committed a major crime, the rate of re-offending is twice as high in the US compared to nine other countries for which there is comparable data. For such individuals the average five-year rearrest rate is approximately 40% for those released from psychiatric hospitals and 60% for those released from jails or prisons. The use of treatment modalities such as extended conditional release, Forensic Assertive Community Treatment (FACT) teams, and Psychiatric Security Review Boards can reduce the rearrest rate from 40–60% to 10% or less.

Methods: All 50 states were surveyed to assess how they were doing in providing follow-up treatment for such individuals.

Results: Sixteen states were found to be making a moderate effort to provide follow-up treatment, and another 13 states are making a minimal effort. However, the other 21 states, 42% of the total, are making virtually no effort, lending to an unnecessarily high rate of re-offending.

Discussion: Using proven treatment approaches the re-arrest rate of individuals with serious mental illness can be reduced from 40–60% to 10% or less.

Plenary

15. ON THE ROAD TO CURING SCHIZOPHRENIA

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Overall Abstract: I began my journey to find out what caused schizophrenia around the time me and my twin brother, Scott David, turned 17. My first step was to conceptualize schizophrenia as a biological, cellular and molecular brain problem. This guided my choice of undergraduate and graduate study. I quickly realized that schizophrenia was not a “genetic” disease, nor was it an “environmental” disease, it was both. I prioritized studying RNA as was the active genome, the subcellular substrate where genes and environment interact. Guided from my own experience of watching my normal twin be tortured by schizophrenia in his teens, I sought to find answers by studying the mammalian brain as it developed and changed during adolescence. For my post-doctoral fellowship, I joined the laboratory of Joel Kleinman, who has the largest and best characterized human brain collection of people with schizophrenia in the world.Along my journey, while at NIMH in the USA, I discovered changes in neuronal growth factors and hormone receptors during stages of postnatal life and in the brains of people with schizophrenia compared to controls using the classical hypothesis-driven approach. Since I moved to Sydney Australia, I choose a different, more open-minded approach and let the brains of those who suffered “tell me what happened to them”, using a modern, sensitive discovery-driven RNAseq approach. When I listened, more carefully at the molecular level, what I found told me that I may be headed down the wrong path with my research and that I needed to change direction. It suggested that the emphasis I placed on development molecules maybe in some ways blinding me from more clearly seeing the neuropathology that existed only in only some people at the time of death. I found elevated inflammatory cytokine mRNAs in ~40% in the brains of those diagnosed with chronic schizophrenia. In this talk, I will review my latest discoveries on neuroinflammation in schizophrenia including evidence of gliosis, blood-brain barrier (BBB) changes and increased white blood cells in the brains of some with schizophrenia. Today, many of my fellow seekers including geneticists (Chr 6, MHC locus) and epidemiologists (maternal infection) and “animal modelers” (poly I:C) are suggesting that the cause of schizophrenia may involve changes to the immune system. These new discoveries suggest that very first steps I took may have been wrong, that perhaps I should have become an immunologist rather than a neurobiologist. However, from my current vantage point, I believe that even if a fault in the immune system