BOOK REVIEW

SCHIZOPHRENIA - AN INTEGRATED VIEW

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This book is a collection of Proceedings from a Symposium held at the Royal Danish Academy of Science & Letters in Copenhagen from 5th to 9th June 1994. These contributions and the subsequent discussion represent the most up-to-date research in Schizophrenia.

Participants were mainly from Scandinavia with the rest coming from the United Kingdom, (notably Dr T J Crow), Germany, the Netherlands, France and the United States.

The book is broadly divided into 4 sections namely: (a) genetics, (b) neuroanatomy, (c) research into neurotransmitters, and (d) neurophysiology.

The first paper by Dr T J Crow attempts to correlate accepted anatomical changes namely brain asymmetry and ventricular enlargement with genetics. It noted that several recent radiological studies have observed that brain asymmetry was more often present in women compared to men. It concluded that sex differences in brain asymmetry can be correlated with the consistent sex difference in cognitive abilities – females having a modest mean advantage in verbal fluency and males an advantage in spatial ability. According to Darwin's theory of sexual selection, this difference must arise from a difference by which males and females select their mates. Quoting a recent cross cultural comparison by Buss[1], intelligence, personality, kindness and understanding were rated especially high by both sexes as desirable qualities in a partner. However, men rate physical attraction more highly than women, and women rate earning capacity higher than men. According to Dr Crow, this is perfectly understandable on the basis that men have an interest in females who are healthy, young and therefore fertile whilst women tend to be interested in a mate who is likely to provide resources. This is reflected in a cross-culturally stable difference in age at marriage; males are a mean 2 to 3 years older than females. This is a sweeping conclusion as the author then concludes that this age difference in mate selection very probably accounts for the difference between the sexes in cerebral asymmetry!! I have read this chapter several times and I still fail to understand how this conclusion can be arrived at.

Other chapters looked at recent advances in neuroanatomy and developmental success. There were extensive reviews of research on morphological abnormalities such as enlargement of the lateral and 3rd ventricles, the 40% to 50% percent reduction in neuron numbers in the mediodorsal thalamic nucleus and nucleus accumbens, and temporal horn enlargement. Others looked into research on the various dopamine receptors, serotonin in the actiology of Schizophrenia as well as exploring the role of glutamate metabolism in the pathogenesis of the illness.

Several questions were posed; the majority remain unanswered. For example, are the brain changes progressive or is it a one-off event? There have been very few studies that have looked into serial changes in the brain. It is also unclear whether the brain abnormalities were already present prior to the onset of Schizophrenia.

As the book consists of chapters contributed by different participants, the style was rather uneven. I personally found the chapters to be fairly heavy going and since the material presented was essentially research based, it is not immediately accessible and requires a certain ground knowledge of the subjects concerned.

It makes a good reference and should appeal to research scientists with their relevant special interests. However it is not a book I would recommend for the general psychiatrist or for the trainee.

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REFERENCE

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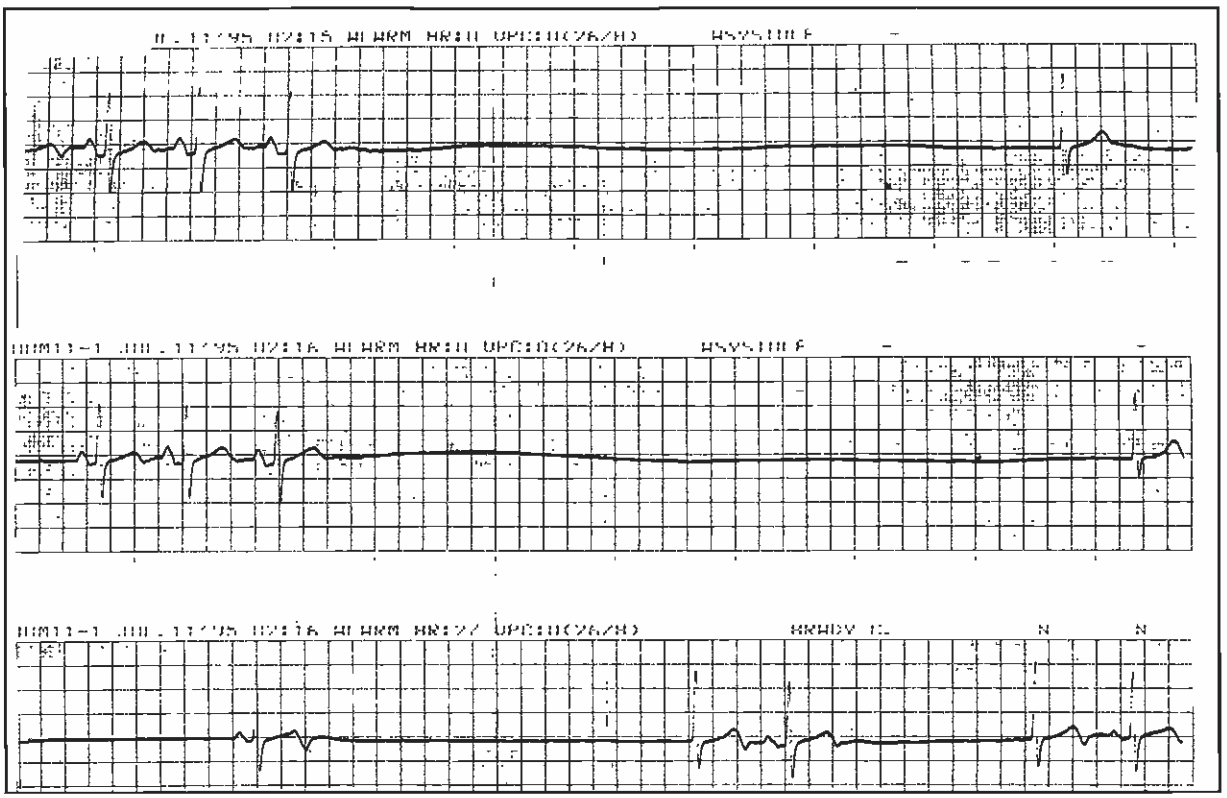
Fig 2 – ECG as monitored by telemetry showed sinus arrest with transient asystole and longest RR interval of 7.2 seconds.



ANSWER TO ELECTROCARDIOGRAPHIC CASE

Diagnosis: Sick sinus syndrome

DISCUSSION

This patient's electrocardiogram shows sinus rhythm with a normal p wave morphology and transient asystole as shown on the rhythm strip. The pause is interrupted by a sinus beat which is not a multiple of the PP interval. This suggests that the asystole is due to sinus arrest rather than sinoatrial exit block provided there is no mark sinus arrhythmia, as described by Irene Ferrer in her classical monograph[1].

The patient was monitored in the ward with telemetry and Fig 2 illustrates the documented arrhythmia associated with giddiness. There was asystole of up to 7.2 seconds which confirmed that the patient's episode of syncope was due to sinus arrest associated with the sick sinus syndrome.

The electrocardiographic differential diagnosis of pauses include sinus arrest, sinoatrial exit block, mark sinus arrhythmia, nonconducted atrial ectopic, Mobitz type II and complete AV block. In mark sinus arrhythmia, holding of breath during ECG recording may minimise the arrhythmia[1]. In nonconducted atrial ectopic, there is a P wave preceding the pause although sometimes it is hidden in the preceding T wave causing a distorted T wave. Sinoatrial exit block is differentiated from sinus arrest by measuring the PP interval. In sinoatrial exit block, the PP interval during pause is a multiple of the PP interval preceding it. Both sinus arrest and sinoatrial exit block are common features of the advanced stage of the sick sinus syndrome.

The sick sinus syndrome is caused by dysfunction of the sinus node in its impulse formation or its ability to transmit its impulse to the atrium[1,2]. It is usually manifested as an inappropriate bradycardia in the early stage. Patients with symptomatic sick sinus syndrome usually have brady-tachycardia syndrome where intermittent supraventricular tachycardia is interposed between the bradycardia. Many patients (30%-50%) have additional conduction defects including AV block, intraventricular conduction defect and bundle branch block[1-3].

In the ECG, the sick sinus syndrome may present as one or more of the following[1-3]:

1. Severe sinus bradycardia.
2. Sinus arrest or sinoatrial exit block with varying severity.
3. Brady-tachycardia syndrome.
4. AV junctional escape rhythm.
5. Chronic atrial fibrillation with failure to resume sinus rhythm post cardioversion.

Clinically sick sinus syndrome can be divided into acute and chronic type[4]. Acute sinus node dysfunction is usually due to acute coronary events eg acute myocardial infarction; drugs induced such as betablockers, digitalis, quinidine, calcium channel blockers, amiodarone; electrolyte imbalance eg hyperkalaemia or post cardiac surgery. Chronic sinus node dysfunction is usually idiopathic or due to underlying heart diseases such as hypertension, coronary artery disease, valvular heart disease or myocarditis/cardiomyopathy[4].

For the diagnosis of sick sinus syndrome, 12 leads ECG with long rhythm strip and/or 24-48 hour Holter monitoring is usually sufficient[5]. Some patients may need pharmacological testing with atropine or isoprenaline. Rarely, invasive electrophysiological studies may be needed to measure sinus node recovery time which is usually abnormally prolonged[6].

In the management of sick sinus syndrome, especially in patients with brady-tachyarrhythmia, it is particularly important to note that the drugs used to suppress the tachycardia may aggravate the bradyarrhythmia. The treatment also include

correcting the primary causes, if possible. A permanent pacemaker is inserted if the patient is symptomatic. Adjunctive medical treatment to suppress tachyarrhythmia include digitalis, betablocker or calcium antagonists. Anticoagulation is also now usually recommended because of its frequent association with thromboembolism[7]. There are preliminary studies to show that pacing the atrium may prevent atrial fibrillation, reduce thromboembolism and improve survival[8]. The above patient was managed by inserting a permanent pacemaker.

References

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Schizophrenic illnesses occur with approximately the same incidence in all human populations with a characteristic distribution (slightly earlier in males) of ages of onset. Given that the predisposition (which presumably is genetic) is associated with a procreative disadvantage why do such illnesses persist?Â Schizophrenia as failure of hemispheric dominance for language. @article{Crow1997SchizophreniaAF, title={Schizophrenia as failure of hemispheric dominance for language.}, author={Tim J. Crow}, journal={Trends in neurosciences}, year={1997}, volume={20 8}, pages={. 339-43 } }. Tim J. Crow. Published in Trends in neurosciences 1997. Schizophrenia. an Integrated Sociodevelopmental-cognitive Model - Free download as PDF File (.pdf), Text File (.txt) or read online for free. Schizophrenia. an Integrated Sociodevelopmental-cognitive Model.Â Much more than documents. Discover everything Scribd has to offer, including books and audiobooks from major publishers. Start Free Trial. Cancel anytime. Schizophrenia. an Integrated Sociodevelopmental-cognitive Model. Uploaded by. andresgarciaf. 0 Up votes0 Down votes. 24 views. 11 pages. Document Information. click to expand document information. Description: Schizophrenia. an Integrated Sociodevelopmental-cognitive Model. Date uploaded. Mar 23, 2015.