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MENTAL HEALTH SYSTEM IN INDIA

Arabinda Brahma

Mental health deals with human thoughts and emotions and helps to form healthy minds. It provides the foundation for creating a mentally healthy human being and in turn a healthy society. Any deviation may lead to mental illness or problem and may cause severe burden upon society and nation¹. Various mental illnesses were well documented in the ancient Indian texts. *Atharva veda*, an ancient Indian scripture, mentioned different mental illnesses, which were thought to occur as a result of divine curses. In the vedic texts, description of schizophrenia and mood disorders like conditions were clearly described. The great Hindu epics like *Ramayana*, *Mahabharata* and in the scripts of the *Bhagavad Gita*, various disordered states of mind were found.² In the modern day psychotherapy, scripts of the *Bhagavad Gita* are used as a very reliable source of different coping strategies during the period of stress. Another ancient branch of alternative medicine in India - *unani* system made different references to the common mental illnesses like schizophrenia, depression, paranoia, organic mental disorders etc.³ The famous Ayurvedic manuscript, *Sushruta Samhita* by Sushruta, described the individual role of the physician (chikitsak), the medicine (dravya), the nursing staffs (upasthata) and the patient (rogi) in case of a successful therapeutic management. Similarly, in *Charak Samhita* by Charak, the administrative aspects of a treatment centre (hospital), maintenance of hygiene were narrated elaborately.

In the British India between 1745 and 1794, three lunatic asylums were established in Bombay (Mumbai), Calcutta (Kolkata) and Madras (Chennai). However, the main purpose of these asylums was to offer custodial treatment than to provide curative treatment. Between 1858 and 1918, a steady increase in the development of mental asylums were noted in different parts of India along with the introduction of drug treatment mainly to control the maladaptive behaviors found in various mental illnesses. The first psychiatric outpatients department in a general hospital was established at the R G Kar Medical College, Calcutta in 1933 by Girindra Sekhar Bose.

A significant strategic development was noticed after the independence in 1947. The government of India gave more focus on establishing general hospital psychiatry units than to building more mental hospitals. This has also helped to develop consultation - liaison psychiatry practice in India. In the last five decades, mental health activities have emphasized more on the prevention and promotion of mental health rather than to treat mentally ill persons only.⁶ Emphasis was placed upon combating stigma related to the mental illnesses and to develop effective community mental health services.

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Following inclusion of mental health care services in Primary health care, a major change was observed in the management of mental illnesses. However, some shortcomings are still evident after incorporating this decentralization policy. It is definitely a challenging task for the present day mental health professionals and the policy makers to bridge this gap. Successful implementation of the recent advances in the area of therapeutics and neuroscience is also important to the mental health professionals in managing the mentally ill persons.

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METABOLIC SYNDROME AND MENTAL ILLNESS

Arabinda Brahma¹, Mahuya Chatterjee Brahma²

Introduction:

Many studies have shown the substantial size of medical problems in the psychiatric ill patients. These medical problems may have adverse effects on quality of life, poor compliance with effective pharmacological treatments and sometimes on long-term physical morbidity and mortality. Metabolic syndrome has gained much attention in the last few decades amongst the mental health professionals because of its close association with some psychotropic drugs. Metabolic syndrome is a complication of established individual risk factors for coronary artery disease (CAD) along with increased incidence of heart diseases and diabetes mellitus (DM). All of these can cause mortality.

In 1988, Reaven proposed the term "Syndrome X" and identified CAD risk factors of dyslipidemia, hyperglycemia and hypertension. Currently metabolic syndrome is also known as "deadly quarter", "dysmetabolic syndrome" and "insulin resistance syndrome". The core components of metabolic syndrome are: Hypertension, Dyslipidemia, Glucose dysregulation, and Obesity.

Two commonly used definitions of metabolic syndrome have been developed by — WHO, and National Cholesterol Education Program - Adult Treatment Panel III (NCEP-ATP III).

	Component	Abnormality
NCEP-ATP III	3 or more of following:	
	Abdominal obesity (waist circumference)	
	Men	> 40 inches (102cm)
	Women	> 35 inches (> 88cm)
	Triglycerides	≥ 150 mg/dL
	HDL Cholesterol	
	Men	< 40 mg/dL
	Women	< 50 mg/dL
	Blood Pressure	≥ 130/≥ 85mmHg
	Fasting Glucose	≥ 100 Mg/dL

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	Component	Abnormality
World Health Organization	Insulin resistance (indicated by any 1)	
	Type 2 Diabetes	
	Impaired fasting glucose	
	Impaired glucose tolerance	
	For those with normal fasting glucose	
	glucose uptake below the lowest quartile for background population under investigation under hyperglycemic, euglycemic conditions	
	Plus 2 of following :	
	Blood Pressure	antihypertensive medications ± BP ≥ 140 mm Hg/ ≥ 90 mmHg
	Plasma Triglycerides	≥ 150 mg/dL
	HDL Cholesterol	
	Men	< 35 mg/dL
	Women	< 39 mg/dL
	Obesity	BMI > 30 kg/m ² ± waist to hip ratio > 0.9 inches in men & > 0.85 inches in women
	Renal function/Kidney ds.	Urinary albumin excretion rate ≥ 20 ug/min or albumin:creatinine ratio ≥ 30 mg/g

Mentally ill patients have an increased risk of developing metabolic abnormalities mainly because of the fact that they receive suboptimal preventive medical care and they are likely to be overweight. On the other hand, various researches have revealed that the uses of atypical antipsychotics are associated with the appearance of diabetes, cardiovascular disease, dyslipidemia, and weight gain. Clozapine and olanzapine are the two drugs most implicated but other atypical antipsychotics may also be involved.^{2,4} Therefore, patients who requires atypical antipsychotic therapy must be evaluated for any characteristics that would influence the decision to prescribe a medication regimen (e.g. for traits that would increase the patient's risk of developing diabetes in the future).⁵

Prevalence:

The prevalence of adult obesity in industrialized countries has been increasing. More than 60% of population in US currently fall into the category of overweight or obese.⁶ Psychiatric patients including those diagnosed as schizophrenia, bipolar disorders, or major depression, may have an increased chance of developing metabolic syndrome.

Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) Schizophrenia Trial to assess the prevalence of metabolic syndrome (as defined by NCEP) in schizophrenic patients,⁷ revealed an overall occurrence rate of 40.9% (51.6% for women; 36.0% for men). Moreover, it was found that males in CATIE study were 138% more likely to have metabolic syndrome in comparison to matched National Health & Nutrition Examination Survey (NHANES) subjects. Women in CATIE study group were 251% more likely to develop the syndrome in comparison to NHANES subjects.

A recent study involving bipolar disorder patients showed that according to NCEP-ATP III criteria, metabolic syndrome is as alarmingly prevalent in this group as it is in the general population.⁸ Another large study involving 3003 individuals screened for depression found that women with a history of Major Depressive Disorder (MDD) were twice as likely to develop metabolic syndrome as those with no history of depression.⁹

Risk groups most vulnerable to the metabolic syndrome include:¹⁰⁻¹¹

- i. Elderly
- ii. Higher Body mass index (≥ 25)
- iii. Women
- iv. Postmenopausal status
- v. Current smoking
- vi. Low household income
- vii. High carbohydrate intake
- viii. No alcohol consumption
- ix. Physical inactivity

Clinical significance:

Research has revealed that patients with metabolic syndrome are at increased risk for premature death, CAD, diabetes, polycystic ovary syndrome, fatty liver, cholesterol gallstones, asthma, sleep disturbances, and some forms of cancer.¹²

Psychiatric patients are at high risk of developing metabolic syndrome, CAD, Diabetes that may lead to increased overall mortality. Research has shown that schizophrenia patients have a 20% shorter life span than general population.¹³ Mood disorders patients also have high standardized mortality rates. In most cases, mentally ill patients have limited access to primary care physicians resulting in less chance of screening for common silent killer diseases like hypertension, dyslipidemia, diabetes and CAD.¹

Various studies revealed high rates of glucose dysregulation, insulin resistance, central obesity, and CAD among schizophrenic patients - both in patients on antipsychotic medications and in psychotropic-naïve patients. The susceptibility of developing diabetes in schizophrenia patients may be caused by a direct genetic link between these two diseases. However, weight gain, poor dietary habits and decreased physical activity seen in schizophrenia patients are probably the major risk factors for diabetes in this population.¹⁴ As patients on antipsychotic drugs have increased risk for developing pre-diabetes, diabetes, and dyslipidemia, well-defined strict guidelines now being developed to reduce onset of metabolic abnormalities in this patient population.¹⁵ Patients with MDD also showed increased chance of metabolic abnormalities and central obesity. Patients with MDD often have elevated pro-inflammatory markers, including C Reactive Protein, Tumour Necrosis Factor, Interleukin-I and WBC count. Studies have thrown light on the fact that the heightened pro-inflammatory state is likely to be at least partially responsible for 2-4 fold increase in mortality found with depressed patients following a myocardial infarction.¹⁶

The Metabolic Tetrad

The metabolic tetrad (core components) of the metabolic syndrome includes Glucose dysregulation, Hypertension, Dyslipidemia and Central obesity (mainly visceral obesity). All of these entities are highly prevalent, easily assessed, potentially deadly, often preventable and usually treatable.

i) Glucose Dysregulation

About one-third of individuals with diabetes is often asymptomatic and remains undiagnosed. Studies have showed that about three times increased prevalence of diabetes in schizophrenic patients in comparison to general population. Moreover, patients on atypical antipsychotics carry increased risk of developing hyperlipidemia and glucose dysregulation. Therefore, patients who are on atypical antipsychotics should check fasting blood sugar before initiation of medication, again at week 12 and annually thereafter.¹⁵

ii) Hypertension

The 7th report of Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure stated the fact that normalizing BP can reduce incidence of stroke and myocardial infarction (MI) by 35% and 25% respectively. Furthermore, normotensive individuals at age 55 have a 90% chance of eventually developing hypertension. Therefore, psychiatrists should refer their patients to primary medical care for monitoring BP at regular intervals.

iii) Dyslipidemia

Dyslipidemia is one of the most important modifiable risk factors. An elevated Serum Triglyceride level ($> 150\text{mg/DL}$) indicates future development of CAD. Elevated LDL cholesterol is another major lipid marker for CAD risk. For every 30mg/DL change in LDL, relative risk for CAD is changed proportionately by 30%.¹² It is always best to check a fasting lipid profile beginning at age 20 and about 5 yrs thereafter.¹⁹ Lifestyle changes with diet, exercise and lipid-lowering agents may be considered as primary preventive measures.

iv) Obesity

The best indicator of obesity is the BMI. BMI $> 25\text{--}29.9\text{ kg/m}^2$ is considered as overweight and BMI $> 30\text{ kg/m}^2$ as obesity. Obesity is an independent risk factor for heart disease and is associated with Type 2 diabetes, hypertension, hyperlipidemia, osteoarthritis, sleep apnoea and increased death rate for most cancers.

Psychogenic reactions

In patients of metabolic syndrome who suffer primarily from obesity may develop depression or anxiety following a weight loss program. When a vast amount of weight is lost, the patients may develop psychosis during or after the program.⁷ Irritability or labile mood is common in metabolic syndrome patients who have fluctuating level of blood sugar. Fatigue is another common psychogenic reaction found in patients of metabolic syndrome.

Management

The primary goal of management of metabolic syndrome is to reduce the risk related to it like vascular-related diseases, fatal and non-fatal stroke and coronary heart disease.²⁰ Treating all CAD risk factors like hypertension, diabetes, dyslipidemia, obesity and cigarette abuse is the cornerstone of therapy. Intensive glucose control can reduce microvascular complications of diabetes (retinopathy, neuropathy, and nephropathy) and lessen cardiovascular events. Treating hypertension reduces risk of future cardiovascular events, especially stroke and MI. Lifestyle modification is also very important. Maintaining normal weight or reducing weight, increasing physical activity, low sodium intake ($< 2\text{g/day}$), reducing alcohol consumption (< 2 drinks/day for men, 1 for women), having diet rich in fresh fruits and vegetables, low fat dairy products, low fat and saturated fat found to be beneficial.

Conclusion

With the proliferation of general hospital psychiatric units all over the world, mental health professionals would need to know the common medical complications, which are associated either with the psychiatric illnesses or with the use of psychotropic drugs. Early recognition and treatment will definitely help to reduce the mortality associated with some fatal medical conditions like metabolic syndrome in the psychiatric patients.

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WHY SO MUCH VIOLENCE ?

— R.K. Brahma

Definition :

Violence has been defined variously as -

- Using excessive physical force directed at another individual.
- Physical aggression against other people.
- Human behavior that seriously injure or destroys persons/ living beings.

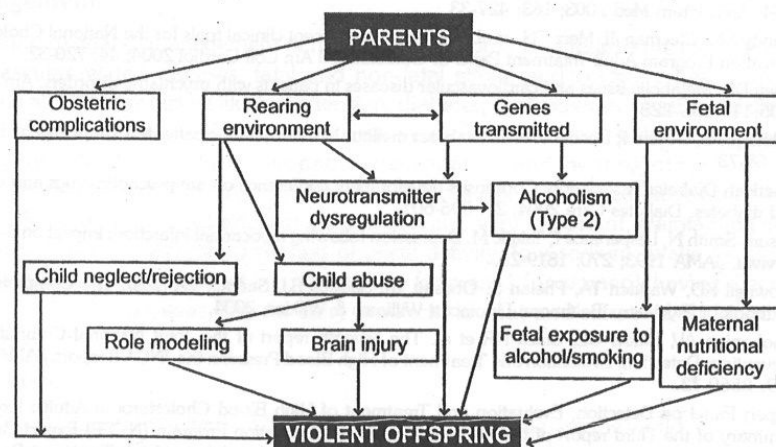
Origin and history:

Anger and aggression are human traits building blocks of human personality. Similarly aggression and violence directed against individuals and groups are as old as the history of man.

Biology of aggressive behavior -

Perpetrators of aggression and violence are not necessarily mentally ill, Nor can it be explained by biological factors alone, e.g. violence perpetrated daily seen in news cast are not by only terrorists and criminals mentally ill nor biologically predisposed. In effect transcription functions of genes are regulated by environmental factors, (vide Fig 1)

Figure 1 (from Wiley-Psychiatry)



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In the individual development portrayed in the table it is very obvious how important is the immediate environment in which the subject grows up along with the genetic make up, physical damage and substance abuse.

In the bigger portrait of life the influence of society become, evident :-

If the ethos of acquiring and possessing is inculcated as the god of life, by foul or fair means then not getting leads to anger leads to aggressive feelings of being deprived and denied rather than taking individual responsibility and introspection. This leads to blame culture that is to find the target and shoot it down.

Racism, sexism, communalism, culture supremacy:

History of aggression & violence is steeped in these isms.-

Racism - e.g. Racial superiority promulgated by Adolph Hitler against the Jews and non Aryans; White South African Regime propagating apartheid policies directing against the blacks; Crusade by Europeans against by non Europeans and latter day Jihads against non Muslims.

Sexisms - Prime examples of ultimate sexism was shown by Taliban regime in Afghanistan.

Communalisms - Alqaida movement proclaiming itself as the cleanser of the world's all Evils.

Cultural Supremacy - Cultural and political superiority claimed by US presidents like Ronald Reagan through its utterances like evil empires thrown at peoples and countries who did not support his ideology or dogma and declaring war against them; Bush putting himself forward as the keeper of democracy in the world.

Wanton and unlimited greed - leads to political murders, formation of mafias, drug cartels and human trading including Flesh trade and Slave trade etc.

Why no progress in eradication of violence:

Because vested interest groups, both on the right and left undermines any real attempt at reduction of violence. They both have their constituencies to pamper, use and exploit. Motives involved in this may be building a vote-bank by politicians of both left and right leanings; NGOs creating fire brand activists to increase their sphere of influence; Paranoid groups or sects in the society to increase their numbers to gain prominence e.g. Jeova's witness, Rosie Cussons, Ku Klux Klan etc, Black Panthers HUIJIS, Hijbul Mujahidins, Nava nirman party etc. Industrialists and right wing politician: too play their part -

a) To frighten tax payers that it is only aimed at taking their hard earned money to distribute amongst the violent criminals and ghettos, spawning such criminals and law breakers.

b) Stoking the fear psychosis by supporting and encouraging gun culture in American society.

c) Selling arms as a condition for aid to poor countries, aiding & abetting their armed conflicts, killings and massacres including spawning child soldiers and Jihadees.

Geopolitics :

Another name for nefarious activities in the International political scenario is geopolitics like Truman pampering a neighboring tyrannical dictator calling him "our son of a bitch"; USA over the decades causing, supporting and stoking strife, death and destruction in various countries around the world. These include supporting dictators like Batista in Cuba, Somoza in Nicaragua and encouraging terrorist activities of contras against elected government of Sandinistas by President Regan; toppling democratically elected socialist government of Chili and replacing it by a dictator - General Pinoche leading to civil war, death and destruction; Genocide and war crimes in Vietnam;; Bush arming Saddam Hussein's regime against Ayatolla's Iran. USA creating / supporting Mujahidins and Taliban against Russian supported government in Afghanistan, also encouraging and financially supporting Pakistan - sponsoring terrorists against India.

Prime examples of destroying of any program for Reduction of Violence :

UCLA Centre for the Study and Reduction of Violence - August 1972 to Dec73. The history of formation of this Centre - UCLA CSRV is a glaring but painful example of internecine warfare and paranoia amongst vested groups successfully destroying an unparalleled centre in the world in this field. A UCLA coed had been murdered in the campus in the parking structure, Assassination of Robert Kennedy, Zodiac Murders, Manson (Charles) "Family" murders and other such frightening examples led to the idea for creating a program to study life threatening behavior: This was conceived by Luis Jolyon West and Robert E Liftman of California University -Psychiatrist and Brain electro physiologists who were both often called upon by courts and other institutions to opine on the cause of extreme violence committed by young veterans & other similar groups. They could not pinpoint any sole biological cause or anyway of predicting such behavior. They felt that "nowhere is the need for holistic, multidisciplinary comprehensive research more obvious than in approaching violence and aggression.

Shaping of the Program:

This was being done with the support of California Dept of Mental Hygiene (now part of an amalgamated Dept of Health)

Its aim was divided in two parts -

a) Support for specific research, training and develop and aid service projects

b) In addition to the central core of admin and support staff-

- Formation of a team of visiting and resident specialists consisting -
- Psychiatrists
- Sociologists
- Humanities scholars
- Anthropologists
- Program planning evaluators
- Health educationists
- Police / Correctional workers
- Behavioral scientists
- Experts in behavioral Problems interface- from law/ Ethics / psychology

This meticulously detailed plan was sent by the Dept of health to the Governor's office. It was approved by the Governor. Establishment of the centre was announced in the "state of the State" address in Jan 1973.

The purpose of the centre :

Was defined as "To study a variety of pathologically violent behaviors, their causes and precursors, condition that foster or aggravate them; acceptable methods of preventing or diminishing such behaviors and preconditions, and techniques for treating or mitigating the harmful consequences. The program was to concentrate on individuals and small groups manifesting such behaviors, though recognizing that by far the most important causes of violence are social and that violent behavior can only be studied properly with in the social, political, economic and cultural context.

Perview of CSRV were to include :

Homicide, suicide, physical and sexual assaults including those occurring within such institutions like hospitals and prisons, senseless or maniacal killings, neighborhood gang killings, assassination, battering or abuse of children and certain aspects of death and destruction on the highways and airways. More than 100,000 crimes involving violence were reported in 1971 in California alone. However the true figures were suggested to be much higher.

Casual / Contributory Factors: Include Social factors:

- Problems within the family or group setting
- Educational / environmental deprivation - recruitment Field
- Poverty, prejudice and racism - Apartheid; aborigines in Australia, N.Z., conquistadors in S America, Crusaders in middle ages and Jihadees - oblates.
- Cultural and social alienations

Psychological factors :

- Impaired maturation - Gullible

- Character disorder - Psychopaths / Sociopaths
- Neuroses - Mass Hysteria induced by Hitler
- Psychosis or situational reaction like broken love affair - crime passionale e.g. Santiniketan Killing of female student by the young man from Ballygaunj phanri

Biological:

- Hormonal -testosterone (Male aggression for securing female partner), estrogen /fall in progesterone during premenstrual phase causing aggressive and violence behavior
- Metabolic - Hypoglycemia
- Dysfunction, damage or disease of CNS - Post traumatic brain damage, temporal dysfunction.
- Use of various drugs including alcohol, amphetamine, Cocaine etc.

Multiple factors are likely to co-exist, requiring complex formulation for the development of useful understanding of violent behavior, and to repeat; the social context is an essential element in every case.

Protocol of CSRV Program:

In the Protocol of CSRV Program it was made clear that studies of the biological / physiological aspects of violence would comprise of only a minor part of the totality of the center's activity in terms of funds spent, number of projects, background and number of staff. Still, radical student's organization called Students for a Democratic Society (SDS) pounced on the Project and associated it with UCLA Psycho - Surgery Project. In wildly accusatory article in "The Daily Brain" and the underground press. They Began to picket with Placards such as "clockwork orange at UCLA", "stop Psychosurgery at UCLA", "Fire Frank Erwin" and "drive west into the sea". All these were based on the fact that Frank R Erwin came from Harvard having served as the director of Stanley Crow Psychiatric Research laboratory of Massachusetts General Hospital where some patients of Psychomotor epilepsy with temporal lobe brain disease were treated by operative intervention. A few of these patients were violent.

The general flavor of these oppositions was overtly political, like "if Governor Regan is for it, we are against it." Some politically radical 2 or 3 Psychiatrists went to community groups and organizations like American Civil Liberties Union and Black Panther Party and persuaded them that the proposed centre was

in fact racist, fascist, sexist and dangerous to human rights; in fact nothing less than a Government sponsored program for mind control. In fact, in their opinion, "to offer hope for Psychiatric/ medical solutions to social problems was to waste time and money, and even worse to shift attention from the true source of violence and to blame the individual offender, rather than the repressive government, capitalistic economic system,

institutionalized racism, brutal laws enforcement policy etc. As the individual or groups would include some violent criminals found in prisons, these anti groups declared "The University will dignify through its research the very processes by which prisoners were abused" and expressed no trust in the university, instead extreme distrust was the rule.

In their view, the knowledge gained by the centre will be abused, probably for control or coercion of citizens who express dissent or deviation. In any case, knowledge is power and this centre would tend to increase the power of the state which already had too much power and have had instances of abusing such power. Not all argument against the centre came from the political left, the right was also against it claiming; A group of professors studying violence has no connection with the reduction of violence; The money should go for police equipment and law enforcement purposes instead. In their view, giving fund to the university to study and reduce violence is ridiculous since it is well known that in fact the Universities spawn and encourage violence.

The above set of facts are cruelly ironical as, groups representing those who had the most to gain from the UCLA work had been so easily persuaded to oppose without finding out the truth of the matter themselves. The statistical fact of violence in ghettos should not necessarily cause concern for research on the subject automatically racist; as research often shows that much of the violence amongst the poor and under privileged urban groups is a consequence of institutionalized neglect. In Effect the truth is to postpone or evade the challenge of this research opportunity was really the most racist of all available options.

Evaluative, Diagnostic and predictive studies:

These are the only logical steps towards prevention -

The Concept of clues (signs, symptoms and history) that aid in prediction or diagnosis is vital for individualizing treatment approaches and providing a humane response to any individual's behavior. In fact, greater predictive power — more likely to earlier release of the less dangerous person, rather than longer confinement for the more violent ones. If prediction is not improved, the most likely consequence is greater repression, more prolonged confinement for all offenders and even maintenance or restoration of death penalty.

Though violent acts may occur, it is only a very infrequent feature of TLE (only 9.1% of the violent criminals referred had distinctly abnormal EEG).

According to some radical theorist "Is it possible to make any sense out of studying organized brain tissue, should one not wait for full understanding of the cellular interactions and synaptic structures and details? Then, after a century of studying these elements and having come to understand them, simply add those elements together and one has a brain"! The argument further states that although one may be clinically impatient and rush in with the

EEG & EPs and similar tests, they are simply a way of keeping out of trouble until the "real scientists understand how to put a brain together". Brain and its surrounds are not to be separated one from another; it is a nexus of a lot of past history and current surround, that this little spacio-temporal matrix is terribly important at the moment, but cannot be usefully or fruitfully treated entirely in isolation if we are to think about it as the organ of behavior. It is absolutely amazing to find how otherwise sensible scientists or indeed, active radical critics of the social system willing to reduce the whole issue of "violence" to a simple construct, and thereby to embrace an idealist metaphysics, which, in the heat of the argument, lead to statements or implications that say "there is no brain" or "The Presence of the brain is irrelevant". This was destroying the intellectual caliber of scientific discourse in the last part of the twentieth century.

Causes of criminal violence:

Main Hypothesis:

1) Aggression as a fundamental instinct which may be expressed in undesirable aggressive acts as well as socially acceptable ways

2) Result of a frustration in a goal directed behavior

3) As learnt behavior resulting either from aggression rewarded or from observation or modeling

4) Several factors determining whether a person is aggressive in a particular social situation may depend on personality, immediate social grouping, behavior of the victim, disinhibiting factors like drugs and alcohol, general environmental factors like noise, social pressure and also presence of mental abnormality (personality disorders are the commonest form responsible for violence-psychopathy with or without abuse of alcohol and drugs or other mental conditions like marked paranoid or sadistic traits

- Part of the persistent pattern of impulsive and aggressive behavior -Impulse discontrol

- Sporadic response to stressful events in over controlled personality (Megargee, 1966) e.g. isolated unexplained campus violence followed by suicide by an otherwise quiet and well behaved student, also the character portrayed in the film psycho by Alfred Hitchcock.

- Cultural norms concerning expression of violence e.g. Honour killings prevalent in lower class of immigrant Muslim families in UK, criminal subculture and gangland culture in East of End of London.

- Bride burning, violent punishment dished out by a select group of self-important members of a caste often on members of another caste, usually of a lower one.

Table

Table 103-3 Key Issues in Assessment of Acute Agitation/Aggression/Violence

1. Somatic conditions
2. Previous history of violence
3. Access to weapons
4. Current ideation, including content of delusions
5. Active substance abuse, including alcohol
6. Comorbid antisocial personality disorder/traits or psychopathy
7. Verbal threats
8. Premonitory physical signs (clenched fist, pacing)

Table

Table 1034 Pharmacological Options for Acute Agitation — intramuscular Agents

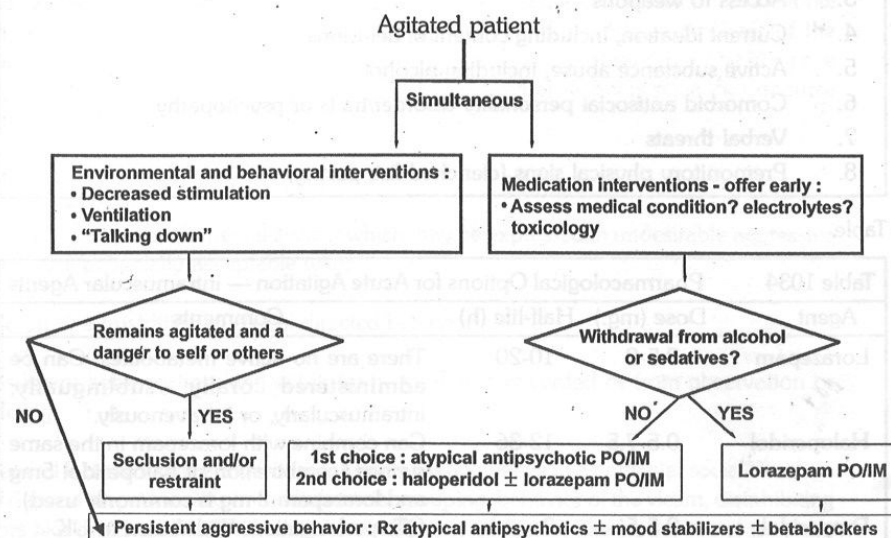
Agent	Dose (mg.)	Half-life (h)	Comments
Lorazepam	0.5-2	10-20	There are no active metabolites. Can be administered orally, sublingually, intramuscularly, or intravenously.
Haloperidol	0.5-7.5	12-36	Can combine with lorazepam in the same syringe (combination of haloperidol 5mg and lorazepam 2 mg is commonly used).
Droperidol	2.5-5	2	QTc prolongation. Withdrawn in UK.
Olanzapine	10 (2.5 for 34-38 patients with dementia)		Superiority over haloperidol (schizophrenia) and lorazepam (bipolar disorder) in clinical trials.
Ziprasidone	10-20	2.2-3.4	Superiority over haloperidol (schizophrenia) in clinical trials. QTc prolongation.

Forms of treatment offered for individual and small groups

- 1) Environmental management
- 2) Restraints
- 3) Compulsory confinement in a heavily programmed and behaviorally oriented management within a very secure campus - a treatment centre in Scandinavia for psychopathy and racistist, crafts centre in Wales for psychopathy and Bradmor hospital.
- 4) **Psychological methods**
 - anger control
 - relaxation

- impulse control
- Therapeutic community and group psychotherapy either in a context of a hospital, open prison or even a secure prison hospital for the criminally insane (e.g. groups conducted by Murray Cox).

(Wiley-Psychiatry) management of agitation - Algorhythm.



Conclusion :

The above suggestions about managing or reducing incidences of aggression and violence relates to only specific situations or scenarios. India from time immemorial has spread the message of peace - Upanishads and Puranas are full of utterances by Sages and Seers invoking peace and goodwill not only for mankind but for the living universe as a whole. In the yearly celebration of ancestral worship and prayers for their pleasure and satisfaction the mantras uttered is an example of the width and breadth of their peace invocations -

আব্রাহামবনাম্লোকা দেবর্ষি - পিতৃমানবঃ । তৃপাস্তু পিতরঃ সর্বের মাতৃমাতামহাদয়ঃ । । অতিতকুলকোটিনাং
সপ্তদীপনিবাসিনাম । ময়া দন্তেন তোয়েন তৃপাস্তু ভুবনত্রয়ম্ ।

More recently Mahahrishi Mahesh Yogi propounded the hypothesis that if only five percent of the population of the world practice Transcendental Meditation the incidence of war and violence will be reduced. This was warmly received in the United Nation Organization. Indeed large numbers of Americans and westerners as a whole are following Mahahrishi's instructions quite faithfully hoping for a peaceful world.

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Neural Underpinnings Catatonic Symptoms

— Madhavan PM¹, Rajmohan V²

Catatonia originally described in 1874 by Kahlbaum in his monograph 'Die Katatonie oder das Spannungsirresein' (Catatonia or Tension Insanity) is a psychomotor syndrome with motor, affective and behavioral features, most commonly associated with affective disorder. Later theorists (Kraepelin and Bleuler) described catatonia as a motor phenomenon associated with schizophrenia. Catatonia is now considered a complex psychomotor syndrome with affective, behavioral and motor symptoms manifested across a wide range of neuropsychiatric conditions. Neuroimaging of catatonia suggests possible involvement of certain brain areas in catatonic syndromes. Multiple neurotransmitter systems may also be involved as revealed by the response shown by pharmacological agents in catatonia. However there is still much disagreement and deliberation regarding the nosologic status, the diagnostic criteria, the assessment methods, the treatment and the neural correlates of catatonia.

Catatonia

The estimated prevalence of catatonia among psychiatric patients across studies varies from 7% to 38%. The most common cause of catatonia being mood disorders rather than schizophrenia. Catatonia is seen in depression, mania, schizophrenia, delusional disorders, dissociative disorders, and post traumatic stress disorder. Catatonia also results from infectious, metabolic, neurologic, autoimmune diseases, toxic, and drug related causes and around 40% catatonia is due to such states. Catatonia originates acutely or insidiously and may even be life threatening. It appears as stupor or excitement neurotoxic syndromes in general medical and neurology hospital services. It is frequent in patients with mood disorders, appearing as mania and depressive illness in adult and pediatric psychiatry services. Patients have mutism, negativism, rigidity, posturing, catalepsy, mannerisms, stereotypy, waxy flexibility, ambitendency, echolalia and echopraxia.¹

Neuroanatomy

Neuropsychological Explorations

Neuropsychological studies shown impaired right parietal function in catatonics as evidenced by their poor performance in the visual-object-space and perception test (VOSP) compared. The parietal defect implies a disability to process spatial position of movements and termination of movements with resultant posturing. Orbitofrontal cortical dysfunction as evidenced by neuropsychological data suggests a failure of the ability to take emotionally

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guided intuitive decisions and an inability to perform online monitoring required for decision making.¹

Electrophysiological Evidence

Movement related cortical potentials (MRCP) shows that catatonics though have preserved ability of 'Plan/strategy', 'Initiation' and 'Execution', have delayed initiation of termination. Therefore they tend to carry their action on for a longer period than normal. The termination deficit may reflect a delay in the online monitoring of spatial position of movements by right parietal cortex.¹

Neuroimaging in catatonia

i) Structural imaging

Catatonics show significant cortical enlargement especially the left frontoparietal on structural imaging studies. Further imaging has shown prefrontal and parietal enlargement in catatonics with schizophrenia. Therefore evidence tends to show a fronto-parital pathology in catatonia.²

ii) Functional imaging

Regional cerebral blood flow (rCBF) studies in catatonia have shown right left asymmetry in basal ganglia, hypoperfusion of left medial temporal area, and hypoperfusion in the right parietal cortex. The right parietal hypoperfusion correlates significantly with impaired visuospatial ability and the motor and affective symptoms in catatonia. In addition to this, it is observed that isolated lesions of the right parietal cortex result in posturing.¹

There is evidence that behavioral and affective symptoms of catatonia correlates with reduced orbitofrontal activity and the motor symptoms correlate with motor/premotor activity. Imaging studies show deficits in the orbitofrontal activation during negative emotional states. There is neuroimaging evidence that the orbitofrontal connectivity with the motor/premotor cortex is abnormal in catatonics. The right orbitofrontal activation during negative emotional stimulation is altered and the activation is shifted to the anterior cingulate and medial prefrontal cortex. This shows a shift of emotional to cognitive control of motor system with a subsequent failure to control motor phenomenon resulting in catatonic motor manifestations.¹

Catatonic patients show a significant decrease in right lateral orbitofrontal (including ventrolateral prefrontal cortex) activation during working memory tasks. This deficit in working memory maybe related to a deficit in the online monitoring of spatial position of movements.³

Neurochemistry 1) GABA

The efficacy of lorazepam in allaying the symptoms of catatonia has increased the focus on GABA in catatonia. SPECT study of Iomazenil binding shows significantly lower binding of GABA-A in the right orbitofrontal cortex and right posterior parietal cortex.

There is also a significantly lesser GABA-A binding in the left sensorimotor cortex in catatonic patients.¹ Study of catatonia with functional magnetic resonance imaging (fMRI) and magnetoencephalogram (MEG) shows that prefrontal cortical activation/ deactivation pattern during negative emotion is modulated by GABA-A receptors.¹ It is also noted that lorazepam shortens the MRCP delay during 'initiation of termination' in catatonia. *idem* is another agent useful in catatonia and like lorazepam is GABA-Aa agonists. However it is reported that the agonism (stimulation) of GABA-Ab receptor (e.g. valproic acid) in the context of reduced GABA-Aa efficacy can cause catatonia, leading to the GABA-Aa versus GABA-Ab hypothesis of catatonia.⁴

2) Dopamine

The studies estimating vanillylmandelic acid (VMA) and homovanillic acid (HVA) in the urine samples of patients with periodic catatonia show an increase in the levels of dopamine metabolites, suggesting a dopaminergic excess in catatonia. Recent evidence also hints at an increase in dopamine particularly in those patients responding to lorazepam. These findings are however contradictory to the observations that dopamine blockers like antipsychotics induce catatonia. The consensus however is that catatonia is characterized by striatal dopaminergic blockade (hypodopaminergic state).⁵

3) Glutamate

It is hypothesized that the catatonia is characterized by a glutamatergic excess in the posterior parietal areas secondary to a reduction in 'GABA levels in the orbitofrontal area and supplementary motor area. The observations that catatonia improves with the NMDA glutamate antagonist amantidine points to the role of glutamate in catatonia. The therapeutic recovery however was delayed. Therefore it is speculated that the glutamatergic system is secondarily altered in catatonia and that primary alteration is in the GABA-A receptors. The role of NMDA glutamate antagonists is further supported by the efficacy of memantine in catatonia.⁶

4) Serotonin

The observation that atypical antipsychotics are associated with catatonia points to a serotonergic role in catatonia. It is proposed that a serotonergic dysequilibrium with an up-regulated 5HT1a receptor and a down-regulated 5HT2a receptor leads to catatonia.⁵

Pathophysiology of Symptoms

Affective symptoms

The affective symptomatology of catatonia possibly results from a defect in the balance between lateral and medial orbitofrontal cortex. The medial orbitofrontal cortex area via the amygdala especially its basal nucleus has rich limbic connections and is involved in the processing of emotions. A shift in function during negative emotions leads to greater activation of the medial orbitofrontal cortex with resultant affective features.¹

Mutism and Stupor

The neuroimaging studies have shown that the orbitofrontal connectivity with the motor/premotor cortex is abnormal in catatonics. The right orbitofrontal activation during negative emotional stimulation is altered and the activation is shifted to the anterior cingulate and medial prefrontal cortex. This shift in function especially to the anterior cingulate, probably results in a heightened activity of the affective division of the anterior cingulate. This then leads to an almost complete down-regulation of the motor portion of the anterior cingulate which may account for mutism. The shift of function to the medial prefrontal cortex (MPFC) with subsequent MPFC dysfunction may result in total inability of verbal and non verbal contact with others resulting in stupor.¹

Waxy flexibility and Rigidity

Striatal especially globus pallidus internum dysfunction leads to rigidity and waxy flexibility in catatonia. It is also speculated that the orbitofrontal connectivity to the motor cortex (horizontal or cortico-cortical regulation) may down-regulate the striatal D2 within the motor loop and result in hypertonus and rigidity.¹

Posturing

Posturing is hypothesized to be due to a failure in the termination of movements. It involves the interaction of areas involved in spatial attention and the 'initiation of termination' of movements. The right inferior parietal cortex dysfunction leads to impaired spatial attention and initiation of termination of movements resulting in posturing.¹

Perseverative behavior

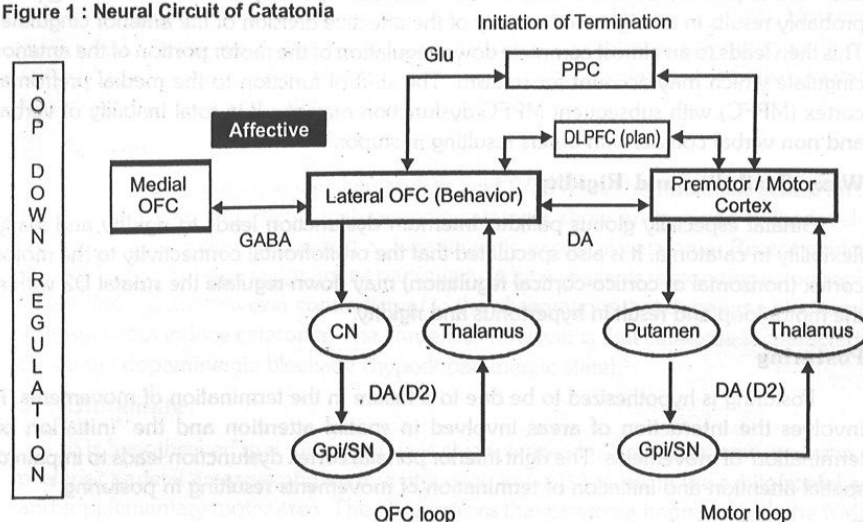
Catatonia has behavioral features like negativism, stereotypies, perseveration, echolalia and echopraxia. These are posited to be the result of a defect in the lateral part of the orbitofrontal cortex including the ventrolateral prefrontal (VLPFC) cortex, the dorsolateral prefrontal cortex (DLPFC) and the posterior parietal cortex (PPC). The lateral part of the orbitofrontal cortex including the VLPFC is related to control of complex behavior. DLPFC is involved in planning of complex and has reciprocal connection with PPC. Thus registration of spatial position is also needed to control and monitor complex behaviors. The VLPFC deficit leads to lack of suppression of behaviors once they have started leading to perseveration. The DLPFC defect leads to an inability to plan and an inability to plan ones behavior forces the person to take behavior from another either by negating them or imitating them.¹

Conclusion

The preliminary explorations into the neuroanatomy and neurochemistry of catatonia have that the OFC is probably the key structure mediating the symptoms of catatonia. The interaction of the OFC with the DLPFC and the premotor loop is postulated to cause the affective, behavioural and motor symptoms of catatonia. (Figure 1) The evidence regarding

the neurotransmitter abnormalities in catatonia comes from pharmacological studies and is inconclusive. Further exploration to elucidate the exact neuroanatomic and neurochemical abnormalities associated with catatonia will help in better conceptualization and treatment of this elusive entity.

Figure 1 : Neural Circuit of Catatonia



CN-Caudate nucleus, Gpi-Globus pallidus, SN-Substantia nigra, DA-Dopamine, Glu-Glutamate

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PARASOMNIAS IN OUR PRACTICE

— Amarnath Maillik

Sleep complaints are among the most frequent complaints voiced by our patients, but often only upon inquiry. Among the patients there remains the idea that sleep complaints may not be taken seriously or that nothing can be done to help. It is a common observation about patients with sleep complaints is that they frequently do not seek professional help for their sleep problem or do not remain the problem when they consult a clinician for other reasons. Studies have found the incidence of insomnia in primary care to be as high as 69%, but only one third of patients had discussed the problem with their physician

Parasomnias are a large category of sleep disorders that might be called the strange things. They are undesirable physical or sensory experiences that occur with sleep. These events often involve bizarre and unusual behaviors. Although some events are uncommon, 3% adults and 30% of children may have recurring nocturnal behaviors. Usually patients are unaware of their parasomnias, but other bed partners and in the case of children, parents are certainly aware of them. Parasomnias may occur throughout the patient's life span, although the type and presentation differ with age.

The parasomnias can be grouped into 2 major categories —

Primary sleep parasomnias :

- a) Non - REM sleep parasomnias
 - (1) Nocturnal walking
 - (2) Sleep terror or Night terror
 - (3) Sleep walking (somnambulism)
 - (4) Nocturnal sleep related eating disorder
 - (5) Sleep Bruxism
 - (6) Sleep related rhythmic movement disorder
 - (7) Sleep related Dissociative
 - (8) Sleep related muscle cramp.
- b) REM sleep Parasomnias
 - (1) REM sleep behavior disorder
 - (2) Night mares
 - (3) Sleep paralysis
 - (4) REM sleep related since arrest
 - (5) Sleep related impaired penile erections.
 - (6) Sleep related painful penile erector.

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Secondary Parasomnias —

- | | |
|---------------------------------|---|
| a) CNS parasomnias | (1) Vascular headaches |
| | (2) Exploding head syndrome |
| | (3) Hypnic headache syndrome |
| b) Cardio-pulmonary Parasomnias | (1) Respiratory Dyskinesias |
| | (2) Sudden unexplained nocturnal Death syndrome |
| c) Gastrointestinal Parasomnias | (1) Gastro oesophageal Reflux |
| | (2) Nocturnal Esophageal Spasm |
| | (3) Sleep related tonic spasm |
| | (4) Sleep related abnormal swallowing syndrome. |

In this article the common parasomnias which are seen in daily practice are discussed.

NIGHT MARE DISORDER :

Nightmares are long, frightening dreams from which people awaken scared. There may be repeated awakening from the major sleep period or naps with detailed recall of extremely frightening dreams usually involving threats to survival, security or self esteem. The awakening usually occur during the second half of the sleep period. It occurs in about 5% - 8% of the general adult population being more common in women. Most individuals who have night mares have no documented psychiatric illness. In some cases, nightmares may herald the onset of degenerative neurological disorder or psychosis. Several medications which have got action of neurotransmitter, acetylcholine and GABA have been reported to increase the incidence of nightmares. A variety of anaesthetics, antiepileptic drugs, may cause nightmare. Low serum lipid level and drugs which lowers lipid level such as Atrovastatin may induce nightmare.^{1,2} Traumatic events, Post Traumatic Stress Disorder are frequently followed by nightmares.

Sleep Terror Disorder :

Sleep terror (also called night terror) is disorder of arousal that occurs during the first 3 hours of sleep when stage III and stage (V) sleep predominate. These episodes begin with a loud cry and the onset of a prolonged period of apparent intense anxiety, with evidence of tachycardia, increased blood pressure, dilated pupils and sweating. Considerable motor activity may be present in adults, including jumping out of bed, running about, sometimes resulting in bodily injury.

Sleep terror occurs in up to 5% of adults.³

- Sleep terrors are more common in deep sleepers and in males and are typically not remembered the next morning. Persons with sleep terrors usually calm down quickly after the episode has run its course and may return to sleep. Sleep terrors are more common in children than in adults.

A variation of sleep terror is confusional arousal. It begins with movement and vocalization (often crying out) and often includes violent thrashing about in the bed. The patient may exhibit uncontrolled crying, bizzare talk, lack of appropriate response to stimulation. It is seen in early childhood and may occur in up to 4% of adults.

Night terror may reflect a minor neurological abnormality perhaps in the temporal lobe or underlying structures. But in a typical case of night terrors, however, no signs of temporal lobe epilepsy or other seizure disorders are seen either clinically or on EEG recordings.

SLEEPWALKING DISORDER :

Sleepwalking also known as somnambulism is a complex behavior that is initiated in the first third of the night during deep NREM (stage 3 and 4) sleep. There is repeated episodes of rising from bed during sleep and walking about, usually occurring during the first third of the major sleep episode. While sleepwalking the person has a blank, staring face is relatively unresponsive to efforts of others to communicate with him or her and can be awakened only with great difficulty. On awakening either from the sleepwalking episode or the next morning the person has amnesia for the episode usually there is no impairment of mental activity or behavior after awakening from the sleepwalking episode but some time a short period of confusion or disorientation is seen. Sleepwalking like other parasomnias causes clinically significant distress or impairment in social, occupational areas of functioning.

Sleepwalking usually begins between ages 4 and 8 with peak prevalence at approximately 12 years of age. The disorder is more common in boys than in girls and 15% of children have an occasional episode. It tends to run in families and a minor neurological abnormality probably underlines the condition. The episode is not fully psychogenic although stressful events are usually associated with sleepwalking. Extreme tiredness, exhaustion or previous sleep deprivation exacerbates attacks of sleepwalking.

Sleepwalking and sleep terror episodes can be dangerous. Approximately three quarters of all patients with sleepwalking and sleep terror disorders have reported actual injuries or the potential for injuries during their episodes.⁴

Although sleepwalking and sleep terror are different episodes, often they can occur together in the same arousal episode.

Clinical approach to Parasomnias :

The proper evaluation of a patient with a sleep disorder requires the ability to establish and appropriate clinical suspicion on the basis of the history and physical examination.

It is important that patients are always be asked during the initial evaluation whether they are satisfied with their sleep. This can be done in about a minute with the following questions —

- 1) Are you satisfied with your sleep?
(This questions will enable physician to pick up most insomnia complaints.)
- 2) Are you excessively sleepy during the day? (This pick up most of the excessive day time sleepiness (EDS) problems such as Narcolepsy and Obstructive apnea).
- 3) Do others (your bed partners) complain about your sleep? (Helps to know most of the parasomnia disorders.)

A positive answer to any of these questions should be reason to consider obtaining more detailed sleep history. Sleep and sleep quality are affected by many variables, a complete physical, neurological and mental status assessment are essential. Psychological assessment is important to note depression, delusional thoughts, cognitive impairment etc. Several laboratory procedures are available to assist in the diagnosis of sleep complaints. These include obtaining all-night sleep recordings with polysomnography (PSG) and multiple sleep. Latency Test (MSLT) to quantify daytime sleepiness. Truly not all patients with sleep disorders require a sleep specialist consultation or sleep laboratory testing.

Few points to note during diagnosis of parasomnias —

- 1) Parasomnias can occur in both NREM and REM sleep, it is important to determine which type is present, because treatment differs.
- 2) Stereotypical, repetitive events and unusual motor behavior during sleep may be due to a seizure disorder.
- 3) The most common REM sleep parasomnia, REM sleep behavior disorders can be the first symptom of a dementing illness that develops later.
- 4) Temporal lobe seizures are to be ruled out when evaluating repetitive nocturnal motor behavioral symptoms. An EEG may be helpful.
- 5) A toxic reaction to drugs, brain injury and dementia with episodic nocturnal wandering is to be differentiated from sleepwalking and sleep terror. Sleep drunkenness is a state of partial awakening that results when an individual is extremely fatigued or affected by sedatives or alcohol. This state may also be seen as a part of narcolepsy syndrome.
- 6) Automatic behavior and dissociative states are usually found in adults with psychiatric disturbances. These states may occur during waking and sleep states. Behaviors in these conditions are much more complex than is seen in a typical sleep walking or sleep terror episode.

- 7) Sleep related bruxism is a rhythmic repetitive sleep movement. Grinding or clenching of the teeth during sleep produces bizarre sounds. Patient may vocalize during the episode. Abnormal wear of the teeth, jaw pain, headache, facial pain or tooth pain may be observed.
- 8) Periodic limb movements of sleep and restless legs syndromes are repetitive stereotyped movements of the lower limbs that occur during sleep consisting of extension of the great toe with dorsiflexion of the ankle and flexion of the knee and hip. Patients are unaware of the movements. Restless legs syndrome involves deep aching sensations in the legs or arms, improved by movement of extremities.

TREATMENT ISSUES :

Outlines of management of Parasomnias are as follows.

- 1) Sleepwalking and sleep terrors in adults, especially if related to earlier or concurrent stress, are frequently responsive to psychotherapeutic intervention, Progressive relaxation or hypnosis. For those who have potentially dangerous sleepwalking or sleep terror episodes, medication is not suitable treatment to consider in addition to psychotherapy.

Following drugs may be used —

- Inipramine 25 mg to 100mg, sertraline 25 mg to 50mg, Paroxetine 10mg to 20mg, Carbamazepine, Diazepam 5mg - 10mg, Divalproex etc. Most patients respond to low doses of these drugs, patients with evidence of depression require full antidepressant dosage.
- 2) One must be careful for elderly patients with new onset sleepwalking which may be due to side effects of medicines they are taking.
- 3) Sleep talking is seldom of any medical concern on its own and generally does not respond to treatment with drugs or psychotherapy.
- 4) Treatment for Bruxism consists primarily of an appropriate dental device to prevent the patient from damaging and wearing down the teeth.
- 5) Sleep paralysis patients are generally treated with reassurance, supportive psychotherapy and if needed with drugs like imipramine 10 - 50mg at bed time or with SSRI.
- 6) Nightmare episodes need reassurance, psychotherapy and if necessary drugs. Case report suggests that antiepileptic drug, topiramate may be used successfully to the patients of PTSD with Nightmare episodes.⁶
- 7) Clinician has to warn the patients and relatives about the factors exacerbating disorders

of arousal such as sleep terrors, sleep walking and confusional arousal. The factors are —

Environrmental disturbances (noise, light, bed partner, children, pets)

Sleeping in unfamiliar surroundings

Sleep deprivation

Obstructive sleep aproea

Periodic limb movement disorder

Headache

Gastrooesophageal reflux diseass.

Hyperthyroidism

Alcohol

CNS depressant medicines

Stress.

Treatment goals of Panasomnia should focus on patient and family safety, reduction of risk harm, and improvement of lifestyle. Many of these disorders have diverse treatment options. Patient should be vested in the treatment plan and understand like risks and benefits of the various therapies.

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ROLE OF CT AND MR IMAGING IN NON-NEOPLASTIC AETIOLOGIES OF SEIZURE DISORDER IN 6-18 YR. AGE GROUP.

— A.SASMAL,¹ T. DHIBAR,² N. K. BERA,³ S. PRADHAN⁴

INTRODUCTION

Seizure (from Latin word 'sacire'-'to take possession of')¹ is most easily defined in physiological terms as occasional, sudden, excessive, rapid and local discharge from gray matter (Jackson 1873)

Epileptic seizure can be defined as an intermittent and stereotyped disturbances of consciousness, behaviour, emotion, motor function, or sensation that on clinical grounds is believed to be result from cortical neural discharge^{2, 6}.

Epilepsy can be defined as two or more unprovoked seizure occur at interval of 24 hrs. or more apart.

Over the past decade the role of neuro-imaging in the diagnosis and management of epilepsy has been changed considerably. MRI is more sensitive in diagnosing in most of the cerebral pathologies associated with chronic epilepsies with an exception of calcification which is better seen in CT

CLASSIFICATION^{2,6}

Classification of seizure (commission on classification and terminology of the International League Against Epilepsy) 1981

PARTIAL SEIZURE (Semue beginning locally)

Simple (consciousness not impaired)

- with motor symptoms
- with somatosensory or special sensory symptoms
- with autonomic symptoms
- with psychic symptoms

Complex (With impairment of consciousness)

- beginning as a simple partial seizure (progressing to complex seizure) impairment of consciousness at onset
- impairment of consciousness only
- with automatism

Partial seizure becoming secondarily generalized.

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GENERALISED SEIZURE

- absence seizure
- simple (petit mal)
- complex
- myoclonic seizure
- clonic seizure -tonic seizure
- tonic-clonic seizure
- atonic seizure

EPIDEMIOLOGY

Although variety of factors influence the incidence and prevalence of seizure, 5-10% of the population will have at least one episode of seizure in their lifetime with the highest incidence occurring in childhood and early adulthood. Using the definition of epilepsy as two or more unprovoked seizure the incidence of epilepsy is 0.3-0.5% in different population throughout the world, where the prevalence of active epilepsy is in the range of 4-10/1000²

Incidence and prevalence is higher in the 3rd world countries than the developed world, with higher rates found in the rural opposed to urban communities, with slightly higher prevalence in men than women and 2/3rd being partial epilepsies and approximately 1/3rd generalised²

Neoplasia overall contribute 4% cases of seizure disorder rests are of non -neoplastic origin,^{5,11} so majority of seizure disorder in 6-18 yr. age group is non-neoplastic.^{5,11}

Seizure in later childhood and adolescence represents most common epileptic problem in general practice.

EPILEPTOGENESIS²

FOCA EPILEPSIES The most studied and clinically relevant model of focal seizure and epileptogenesis is the hippocampus and hippocampal sclerosis, the most common form of focal epilepsies in man. Mesial temporal structures are interconnected by a reverberating loops, involving entorhinal cortex, dentate gyrus, CA3, CA1 neurones. Normal spontaneous activity of CA3 pyramidal neurons consists of paroxysmal depolarization shifts and associated burst firing of cell body and apical dendrites (Wong & Prince - 1979, 1981). In normal brain material bursting activity in CA3 neurones has about a 30% chance of evoking burst in connected neurons. This probability increases when pyramidal cells are excited by many inputs simultaneously.

Three phenomenon-cell loss, mossy fibre sprouting and neurogenesis may all enhance potential for seizure generation and the corresponding pathological change is hippocampal sclerosis. It appears that limbic system are particularly sensitive to development of kindling, when compared to neocortex, a situation that is reflected in man. Temporal lobe is by far the most common site of seizure onset.

GENERALISED EPILEPSY

The cellular substrate for generalized epilepsies is now well understood. It is dependent on a thalamo-cortical circuit that includes the nucleus reticularis thalami. The circuits involve excitatory glutaminergic synapses and inhibitory gabergic synapses. The behavior of thalamic neurons and the circuit largely determined by the presence of high density of calcium T channels, which results in Ca²⁺/K⁺ dependent burst firing. These in turn can give rise to strong inhibitory post synaptic potential mediated by GABAB receptors in the thalamo-cortical relay neurons.

PATHOLOGY⁷

The spectrum of pathological substrates associated with seizure is as follows

1. DEVELOPMENTAL DISORDERS

- Neuronal migration disorder (Tuberous sclerosis, focal cortical Dysplasias, polymicrogyria, schizencephaly, heterotopias, lissencephaly, hemimegalencephaly, microdysgenesis)
- Hamartomas
- Vascular malformation

2. NEOPLASM

- Gliomas
- Mixed neurological tumours
- Dysembryoplastic neuroepithelial tumours
- Others

3. MESIAL TEMPORAL SCLEROSIS

4. BRAIN INJURY

- Infection (bacterial, fungal, viral, parasitic disease)
- Immune mediated disorder (Rasmussen's syndrome)
- Cerebrovascular disease including stroke, porencephaly, ulegyria)
- Trauma

ROLE OF CT AND MRI

Certain type of seizure disorder likely to be associated with structural brain lesion including tumor, infection, infarction, traumatic brain injury, vascular malformation, developmental anomalies and seizure associated brain pathology.

Neuroimaging is one of the most important advances made in the past decade in the management of seizure disorder. Neuroimaging has increased substantially the ability to plan medical, surgical therapy and to prognosticate the outcome of disorder and therapy.

Neuroimaging helps to determine whether seizure was provoked or unprovoked⁴.

Distinction between two type of epilepsy is a major determinant in selecting the anti-epileptic medication to treat the disorder. Determining whether focal lesion is the cause of seizure also helps in diagnosis of epilepsy syndrome of childhood.

Neuroimaging also useful to determine whether early intervention is required or not as in cases of traumatic seizure and vascular aetiologies of seizure.

Mesial temporal sclerosis, low grade neoplasias, vascular lesion particularly cavernomas and developmental abnormalities are all likely to be missed by CT. MRI is more sensitive than CT for early detection of causative lesion.

Almost all patient with new onset seizure should have a brain imaging study to determine whether there is a structural abnormality which is responsible. The use of newer MRI method such as fluid attenuated inversion recovery (FLAIR) has increased the sensitivity for detection of abnormal cortical echotexture including hippocampal atrophy as well as abnormalities of cortical migration.

MRI

Although the imaging evaluation of epilepsy greatly advanced by the clinical introduction of CT in the early 1970 because of MRFS superior soft tissue contrast, multiplanar imaging capability, lack of beam hardening artifact, and lack of radiation exposure virtually all the substrate of epilepsy visualized with greater sensitivity and accuracy by MRI. As a consequence MRI has become the modality of choice for imaging a patient with seizure disorder.^{7,8,9,11}

MRI is indicated in all patient with epilepsy who appear refractory to pharmacological treatment irrespective of previous imaging study

MRI plays several important role in the clinical management of patient with epilepsy, most important being to localize and identify the structural substrate of partial seizure.

PROPOSED MR SCAN PROTOCOL

Imaging plane	Conitionpo	Comments
Sagital	T ₁ weighted	Anatomical changes including sellar and suprasellar region
Sagital	T ₂ weighted	Parenchymal lesion including sellar and suprasellar region
Axial	Gradient Recalled Echo (GRE)	1. Useful in presence of calcification and haemorrhage in brain 2. To identify vascular malformation 3. To identify bony fracture
Axial	T ₂ weighted FSE OR TSE	Parenchymal lesion
Axial	T Weighted	Anatomical evaluation
Axial	Diffusion Weighted Imaging (DWI)	Evaluation of stroke
	Magnetic Resonance spectroscopy (MRS)	1. To differentiate between neoplastic and non-neoplastic aetiologies 2. To evaluate temporal lobe epilepsy
Coronal	Fluid Attenuated Inversion Recovery (FLAIR)	Sequence of choice for small cortical lesion and temporal lobe lesion
Coronal	T ₂ Weighted	To identify cortical lesion and temporal lobe lesion

Although seizure are usually classified on the basis of clinical and EEG finding precise classification may be at times uncertain. At times secondary generalized partial onset seizure may be misinterpreted as primary generalized, the identification of epileptogenic foci on neuroimaging should clarify seizure classification as focal onset.

As the classification of seizure shifts from electroclinical phenomenon towards a emphasis on the structural substrate that produce the abnormalities, the role of MRI become more central in conceptually defining the epilepsy. MRI is an essential tool in surgical planning and used to define volume of tissue targeted for resection, to identify the surgical approach and asses the relationship of the lesion to functionally eloquent areas of brain.

The sensitivity of MRI technique to the structural substrate responsible for epilepsy based on recent imaging literature is estimated as 90-100% for neuronal migration disorder, 100% for vascular malformation, 90-95% for mesial temporal sclerosis, 80-90% for brain injury due to trauma, infection and infarction and 100% for neoplastic aetiologies.^{7,10}

Proton MRS has a major clinical application in the localization of subtle epileptogenic foci that are not evident on conventional MRI sequence. It also plays a pivotal role in the planning and prognostication of epilepsy surgery. Although in classical cases of mesial temporal sclerosis the involved hippocampus is easily identified by a volume loss and signal hyperintensity on T2 weighted coronal FLAIR sequence, 20% patient with temporal lobe epilepsy has normal MRI scan, and the finding in children are generally more subtle than those of adult. Even when seizure onset and structural abnormalities are clearly unilateral, MRS has shown that bilateral temporal lobe abnormalities are found in approximately 40-50% of temporal lobe epilepsy. High resolution MRI detects surgically relevant lesion upto 80% people undergoing temporal lobectomy and 60% of those undergoing frontal lobe surgery.¹¹

FUNCTIONAL MRI (fMRI)⁷

* MAGNETIC RESONANCE SPECTROSCOPY

N-acetyl aspartate (NAA), a functional neuronal marker, is a metabolite primarily distributed in neurons. Decrease in NAA has been demonstrated in a variety of neuropathological disorder associated with neuronal loss. Proton MRS has shown 15-37% decrease in NAA in 80-100% patient of temporal lobe epilepsy. Post-ictal MRS Studies have demonstrated the localized presence of lactate after seizure in patient with temporal lobe epilepsy.

* PERFUSION MR

Cerebral blood volume assessment can be made during the first pass of injected contrast agents such as gadolinium chelate through the brain. Perfusion MR has demonstrated regional hyperperfusion concordant with SPECT, EEG and neuronal finding in status epilepticus.

* DIFFUSION MR (DWMR)

Diffusion weighted magnetic resonance employs a novel MR scanning sequence that sensitizes to random motion of water molecule in tissues. A disturbance in the motion of water molecules produces an area of increased signal on MR images. DWMR has been shown to be more sensitive than conventional imaging sequence in ischaemic changes of brain.

* ACTIVATION MR

Activation MR technique takes advantage of blood oxygen level dependent (BOLD) contrast effect that accompanies cerebral activity to localize areas of functional activation during the performance of physical or mental task. There is an increase in cerebral blood flow without a commensurate increase in oxygen consumption rate. The transient net effect of these changes is relatively less deoxy haemoglobin within the blood capillaries and venules draining that portion of section of brain. Region with relatively less paramagnetic deoxy haemoglobin will demonstrate increased signal on T2 weighted MR images. Utilization of this technique is to localize eloquent cortex as a pre-operative assessment in patient with focal brain lesion.

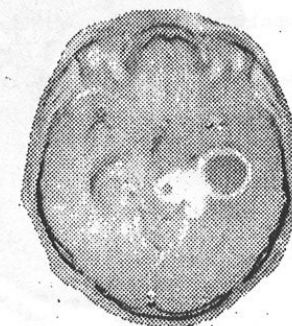
CT SCAN

It is generally initial brain imaging procedure in neurological emergencies. It is better compared to MRI when artifacts associated with critically ill patients such as catheter, ventilation devices produce interference on MRI.¹¹

CT scan is valuable for early detection of blood densities as in subarachnoid haemorrhage or haemorrhagic stroke lesion. Also CT is more sensitive in calcified lesion which appears as signal void on MRI. In patient with suspected CNS infection CT will be performed emergently when MRI not readily available. It is usually appropriate to obtain a MRI study within a few days for better evaluation. CT is readily available compared to MRI in a developing country like us which made CT scan as an initial neuro imaging procedure in developing countries like us.

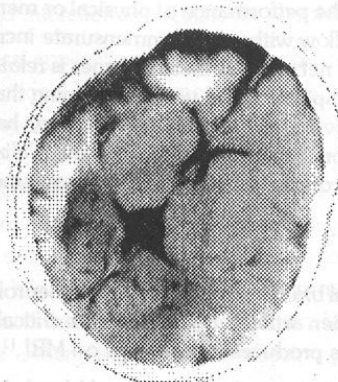
It is an alternative to MRI for patient who cannot undergo MRI because of severe claustrophobia, or ferromagnetic object in eye, head, neck.

SOME COMMONLY DIAGNOSED AETIOLOGIES OF SEIZURE DISORDER IN THIS AGE GROUP IN CT & MRIMAGING



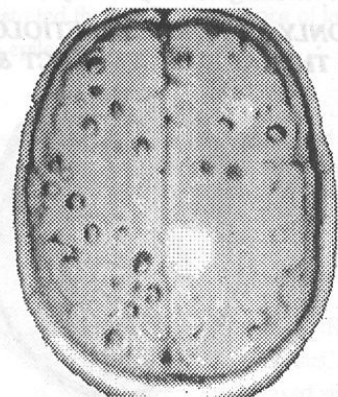
TUBERCULOMA PRESENTING AS A SINGLE RING ENHANCING LESION

PLAIN AXIAL CT SCAN OF BRAIN IN A 3YR OLD PATIENT PRESENTING WITH LT SIDED FACIAL NAEVUS WITH CONVULSION



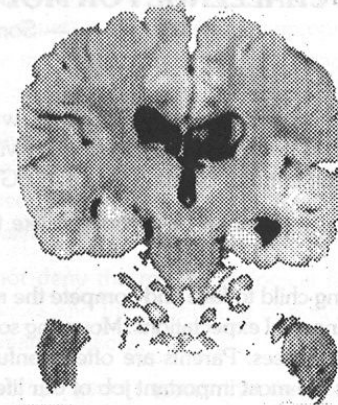
A CASE OF STURGE WEBER SYNDROME

AXIAL FLAIR SEQUENCE OF A 17YR OLD MALE STUDENT PRESENTING WITH FOCAL SEIZURE



A CASE NEUROCYSTICERCOSIS

CORONAL FLAIR SEQUENCE OF A 16 <\$R MALE PATIENT PRESENTING WITH FOCAL SEIZURE



BILATERAL TEMPORAL LOBE SCLEROSIS

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PARENTING A CHALLENGE FOR MODERN TIMES

— Somsubhra Chattopadhyay¹,
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'The children of the next generation will be spendthrifts, will not think much and will not know much about life, We do not know what their future will be. only the sun god Ra save them' an inscription on the tomb of oldest pyramid in EGYPT about 5000 BC.

Each successive generation of parent have thought like this and tried to bring up children to their ideals.

Parenting means preparing child to face and compete the reality of the world to meet the social, occupational and financial expectations. Mounting social and financial pressure have added to the parenting stresses. Parents are often confused, guilt ridden with no professional help. Though it is the most important job of our life we are ill equipped.

Each day, more than three quarters of a million adults around the world experience the joy and heartache, and, the challenge and reward of becoming new parents. Despite the fact that most people become parents and everyone who ever lived has had parents, parenting remains a somewhat mystifying subject about which almost everyone has opinion but about which few people agree.

Parenting is a complex activity that includes many specific behaviors that work individually or together to influence child's future outcomes. It prepares the child to enter into the next phase of life. When we compare the conventional role of a to-day's child with that of yesterday, i.e. two decades back, we find a sea difference. Two decades ago children were reared in a relatively protected environment and taught to be subservient to their elders. The male child was expected to acquire modest education and make a career or else join the family profession. The female child was taught to be submissive, tolerant and compliant; to acquire only minimal education, but to be proficient in domestic work (cooking, tailoring, home keeping etc.) so that she could adopt the primary role of a home maker. In contrast, today's child is gripped with multitude of stresses. He/She is expected to excel in his academic pursuits, face stiff competition to get into one of the elite professional courses and make a career. Mounting parental pressure and economic constraints, nuclearization of families, rising number of working mothers and women's equal rights movement, have added to the problems.

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In some of the developed countries like Hong Kong this has resulted in crisis in child rearing with increase in unwanted children, avoidance and refusal to rear the children, confusion and inappropriateness in child rearing; inappropriate discipline; overprotection and over control; change in sex roles and attitudes, preoccupation with education and endless pressure on scholastic achievement.¹

A WHO sponsored multi-centred study in 4 developing countries reported a prevalence of suicide in 12-29% children.² There is a peak rise in suicide among males in our country in late adolescence. The causes are largely 'social', with only 3.4% attributed to mental illnesses. The prevalence rates of drug abuse in students have varied from 5-56.5%.³

Although one cannot deny the role of biological factors, one must accept the contribution of social stresses and the importance of positive parenting, especially with regard to non-psychotic manifestations.

Research over the past 20 years suggests that the quality of the parent-adolescent relationship significantly affects the development of risk behaviors in adolescent health. The review supports the substantial influence of parenting style on adolescent development. Adolescents raised in authoritative households consistently demonstrate higher protective and fewer risk behaviors than adolescents from non-authoritative families. There is also considerable evidence to show that parenting styles and behaviors related to warmth, communication and disciplinary practices predict important mediators, including academic achievement and psychosocial adjustment. Careful examination of parenting style patterns in diverse populations, particularly with respect to physical activity and unintentional injury, will be a critical next step in the development of efficacious, culturally tailored adolescent health promotion interventions.⁴

The health of future generations, both physical and psychological, depends upon parental and early environmental influences. Education about these negative influences is urgent, especially to encourage childbearing women to adopt healthy diet and lifestyle. The detrimental effects of cigarette smoking in pregnancy have been known since the 1970s, yet in Germany, for example, 60% of all children are conceived, carried, born into a household where at least one adult smokes. Alcohol early in the pregnancy—just when many mothers are unaware they are pregnant can produce significant physical malformation, especially in the face. Prenatal exposure to alcohol has significant effects on the intelligence and behavior of the child. Many of these children are very restless. Even slight amounts of poisoning during the pregnancy are related to the development of a negative self-image and the compensatory behavior of the Narcissistic Personality Disorder in later life. Undue stress of any kind during the pregnancy leads to problems for the developing child.⁵

Yap MBH et al (2008) through their radiological volumetric estimation of brain parameters like amygdala, hippocampus and cingulate cortex proved significant correlation between adverse parenting, brain structural abnormality and adolescent depressive symptom.⁶

Abidin R (1995) devised a model of parenting stress. It has three factors, like Child factors (age, social skill, problem behavior), Parent factors (gender, health, race, ethnicity), Contextual factors (family structure, conflict, social support, education, income).⁷

Parenting stress associated with negative parenting practices, leads to increased youth health risk behaviors. Parents of adolescents experience a high level of parenting stress that can compromise their ability to parent effectively.⁸

Parenting self-efficacy has been identified as one determinant of positive parenting. Birth perception is a correlate of parenting self-efficacy that is modifiable; therefore, nurses have an opportunity to strive to create a positive birth experience for all women to enhance their early parenting self-efficacy.⁹

Parents play a significant role in the sexual development and behaviors of their children. Parental monitoring and supervision are important avenues for keeping adolescents from risky situations and activities while the teen develops decision-making skills.¹⁰

Early puberty is a risk factor for delinquency, and early puberty combined with low parental nurturance, communication, or parental knowledge of the child's activities presents a risk for aggressive behavior in early adolescent girls. Early-maturing girls may benefit from increased parental nurturance, communication, and knowledge.¹¹

Human behavior is far away from perfection. Parents can have their whims altered and change their reaction to situations. There are a few pitfalls that can be avoided e.g, fighting with spouse, reckless spending, lifestyle, commenting, comparison, over protection, excess freedom, disrespect, swearing, physical abuse, verbal abuse, complaining to others about own children, worrying too much about children, criticizing, forcing children to work according to parent's choices, Condemning, mis-understanding, mis-communicating, mis-interpreting, mis-representing their thoughts/ words/ deeds.

Physical, sexual and psychological violence and neglect of children falls under the category of child abuse. Child abuse can occur anywhere - at home, in school, in temple, in church, in the streets, or any other place. Child abuse is found to be more rampant in broken families, where the parents are living separately according to a report generated by UNICEF. For this reason United States and United Kingdom are found to rank very low in terms of children's well being and mental health.

Parents often become less involved in the lives of their children as they enter the middle grades. But young adolescent needs as much attention and love from their parents as they needed in their childhood. A good relationship with parents best safeguard the child as he grows and explores.

Some aspects of positive parenting are:

Showing love. Young adolescents need adults who are there for them—people who connect with them, communicate with them, spend time with them and show a genuine interest in them. This is how they learn to care for and love others.

Providing support. Young adolescents need support as they struggle with problems that may seem unimportant to their parents and families. They need praise when they've done their best. They need encouragement to develop interests and personal characteristics.

Setting limits. Young adolescents need parents or other adults who consistently provide structure and supervision that is firm and appropriate for age and development. Limits keep all children, including young teens, physically and emotionally safe. Psychologist Diana Baumrind identifies three types of parents: **authoritarian, permissive and authoritative**. By studying about findings from more than 20 years of research, she and her colleagues have found that to be effective parents, it's best to avoid extremes. **Authoritarian** parents who lay down hard-and-fast rules and expect their children to always do as they are told or **permissive** parents who have very few rules or regulations and give their children too much freedom are most likely to have the most difficult time as parents. Their children are at risk for a range of negative behavioral and emotional consequences. However, **authoritative** parents, who set limits that are clear and come with explanations, tend to struggle less with their adolescents (Baumrind D, 1966).

Being a role model. Young adolescents need strong role models. Your actions speak louder than words. As adolescents explore possibilities of who they may become, they look to their parents, peers, well-known personalities and others to define who they may become.

Teaching responsibility. We are not born knowing how to act responsibly. A sense of responsibility is formed over time. As children grow up, they need to learn to take more and more responsibility for such things as:

- completing chores, such as doing yard work, cleaning their rooms or helping to prepare meals, that contribute to the family's well being
- completing homework assignments without being nagged
- taking on community activities
- finding ways to be useful to others
- admitting to both the good and bad choices that they make.

Providing a range of experiences. Adolescence is a time for exploring many areas and doing new things. The child may try new sports and new academic pursuits and read new books. He may experiment with different forms of art, learn about different cultures and careers and take part in community or religious activities. It is better to open doors for the child. Parents should introduce him to new people and to new world. In doing so, parents may renew in themselves long-ignored interests and talents, which also can set a good example for their child.

Showing respect. It is tempting to label all young adolescents as being difficult and rebellious. But these youngsters vary as much as do children in any other age group. The child needs to be treated with respect, which requires the parents to recognize and appreciate their differences and to treat them as individuals.

Mental Health professionals cannot afford to be complacent at this point of time. While assessing children, special attention should be paid to coping behaviors and pattern of parenting. It would be interesting to study the parenting styles of parents, whose wards have been high achievers. They could serve as models for other parents. Also, identification of parents/children who find themselves difficult to cope with demands on parenting is needed, so that they could be provided professional help geared towards sharpening their parental skills. In parenting, as in other endeavors, nothing works all the time. It is safe to say however, that authoritative parenting works better than most of the parenting styles in facilitating the development of social competence in children at home and in the peer group. High level of nurturance combined with moderate levels of control help adults become responsible child rearing agents for their children and help children become mature competent members of society. With a little bit of luck, the children of authoritative parents should enjoy more than their share of success in the peer group.

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HIGH PREVALENCE OF CARDIAC AUTONOMIC DYSFUNCTION IN WOMEN WITH PRIOR GESTATIONAL DIABETES MELLITUS

— Sankha Jyoti Saha

Cardiac autonomic neuropathy is a common dysfunction in manifest diabetes mellitus and is associated with the duration of diabetes and/or inadequate glycaemic control. Heart rate variability (HRV) reflects autonomic heart dysfunction.

The overall prevalence of autonomic neuropathies is about 10% in the general diabetic population; autonomic neuropathies begin 10yrs after the onset of diabetes mellitus and slowly progress. Autonomic function tests are helpful when the history and examination finding are inconclusive, to detect subclinical involvement, or to follow the course of an autonomic disorder.

AUTONOMIC TESTING

- 1) Heart rate variation with deep breathing-It is a test of parasympathetic function on cardiovascular reflexes, via the vagus nerve.

6 deep breathes/minute and a forced vital capacity (FVC) > 1.5L are optimal. The lower limit of normal heart rate variation with deep breathing in persons < 20yrs is > 15-20 beats/min, but for persons over ages 60yrs is 5-8 beats/min.

- 2) Valsalva Response-It assesses the integrity of the Baroreflex Control of heart rate (Parasympathetic) and BP (Adrenergic). The response is obtained with the subject supine. A constant expiratory pressure of 40mm Hg is maintained for 15 seconds while measuring changes in HR and beat-to-beat BP. There are 4 phases of BP and HR response to the Valsalva maneuver. The divided VALSALVA RATIO is defined as the maximum phase II tachycardia divided by the minimum phase IV bradycardia. The ratio reflects Cardiovascular Function.

- 3) Sudomotor function-Sweating is induced by release of acetylcholine from sympathetic postganglionic fibres. The quantitative sudomotor axon reflex test (QSART) is a measure of regional autonomic function mediated by acetylcholine induced sweating. A reduced/absent response indicates a lesion of postganglionic sudomotor axon.

The thermoregulatory sweat test (TST) is a qualitative measure of regional sweat production in response to a elevation of body temperature combining QSART and test results will determine the site of the lesion.

Postganglionic lesion:-Both QSART and TST show absent sweating.

Preganglionic lesion:-QSART is intact but TSART shows anhidrosis.

- 4) Orthostatic BP recording-Beat-to-beat BP measurements determined in supine, 70% tilt and tilt back positions are useful to quantitative orthostatic failure of BP control.

MD (Med.), Malda

It is important to allow a 20-min period of supine test before assessing changes in BP during tilting.

- 5) Tilt table testing for syncope-It is used to diagnose vasovagal syncope. Beat-to-beat BP response and HR response are used in BP during tilting.
- 6) Pharmacologic tests-These help localize an autonomic defect to the CNS or PNS. Norepinephrine, Tyramine, Phenylephrine are some of the agents used.

The purpose of the present study was to investigate whether in women with prior Gestational Diabetes; alteration of cardiac autonomic function could be observed after delivery in relation to insulin sensitivity and glycaemic control.

MATERIALS AND METHODS

38 healthy women with prior Gestational Diabetes Mellitus were consecutively admitted to the study. The study was undertaken in Malda nursing home, Malda, West Bengal.

Autonomic function was correlated with heart rate variability (HRV) with deep breathing. HRV was analysed by both time, as well as frequency; domain methods using 24hr halter monitoring.

In addition, 18 women with normal glucose tolerance during and after pregnancy were investigated as control subjects. All women underwent a frequently sampled intravenous glucose test for measurement of insulin sensitivity.

OBSERVATION

Time domain analysis (standard deviation of normal RR intervals) showed a reduced HRV in 28 out of the 38 women with prior Gestational Diabetes Mellitus = 74%. In this 28 subjects, frequency analysis revealed impairment of both sympathetic as well as parasympathetic components. However, a relative predominance of the sympathetic over parasympathetic cardiac function was observed. The impairment of cardiac autonomic function was correlated with HbA1c values and the 2-hr blood glucose concentration (oral glucose tolerance test) but not with insulin sensitivity.

ANALYSIS AND INTERPRETATIONS

From this study, it was demonstrated that in 74% of the women who had prior Gestational Diabetes Mellitus, an impairment of cardiac sympathetic as well as parasympathetic function was present, which related to glycaemic control, but not to insulin sensitivity.

It may be inferred that functional autonomic changes could be an early prognostic indicator in pre-type 2 Diabetes Mellitus; as in pregnant Gestational Diabetic women (shown in this study).

So, cardiac autonomic changes/dysfunctions Pre-Diabetics/Gestational Diabetes Mellitus would provide the clinician a guide for future management of the Diabetes menace.

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20-21 March 2010, Hotel Sea Bird International, Falta, 24 Pgs (S)

Organising Chairman: Dr. Debasis Bhattacharya

Organising Secretary: Dr. Arabinda Brahma

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23-25 April 2010 Guwahati, Assam

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VII Int. Symposium about Current Issues and Controversies in Psychiatry, Barcelona (Spain), April 15 and 16, 2010

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CINP Congress 2010, Hong Kong, 6-10 June 2010

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www.cinp2010.com for regular updates.

WPA International Congress, Beijing, China, from 1 to 5 September 2010

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Email: tokasha@internetegypt.com

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WPA website: www.wpanet.org

Addictions 2010, October 28-31, 2010, Sheraton National, Arlington, VA, USA

www.addictions-conference.elsevier.com

International Seminar on Mind, Brain and Consciousness

14th, 15th January 2010, Thane, India

13th European Symposium on Suicide and Suicidal Behaviour

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Place Rome, Italy

WPA Regional Meeting

21-23 January, 2010, Dhaka, Bangladesh.

The last date of early bird registration is 30.11.2009

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1-4 February 2010

Dubai, UAE

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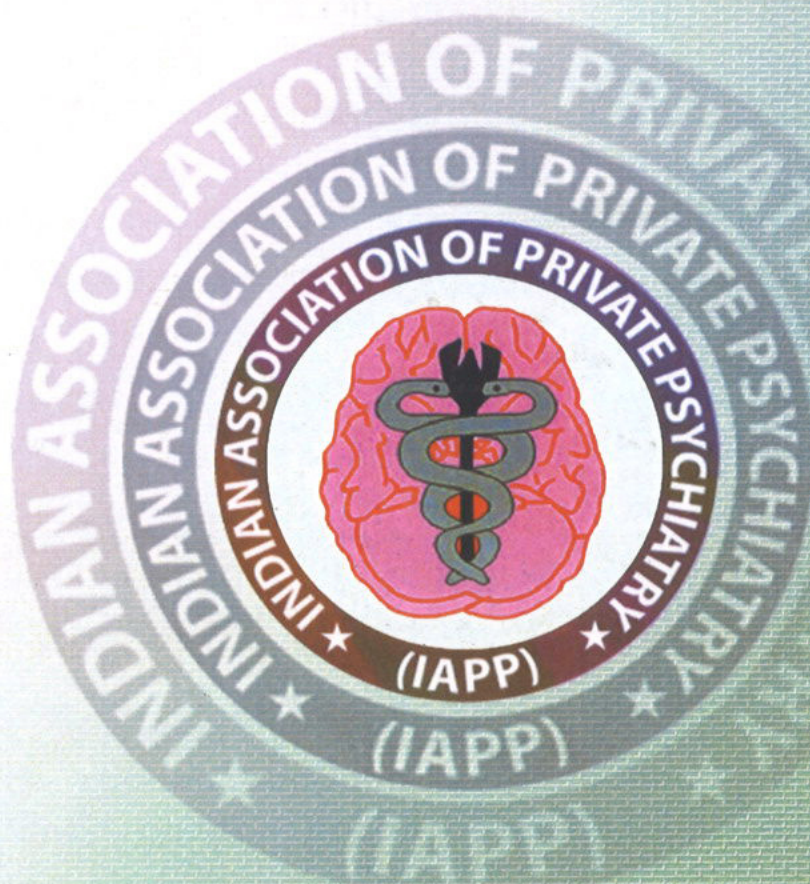
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