ECT IN NEUROLOGICAL CONDITIONS

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ABSTRACT

It is a myth that electroconvulsive therapy (ECT) produces greater side effects and worsens the neurological condition when used in neurologically ill patients. With the advancement and sophistication in ECT practice standards and modification procedures, it can be safely administered either to treat selected neurological conditions or the co-morbid psychiatric illnesses without additional risks. However ECT should be administered only after thorough evaluation of risks and benefits in such individuals.

Key Words: ECT , Neorological illnesses

Electroconvulsive therapy (ECT) is a technique to induce convulsions for therapeutic purposes using pulsed direct current. Use of ECT in the treatment of major psychiatric illnesses has been established (Abrams,1997). ECT is also useful in the treatment of co-morbid psychiatric illnesses associated with various neurological conditions (Dubovsky,1986). In addition ECT seems to hold promise for the treatment of certain neurological disorders (Gangadhar et al.,2001).

Patients with neurological disorder may have either a primary or secondary psychiatric illness (Lishman, 1998). Approach towards underlying aetiology is important in all such cases. However, many such patients need additional psychiatric intervention. It may be counselling, psychotropic drug or ECT. In general, ECT may produce a transient organic mental syndrome in nearly 50 percent of cases (Summers et al.,1979). If such were the case one would find it strange to use this treatment in patients with conditions affecting the structure or function of the brain. In practice however, ECT has been found to produce fewer adverse effects than might intuitively be expected (Turek and Hanlon,1977). Recently conducted neuroimaging studies found no changes in the brain structure or brain water content after ECT (Girish ,1999). Thus ,use of ECT in neurological conditions may be safe. This article briefly reviews the use of ECT in the treatment of neurological disorders and associated co-morbid psychiatric illnesses.

<u>Delirium and epilepsy</u>: For management, the underlying cause for delirium must be evaluated, while antiepileptics form the mainstay in the treatment of epilepsy. In both these conditions prolongation of the illness and / or inadequate control increases risk of mortality. Though rarely used, ECT is found to be helpful in such conditions.

<u>Delirium:</u> At least 10% of individuals receiving ECT have acute confusional state/delirium (Abrams,1977). The factors that increase the risk of ECT-induced delirium are male sex, higher age, first ECT session, bilateral electrode placement and concurrent use of psychotropic drugs like lithium. Besides this, ECT has been proven to be of use in patients with delirium not adequately responding to medications. Benzodiazepines or antipsychotic drugs are used to manage confusion, agitation, disruptive behaviour and hallucinations in delirious patients. However, their side effects,

in particular, the extrapyramidal side effects (with neuroleptics)must be monitored. When delirium is marked and persistent, ECT can be used as an alternative. Patients with delirium due to head injury, meningitis, encephalitis, pneumonia, delirium tremens, porphyria, uraemia, Wernicke's encephalopathy and who have inadequately responded to drugs (e.g.barbiturate, phencyclidine or bromides) were treated sucessfully with ECT (Dubovsky, 1986). Though ECT- induced complications (prolonged and severe confusion, amnesic effects) are higher in patients with pre-existing neurological disorder (Krystal and Coffey, 1997), none of these patients had such complications. Delirium can also follow cluster attacks of epilepsy; ECT was found useful in such situations (Dubovsky, 1986). However, the approach should be to treat aggressively the underlying aetiology for delirium.

Epilepsy: During the ECT course, seizure threshold increases progressively. The threshold when reassessed in sixth ECT session is found to be one-and half to two times higher than threshold at first ECT session (Abrams, 1997). This suggests anticonvulsant property of ECT. In patients with intractable epilepsy (who are poor responders to pharmacological agents) ECT has been used with success (Krystal and Coffey, 1997; Sackeim et al.,1983). There is difference in opinion among physicians in altering the antiepileptic regime when these patients are prescribed ECT. Abrupt reduction or withdrawal of anticonvulsants may precipitate seizures/ status (Hauser, 1983). Hence they need to be continued during the ECT course. Some advocate reduction in the dosage of anticonvulsant medication prior to ECT because of its effects on seizure threshold and seizure induction. It is better to reduce the drugs only when induction of seizures is difficult or patient has repeated subshocks (American Psychiatric Association, 2001) The ECT stimulus dose required for seizure induction may be higher in patients who are on antiepileptic drugs (Roberts and Attah, 1988). The morning dose is usually withheld before ECT. Blood levels may be monitored and maintaining anticonvulsants at the lower end of therapeutic

range may be at time useful; thereby minimizing the interference with ECT seizure induction Serial monitoring of blood levels should be considered during the ECT course, particularly if levels have not been stable in the recent past.

Epilepsy is associated with high prevalence (>30%) of psychiatric co-morbidity (Levin et al.,1988), although it is difficult to form an accurate estimate. The type of epilepsy and range of psychiatric illnesses seen during the lifetime course of epileptic individuals compound it. Ictal or post-ictal confusional state or psychosis, epileptic personality chnage, impulsivity and aggressiveness are common. Nearly one-fourth may develop depression (Robertson and Trimble,1983). Control of seizure is the mainstay of treatment in all these cases. ECT is effective in epileptic psychosis poorly responding to psychotropic drugs. Marked abnormalities of mood or florid delusions or hallucinations have better response to ECT (Lishman, 1998). ECT can be prescribed even in the presence of confusion and clouding of consciousness. However, the epileptic basis of the disease should be well established and other intracranial causes have to be excluded. There may be increased risk of prolonged seizures during ECT (Devinsky and Duchowny, 1983).

Stroke and brain tumours: ECT may also be used when psychiatric illnesses arise secondary to neurological conditions or when they coexist with neurological conditions. Stroke and brain tumours are some such conditions wherein ECT has been found useful to treat the psychiatric illness.

Stroke: Depression is seen in around 30% of patients in the first few weeks following stroke. Two-thirds of these have major depression (Burvill et al.,1995). This is also the commonest comorbid psychiatric illness in patients with stroke. In those with severe depression ECT has been found useful. ECT produces steep rise in heart rate and blood pressure. It is presumed that prescribing ECT in patients who had a cerebrovascular accident or stroke posses additional risk. But this has not been seen in practice (Miller and Isenberg, 1998). This may be because the rise in haemodynamic status due to ECT is transient. Although rare, death recorded in the past may be

due to co-morbid cardiovascular complications in stroke patients (Heshe and Roeder,1976).

There is hardly an increased risk of cognitive impairment with ECT in stroke patients; some even reported improvement in their cognitive status (Murray et al.,1986). There is however increased risk of ECT- induced delirium in patients who had stroke (Martin et al.,1992) or MRI hyperintensities in basal ganglia (Figiel et al.,1991). To minimize any additional risk of ECT- induced complications, it is best prescribed at least a month after stroke (Murray et al.,1986). ECT in individuals with recent stroke or cerebral aneurysm must be avoided. The acute ECT-related hypertensive surge should be pharmacologically blunted when ECT is given for patients at risk for a bleed (Krystal and Coffey,1997). Maintenance of blood pressure within fairly narrow range (using β blockers, nitroprusside) is required (Allman and Hawton, 1987), in order to avoid the potential risk of cerebral bleeding with severe hypertension or cerebral ischemia with hypotension. Aggressive antithypertensive medication may increase the risk of hypotensive morbidity. ECT has also been used successfully to treat depression in patients with intracerebral arterial (Husum et al.,1983; Drop et al.,1988) or venous malformations (Greenberg et al.,1986). In patients who are on anticoagulant therapy (either heparin or warfarin), ECT can be used safely without any untoward complications (Abrams, 1997). However, close monitoring of coagulation parameters is mandatory.

Intracranial space occupying lesions: The incidence of psychiatric illness in patients with brain tumour can vary between 10% and 100% (Lishman, 1998). Large and rapid growing tumours produce raised intracranial pressure (ICP). Raised ICP is a contraindication for ECT. ECT increases the ICP transiently. Though reported, serial monitoring of spinal fluid pressure and dynamics with ECT did not show any rise(Dressler and Folk,1975).Steroids are used to reduce brain oedema before prescribing ECT to treat co-morbid psychiatric illness in patients with brain tumour (Zwil et al.,1990). Small and slow growing neoplasms hardly raise ICP (such as

meningiomas). ECT can be safely administered in such cases. On the other hand ECT in presence of large and malignant tumours may further increase ICP and potentiate adverse effects (Fried and Mann, 1998; Zwil et al.,1990). These include noncardiogenic pulmonary oedema, cerebral oedema, brain haemorrhage, brain herniation, neurological deterioration and rarely death (Krystal and Coffey, 1997). However such events are rare in practice (Maltbie et al.,1980). A neurological catastrophe in an apparently healthy patient receiving ECT may be the first sign of an unsuspected brain tumour. ECT was used successfully in the treatment of organic depression in patients with other space-occupying lesion such as chronic subdural haematoma (Malek-Ahmadi et al.,1990) and intracranial arachnoid cysts (Escalona et al.,1991) without any additional risk. In patients with raised ICP one must evaluate the risk benefit ratio in each individual case.

Craniotomy patients with psychiatric illness may at times be referred for ECT. Stimulus electrodes are placed farthest from the cranial defect without any additional special precautions. Patients with co-morbid depression in meningioma (Hsiao and Evans,1984), pituitary adenoma (Ries and Bokan, 1979), third ventricle colloid cyst (Roccaforte and Burke, 1989) or bullet shot injuries (Ruedrich et al.,1983) who underwent craniotomy improved with ECT.

Normal pressure hydrocephalus (NPH) may present with psychiatric symptoms like depression, psychosis or dementia (Lishman, 1998). These symptoms may quite often predate the appearance of classical symptoms of NPH (Pujal et al.,1989). Psychiatric illness remits with surgical correction of NPH. However, some needed ECT for treating depression (Cardno and Simpson, 1991) with improvement in neurological condition (Mansheim,1983). Other had marked post - ECT confusion and memory loss (Price and Tucker,1977).

Disorders of basal ganglia: Basal ganglia is implicated in various psychiatric conditions associated with movement disorders. Some of the

neurological conditions too arise due to basal ganglia pathology. Use of ECT has helped to treat both neurological conditions and/or associated psychiatric illness.

Parkinson's Disease: The commonest degenerative disorder of basal ganglia is Parkinson's disease (PD). It is more prevalent after forty years of age and males outnumber females. They typically present with rigidity, tremors, bradykinesia and postural instability. Hypodopaminergic state is a well-established aetiology in these patients (Adams et al.,1977).

Among all the neurological conditions, use of ECT in the treatment of neurological symptoms of PD is extensively studied. Experience with ECT in PD patients is promising (Rasmussen and Abrams, 1991; Kellner et al.,1994). Independent of effects on psychiatric symptoms, ECT commonly results in general improvement in motor functions (Pridmore and Pollard, 1996). Motor rigidity and bradykinesia improve markedly followed by improvement in tremors. Antiparkinsonian agents such as levodopa and amantadine used in PD have 'on-off' phenomenon. During ' on ' phase the drug is ' active ' and the motor symptoms are better. This lasts for few minutes to hours. During the interval period, until the patient receives the next dose, the drug is no more active and the symptoms are obvious i.e. ' off phase'. With long term use the effectiveness of the drugs diminishes, so also the duration of symptom-free periods. When used for prolonged period (months to years) it invariably results in paradoxical worsening of the neurological symptoms in PD and increases the incidence of depression (15-25%; Koller et al.,1994). Continuation may also produce independent dyskinetic movements adding to their disability. This is possibly due to hypersensitivity of dopaminergic system. Rarely antiparkinsonain agents, in particular amantadine, may precipitate psychosis in PD patients (Celesia & Barr,1970). Use of antipsychotic drug in such cases complicates the clinical picture. ECT in such cases is an alternative. Patients with the 'on-off' phenomenon may show considerable improvement with ECT (Andersen et al.,1987).

PD patients refractory to antiparkinsonian agents or intolerant to their side effects with severe disability (e.g., bedridden, on- off status) are suitable candidates for ECT. Dose of levodopa should be reduced to half to prevent emergent dyskinesia (levodopa and ECT together may result in dopaminergic surge). The adjunctive agents (e.g., anticholinergic drug, amantadine) should be discontinued before starting ECT in view of their potential side effects with concomitant use. Brief-pulse, suprathreshold, non-dominant, unilateral ECT reduces the possible risk of cognitive side effects (Abrams,1997). Change over to bilateral may be considered in case of no response to first three unilateral ECTs. The antiparkinsonian medication should be restarted after the course of ECT. Some of PD patients are either non-responders or intolerant to antiparkinsonian agents. They are potential candidates for maintenance ECT (Pridmore and Pollard, 1996). It helps to maintain improvement in motor symptoms without worsening the cognitive functions. Maintenance ECT is administered initially once a week and gradually made once a month. However treatment strategies should be individualized to the patient. Rarely, in PD patients ECT may result in transient emergent dyskinesia (Rasmussen and Abrams, 1991). This may be due increased postsynaptic dopamine receptor sensitivity caused by ECT.

The beneficial effect of ECT on the motor symptoms of PD is highly variable in its duration; more so in patients who are resistant or intolerant to standard pharmacotherapy. In general, improvement begins with the first treatment, and is maximum after three to four ECTs. The improvement is maintained for two to four months after a course. There is preliminary evidence that continuation of maintenance ECT may be helpful in prolonging the therapeutic effects (Pridmore and Pollard,1996). Laterality, stimulus dose (threshold v/s suprathreshold), duration of illness and prior drug treatment had no influence on outcome. However, the higher the age greater is the improvement (Rasmussen and Abrams, 1991).

Depression in PD is endogenous rather than exogenous in origin (Schrag et al.,2001).

Nearly 50% of PD patients have depression and one third of them have moderate to severe depression (Robins, 1976). When depression is mild to moderate, antiparkinsonian agents and counselling lift it appreciably (Brown and McCarthy,1990). Low dose antidepressants are also helpful in such conditions. PD patients may have marked disability due to rigidity and bradykinesia. Depression adds to their disability. Antidepressants are necessary to treat severe depression (Lishman,1998). Alternatively, ECT produces marked and early remission of depression (Douyon et al.,1989). ECT is preferred when depression is severe with melancholia/ psychotic symptoms, with high suicidal risk/ deliberate self harm or when associated with marked disability. Increase in dopamine at synaptic levels with ECT brings early remission of depression in PD patients (Rasmussen and Abrams,1991).

As many as 20% of PD patients may develop psychosis; antiparkinsonian medication has been implicated in the causation (Ron,2000). Classical antipsychotic drugs by blocking postsynaptic dompaminergic receptors in the striatum worsen the neurological symptoms of PD (Koller et al.,1994). Low doses of atypical antipsychotic drugs such as olanzepine (2.5 mg) or clozapine (25mg) are found useful to treat psychosis in PD (Juncos,1999). ECT may be preferred when the patient is intolerant to antipsychotic drugs, grossly psychotic, violent and/ or unmanageablle. How ECT works in such conditions without worsening symptoms of PD remains unexplained. ECT- induced delirium or memory disturbances may be minimised when unilateral ECT is used to treat co-morbid psychiatric illness associated with PD (Abrams,1997).

Huntington's Disease: Huntington's disease (HD) is a triad of choreoathetosis, dementia and dominant inheritance. Though HD has a chronic course, antipsychotic drugs are effective in suppressing the movement disorder. Chorieform movements resistant to pharmacological treatment improved with ECT without any untoward side effects (Beale et al.,1997). Very often psychiatric illnesses are seen along with HD and in some it may be the initial presentation. Depression is the commonest next to dementia (30-35% ; Folstein et al.,1983). Six out of seven such patients not responding to antidepressants improved markedly with ECT (Lewis et al.,1996; Ranen et al.,1994). The two patients who had prominent delusions showed the greatest improvement. There was no worsening of cognitive status or movement disorder; rather the latter improved with ECT. However, one patient developed delirium and the movement disorder worsened in another patient. ECT should be a treatment option in the management of HD particularly with resistant depression.

Movement disorders: Neuroleptic-induced movement disorders are common. These include extrapyramidal symptoms (EPS) such as parkinsonian tremors, akathisia, dystonia and long term side effect- tardive dyskinesia. History of antipsychotic drug intake must be established in all such cases. Stopping or reducing the dose of psychotropic drug brings marked response.

Neuroleptic-induced EPS also improves when ECT used to treat the underlying psychiatric illness (Gangadhar et al.,1983; Goswamy et al.,1989), even in patients who continue on neuroleptic drugs. In patients with tardive dyskinesia, tardive dystonia, tardive akathisia, or tardive parkinsonian symptoms ECT has been tried with promising results. It is particularly useful in tardive dystonia. However data on maintenance ECT in these patients is limited.

ECT was found useful in buccolingual or orofacial dyskinesia induced by long term antipsychotic medications (Abrams, 1997). Improvement in dyskinetic movements was maintained until one year (Gosek and Weller,1998). In all these patients the underlying psychiatric illness (either psychosis or secondary depression) remitted with ECT. Most patients showed improvement after 3-4 ECTs. However, Yassa et al.(1990) in their prospective study found improvement in dyskinetic movements in only one out of nine TD patients.

Severe dystonic gait posture (Kwentus et al.,1984) and sternomastoid dystonia (Adityanjee

et al.,1990) improved with ECT when prescribed for co-morbid psychosis. The remission was maintained when patients were followed for one year. However in some patients there was worsening of dyskinetic movements after ECT and some had developed dyskinetic movements with ECT (Roth et al.,1988). ECT- induced dyskinetic movements were transient and disappeared either immediately or within few weeks after stopping ECT without any additional intervention.

Catatonia and Neuroleptic Malignant Syndrome: Catatonia is a medical emergency and should be treated as an organic condition unless and otherwise proved. Presence of clouding of consiousness that worsens in the evening, perseveration and neurological signs during examination favour an organic basis for catatonia. Parenteral administration of benzodiazepines may relatively worsen organic catatonia. Neurological disorders and medical illnesses may also present with catatonia as a complication. Hence catatonia calls for immediate and thorough investigation to identify the causative factor. Treatment of underlying pathology resolves catatonia. Neuroleptic malignant syndrome (NMS) also presents with catatonic symptoms rarely as a side effect of neuroleptic drugs. ECT is useful in both these conditions.

Catatonia: Patients with typhoid, pellagra, pernicious anaemia who had catatonia not responding to pharmacological agents improved adequately to ECT (Dubovsky, 1986). Thus, though rare, irrespective of the causative factor ECT has been used with success in catatonia. However, in the presence of underlying medical or neurological condition, ECT may result in further deterioration of clinical condition. Hence ECT should be judiciously used in organic catatonia explaining the additional risk to the patient and / or the family.

Functional illnesses such as mania, depression, schizophrenia or dissociative disorder may present with catatonia or catatonic symptoms. Periodic catatonia though rare is associated with an extreme shift in the metabolic nitrogen balance (Gjessing, 1974). Majority of functional catatonia respond to lorazepam trial (6-8 mg per day for at least three days; Bush et al.,1996). ECT is effective in those not responding to lorazepam (Bush et al.,1996). Antipsychotic drugs have also been found useful. However there is no single clinical trial comparing the effectiveness of antipsychotic drug and ECT in non-affective catatonia. Similarly no study has compared at the effectiveness of ECT with antidepressants in depressive catatonia. It is generally believed that ECT is a better choice compared to psychotropic drugs both in terms of clinical efficacy and side effects. Even the more malignant from- lethal catatonia (characterised by mutism, extreme motor exceitment, clouding of consciouness and fever progressing to severe autonomic disturbances, stupor, coma and death) may respond to ECT (Mann et al.,1986).

Neuroleptic Malignant Syndrome: Among all the psychiatric conditions, NMS has the highest mortality. Nearly 10% of these succumb to this condition (Shalev et al.,1989). Early identification and prompt treatment reduces the fatality. Fatality is reduced to less than half with the use of ECT. One should be cautious when ECT is used to treat NMS. One must withdraw all antipsychotic drugs, control fever, correct electrolyte imbalance and autonomic instability prior to ECT.

ECT is preferred to other drugs in treating NMS. In addition, ECT also helps to treat the underlying psychiatric illness. Modification procedure during ECT needs to be alternated. Succinylcholine, a muscle relaxant may predispose for malignant heperthermia in NMS (Galloway and Denborough, 1986). Instead atracurium is used. In such cases anaesthesia is reversed using neostigmine. Besides risk, succinylcholine has been used successfully in nonactive NMS by many psychiatrists without any additional risk (American Psychiatric Association,2001). If NMS is severe succinylcholine increases the risk of hyperkalemia. It is important for patients with active NMS symptoms to be observed for metabolic or cardiovascular instability over the ECT course (Scheftner and Schulman, 1992). ECT by potentiating autonomic instability may precipitate cardiovascular complications in patients with NMS and may rarely result in death.

Multiple-monitored ECT (multiple seizure inductions in a single ECT session) has been suggested to hasten the time of recovery in severe NMS (McKinney and Kellner,1997).

Degenerative disorders: Psychiatric illnesses are commonly associated with degenerative disorders. They tend to be progressive and severely impair the quality of life in these patients. Hence treatment of psychiatric conditions needs at most atention. ECT has been found useful in some of these patients besides counselling and psychotropic drugs.

Dementia: The prevalence of dementia rises markedly with age from about 2% in persons aged 65-70 years to 20% in those over 80 (Lishman, 1998). During the course there is progressive decline in several higher cortical functions including memory, thinking, comprehension and language . Dementia is commonly associated with comorbid psychiatric illnesses. A major depressive episode is found in approximately 10% of patients with dementia, minor depressive episode in 25% and some features of depression in 50% (Rovner et al.,1989). Depression when marked may be associated with high suicidal risk, psychomotor retardation, psychotic symptoms or agitation. ECT has been used with success. ECT induced cardiovascular complications can be prevented by thorough examination of cardiovascular system, active control of hypertension and looking for cardiac efficiency before prescribing ECT. Old age is at risk for ECT induced cognitive side effects. However, systematic studies in large populations have not confirmed this (Nelson et al.,1991; Price et al.,1989). Instead there was mild improvement in cognition in a small group of patients. This may possibly be related to improvement in pseudodementia in depression. However, disorientation immediately after ECT was profound and prolonged (Summers et al.,1979).

Nonetheless, to minimise ECT- induced cognitive side effects one should use brief pulse stimulus instead of sinewave stimulus, unilateral instead of bilateral ECT and twice weekly instead of thrice weekly. EEG monitoring should be encouraged. Use of pulse oximeter to record heart rate, blood pressure and ECG throughout at ECT procedure is recommended in the elderly (American Psychiatric Association,2001).

Neuromuscular and Neurodegenerative disorders: Multiple sclerosis is commonly associated with depression and suicide. Depression is present in about 50% of patients (Ron and Logsdail,1989) and there is seven-fold increase in the expected rates of suicide (Stengager et al.,1992). Those not responding to antidepressants showed good response to ECT (Kwentus et al.,1986; Coffey et al.,1987). There was no worsening of the neurological status. The later study did not find any change in white matter lesion in MRI scans done before and after the ECT treatment. ECT has also been successfully used to treat psychiatric patients suffering from cerebral palsy, myasthenia gravis, muscular dystrophy and Friedrich's ataxia all without any complications (Dubovsky, 1986).

During anaesthesia, succinylcholine used in patients with neuromuscular disorder may have potassium releasing and muscle-depolarising action. Increased sensitivity to succinylcholine may also be seen in patients with upper motor neuron diseae e.g., quadriplegia or amyotrophic lateral sclerosis (Janis et al.,1995). Instead atracurium, a non-depolarising muscle blocker is used (Hicks, 1987).

Guidelines for approach: Neurologically ill patients when prescribed ECT for their neurological symptoms or for associated psychiatric co-morbidity can have relatively higher risk of ECT - induced side effects. There can also be some worsening of neurological symptoms. The following paragraph summarizes the approach in neurological disorder patients prescribed ECT (American Psychiatric Association, 2001).

1. The decision for ECT must be individualised depending upon the risk and benefits.
2. Pre-ECT evaluation should include pertinent laboratory tests and specialist consultation when indicated.
3. Informed consent should include increased risk.
4. Necessary changes in ECT procedure should be done to minimise the risk. They may include altering ECT technique (stimulus dose, laterality and anaesthetic drugs), pharmacologic regimens

and monitoring procedures.

5. Patients with increased ICP have substantially elevated risk with ECT; its use must be adequately justified in terms of risk- benefit consideration. In such cases, treatment modifications, such as use of potent antihypertensive agents, steroids, diuretics and hyperventilation, should be considered to diminish risk.

6. Short-acting antihypertensive agents should be considered at the time of ECT in patients at risk for haemorrhagic cerebrovascular events (e.g., aneurysms, arteriovenous malformations).

7. Certain neurological conditions may be associated with increased severity of cognitive dysfunction during and immediately following ECT e.g., dementia, brain trauma, PD and multiple sclerosis. This does not imply that ECT is contraindicated in these conditions.

8. In patients with epilepsy, doses of anticonvulsant drugs should be optimised to maintain effective seizure control, yet still permit adequate seizure induction with ECT.

9. In patients with PD, dosage of dopaminergic agents should be optimised to maintain control of motor symptoms while also adjusting for increasing dopaminergic effects with ECT.

10. Neuroleptics should be avoided in NMS. In severe NMS nondepolarizing muscle relaxants should be considered at the time of ECT. Such patients should also be observed for metabolic and cardiovascular instability.

Conclusions: ECT is used, though infrequently, to treat some of the neurological illnesses. These include PD, particularly with the 'on' & ' off ' phenomenon, NMS, delirium and intractable seizure disorders. ECT seems appropriate for patients with a combination of an affective and a neurological disorder and for patients who are dangerously suicidal, self-destructive, agitated, and/ or grossly negativistic. Neurological diseases, including brain tumours, are not necessarily contraindications to the use of ECT. However, ECT is reserved for patients who are resistant or intolerant to standard medical treatments or who require an urgent response.

Detailed neurological evaluation of risk factors should be carried out prior to ECT.

Concurrent medications must be adjusted appropriately. ECT procedures must be optimised which may diminish the level of risk. Specific conditions that may be associated with substantially increased risk include : a) aneurysm or vascular malformation that might be susceptible to rupture with increase blood pressure, b) increased ICP as may occur with some brain tumour or other space occupying lesion and c) recent cerebral infarction.

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