REVIEW ARTICLE

STEROIDS IN ANAESTHETIC PRACTICE – REVIEW ARTICLE

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SUMMARY:

Steroids are perhaps one of the most widely used group of drugs in present day anaesthetic practice, sometimes with indication and sometimes without indications. Because of their diverse effects on various system of the body, there has been renewed interest in the use of steroids in modern day anaesthetic practice. This brief review focuses on the synthesis and functions of steroids and risks associated with their supplementation.This review also highlights the recent trends, relevance and consensus issues on the use of steroids as adjunct pharmacological agents in relation to anaesthetic practice and intensive care, along with emphasis on important clinical aspects of perioperative usefulness and supplementation.

KEY WORDS:

Steroids, Perioperative steroid replacement, PONV, ESI(epidural steroid injection), Analgesic adjuncts, Anaphylaxis, Sepsis, Septic shock, Post intubation laryngeal odema, Post extubation stridor, Adverse effects. Cerebral odema, Cardiac arrest.

INTRODUCTION:

Corticosteroids and their biologically active synthetic derivatives differ in their metabolic (glucocorticoid) and electrolyte-regulating (mineralocorticoid) activities. The effects of corticosteroids are numerous and widespread, and include alterations in carbohydrate, protein, and lipid metabolism; maintenance of fluid and electrolyte balance; and preservation of normal function of the cardiovascular system, the immune system, the kidney, skeletal muscle, the endocrine system, and the nervous system. In addition, corticosteroids endow the organism with the capacity to resist such stressful circumstances as noxious stimuli and environmental changes[1,2,3].

EFFECT OF ANAESTHESIA AND SURGERY:

Plasma cortisol levels typically increase (two to ten folds) following induction of anesthesia, during surgery, and in post-operative period. The maximum ACTH

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and cortisol levels are reached in the early post operative period, especially following anaesthesia reversal and endotracheal extubation (30min post extubation)[8,9,10].

As with other types of stress, the episodic release of cortisol remains intact, but the amplitude of these episodic release is increased. The increase in the plasma cortisol concentration may in part be due to bar receptors and spinal reflexes that signal the hypothalamus of tissue injury associated with surgery[3,4].

Other factors that activate HPA axis to release cortisol include pro-inflammatory mediators released by damaged tissues and presence of post-operative pain.

Plasma cortisol concentrations typically return to normal levels within 24 hrs post-operatively but may remain elevated as long as 72 hrs, depending upon severity of the surgical trauma.

Return of the plasma concentration to normal following surgery is initially characterized by increased plasma concentration of ACTH and cortisol (in first 24hrs)[11,12] followed by a second phase(48-72hrs)[10,13] in which plasma ACTH concentrations are low and increased plasma cortisol concentrations are presumably independent of HP system[14].

In addition to surgical trauma, choice of anesthetic drugs and techniques may influence the HPA response. For Eg. Large doses of opiod may attenuate the cortisol response to surgical stimulation[15]. Volatile anesthetics provide less suppression to this stress induced endocrine response[15]. Etomidate is unique among drugs administered to induce anesthesia with respect to its ability to inhibit cortisol synthesis (selectively inhibits adrenal 11beta hydroxylase, the enzyme that converts 11 deoxy cortisol to cortisol) even in the absence of surgical stimulation[16].

Previous studies have shown cortisol and ACTH levels increase during normal pregnancy, particularly in the second and third trimesters[18]. Some authorities have recommended increasing glucocorticoid replacement doses by 50% in the last trimester of pregnancy for women with adrenal insufficiency. Whether this is advisable may depend on the patients usual treatment dose, as it has been shown that a dose increase is rarely necessary in women treated with 20-30mg hydrocortisone daily.

IMPORTANT INDICATIONS OF STEROID IN ANAESTHETIC PRACTICE:

Perioperative steroid replacement therapy:

Corticosteroid supplementation should be provided for patients being treated with steroids either for hypocortisolism or for other diseases.

This is based on the concern that these patients are more prone to cardio-vascular collapse as release of additional endogenous cortisol in response to surgical stress is not likely. Some patients may display suppression of pituitary-adrenal axis with atrophy of adrenal cortex from long continued therapy with steroid drugs.

Patients on long term steroid therapy[4]:

Indications :

1) Hypocortisolism – primary, secondary
2) Other diseases : Rheumatoid arthritis, Psoriasis, SLE, Ulcerative colitis, Crohn's disease, Bronchial asthma, Idiopathic thrombocytopenic purpura, Myasthenia gravis

Steroid administration is necessary in perioperative period in patients treated for hypoadrenocorticism or in patients with suppression of HPA axis owing to previous or present steroid intake[5]. The increase in circulating cortisone levels from normal of 25mg/day to upto 300mg/day in

severe surgical stress is one of the important components of stress response of our body. In the perioperative period due to adrenal suppression, there can be increased vascular permeability, inadequate vasomotor response, decrease in cardiac output and decrease in systemic vascular resistance and left ventricular stroke volume index which can lead to severe hypotension and cardiovascular collapse, respiratory depression, hyponatremia, hypoglycemia, hypercalcemia and hemoconcentration[6].

The specific duration and dose of steroid that can produce HPA suppression is controversial. The recovery time of normal HPA axis varies from 2-5days to 9-12months after discontinuation of steroid therapy. But the ability to respond to stress returns by 2months[7].

Traditionally it was believed that the degree of HPA suppression and adrenal atrophy in patients receiving exogenous glucocorticoids was related to duration and dose of therapy[8,9]. In patients taking steroids for less than 3weeks, suppression of HPA axis is rarely clinically insignificant[23]. Conversely, any patient who has received the equivalent of 15mg/day of prednisolone for more than 3weeks should be suspected of having HPA suppression[9]. However recent studies have found poor corelation between HPA axis function and the cumulative dose or the duration of therapy[10,11]. Because of considerable inter-individual variability in the degree and duration of adrenal suppression, it is difficult to accurately predict which patients will develop adrenal insufficiency when glucocorticoid treatment is discontinued.Thus the need to evaluate HPA is a frequent consideration.

Under peri-operative conditions adrenal glands secrete 116-185mg of cortisol daily. If plasma cortisol is measured during acute stress, a value of more than 25µg/dl assuredly and more than 15µg/dl probably indicates normal pituitary-adrenal responsiveness[12].

The intactness of the HPA axis and need for steroid may be assessed by provocative tests which measure the plasma cortisol response to administration of ACTH, CRH, lysine, vasopressin, metyrapone and insulin induced hypoglycemia[12]. The gold standard for assessment of HPA function is the insulin tolerance test but short synacthen test is cheaper and less unpleasent[13].

Original recommendations for perioperative steroid supplementation were in excess of what was actually required.

The present approach is to replace the amount equivalent to normal physiologic response to surgical stimuli. The amount of steroid supplementation, dose and duration should be based on the magnitude of surgical stress as well as perioperative steroid dose and degree of HPA suppression. Also it is important to note that oral steroids must be supplemented by parenteral steroids in equivalent doses[15].

Salem et el categorised[16],

a) Minor surgical stress as inguinal hernioraphy, appendicectomy, dental procedures of >1hr duration under local anaesthesia, caesearean section[16].

b) Moderate surgical stress as non laproscopic cholecystectomy, abdominal hysterectomies, lower limb revascularisation, segmental colon resection, THR[16].

c) Severe surgical stress as whipple's resection, esophagectomy, pitutary adrenalectomy, total proctocolectomy, liver resection, cardiopulmonary bypass, dental procedures under general anaesthesia, orthognathic surgery, severe facial trauma[16].

One of the widely practised steroid replacement in perioperative setting is given by Kehlet, Symreng, Salem et al[16].

Patient currently taking steroids	<10 mg/day (Prednisolone)	Assume normal HPA Response	Additional steroid cover not required
	>10mg/day	Minor surgery	25 mg hydrocortisone at induction
		Moderate surgery	Usual perioperative steroid + 25 hydrocortisone at induction + 100mg/day for 24 hrs
		Major surgery	Usual perioperative steroid + 25 hydrocortisone at induction + 100mg/day for 48-72 hrs
	High dose immunosuppressive	Give usual immunosuppressive dose during perioperative period	
Patient stopped taking steroid	<3 months	Treat as if on steroids	
	> 3 months	No perioperative steroid necessary	

Alternately, another regimen is to give 100mg hydrocortisone followed by, in minor cases by 100mg 6-8hrly for 24hrs and in major cases by 100mg 6-8hrly for 72hrs[17].

Steroid Coverage for illness or surgery in hospital[18]:

a) For minimal illness(non febrile cough or upper respiratory tract infection), usual replacement dose should be given[18,19].

b) For minor illness (viral illness, bronchitis, uncomplicated urinary tract infection) double or triple the usual dose of glucocorticoid until recovery.

c) For moderate illness (gastroenteritis, pneumonia, pyelonephritis) give hydrocortisone 50mg bd orally or iv. Taper rapidly to maintenance dose as patient recovers.

d) For severe illness (pancreatitis, myocardial infarction, labour) give hydrocortisone 100mg iv every 8th hrly. Taper dose to maintinence level by decreasing by half every day. Adjust dose according to course of illness[18,19].

e) For minor procedures under LA & most radiological studies, no extra supplementation is needed[18].

f) For moderately stressful procedures, such as barium enema, endoscopy or arteriography, give a single 100mg iv dose of hydrocortisone just before the procedure[18].

g) For major surgery, give hydrocortisone 100mg iv just before induction of anaesthesia & continue every 8th hrly for first 24hrs. Taper dose rapidly to maintinence level by decreasing by half every day[18].

Patients with HPA axis depression:

Based on Symreng et al[20], in patients with proven adrenocortical insufficiency a low dose physiological substitution regimen results in circulating cortisol values greater than in normal patients and is sufficient to prevent intra operative hemodynamic instability. An infusion is preferable as it avoids large increase caused by bolus doses[20]. A rational regimen for steroid supplementation in the perioperative period is administration of cortisol 25mg iv, at the induction of anaesthesia followed by continuous infusion of cortisol 100mg during the following 24hrs. This approach maintains the plasma concentration of cortisol above normal during major surgery in patients receiving chronic treatment with steroids and manifesting a subnormal response to preoperative infusion of ACTH[20]. In addition to intravenous supplementation with cortisol, patients receiving daily maintinence doses of steroids should also receive this dose with premedication on the dey of surgery. In those instances like burns or sepsis which exaggerate the need for exogenous steroid supplementation, the continuous infusion of cortisol, 100mg every 12hrly is sufficient[20].

STEROIDS AND PONV:

The major risk factors for PONV include female gender, nonsmoker status, history of PONV or motion sickness, intraoperative use of volatile anesthetics and high-dose opioid techniques, as well as postoperative opioid analgesic use[21]. In adults, a multidrug antiemetic prophylaxis strategy is recommended for patients who present with two or more risk factors.

The antiemetic mechanism of corticosteroids is unknown. Various studies have been conducted to evaluate the efficacy of steroids in managing PONV. Dexamethasone, a corticosteroid with strong anti inflammatory effects, provides postoperative analgesia, prevents nausea and vomiting in patients undergoing chemotherapy [22,23], and reduces post-operative nausea and vomiting (PONV). Dexamethasone may inhibit the synthesis of prostaglandin and various other inflammatory mediators, which are known to act on chemoreceptor trigger zone and cause emesis [24]. Previous studies suggested that decreased serotonin release in the central nervous system and changes in the permeability of the blood cerebrospinal fluid barrier to serum proteins[25] may also play a role in the antiemetic effects of corticosteroids.

Jhi- Jhung et al[27] evaluated the effect of timing of dexamethasone administration on its efficacy as a prophylactic antiemetic on postoperative nausea and vomiting and stated that dexamethasone, when given immediately before the induction of anesthesia, was more effective than when given at the end of anesthesia in preventing PONV in patients undergoing abdominal total hysterectomy, thyroidectomy, adenotonsillectomy and cholecystectomy.

STEROIDS AND AMBULATORY SURGERY:

Post operative and post discharge nausea and vomiting (PONV/PDNV) are the most common complications of ambulatory surgery. Gupta et al[29] in a meta analysis review, showed the incidence of POV (0-55%) and PDV (0-16%). Patients with PONV are significantly more likely to have problems performing activities of daily life, have a lower satisfaction score and higher negative economic impact than those not experiencing PONV.

Steroids decrease the incidence of PONV, post operative pain, establish early oral intake, stimulate appetite and induce a sense of well being (due to increase in release of endorphins)[30,31]. Dexamethasone is a corticosteroid with potent antiinflammatory effects that contributes to decreased wound pain after

oral surgery[32]. It also has antiemetic properties in patients receiving highly emetogenic chemotherapy[33].

More recently, dexamethasone has been used as a prophylactic antiemetic in the ambulatory surgery setting . Aesboe et al conducted a study on use of intramuscular single dose 12mg betamethasone, 30min before ambulatory hemorrhoidectomy or hallux valgus correction and they found significantly less PONV's, less post operative pain and better patient satisfaction.

STEROIDS AS ANALGESIC ADJUNCTS:

Analgesic effect of steroid is suspected to be mediated by anti-inflammatory and immune suppressive effect. Its anti inflammatory action results in decreased production of various inflammatory mediators that play a major role in amplifying and maintinence of pain perception. Some studies have demonstrated the analgesic effect of local spinal and systemic corticosteroids in combination with bupivacaine [34]. Dexamethasone microspheres have been found to prolong the block duration in animal and human studies[35], and adding methylprednisolone to local anesthetic increases the duration of axillary brachial block[36]

Paracetamol, NSAIDs, and glucocorticoids have a ceiling of analgesic effect, not being sufficient as monotherapy after extensive surgery[38]. As glucocorticoids act on the prostaglandin system differently than NSAIDs, and have other antiinflammatory effects, there may be better analgesia when glucocorticoids are added to NSAIDs.

In a study conducted by Romundstad et al., a single dose of 125 mg metylprednisolon was found to be analgesic for 3 days [40]. Bisgaard et al. reported reduced pain through 1 wk after a single dose of dexamethasone 8 mg during laparoscopy [41]. The plasma elimination half-life of dexamethasone is only about 6 h[39], suggesting a persistent drug effect unrelated to plasma concentration. Because glucocorticoids inhibit transcription [42], changes in protein expression can be expected to persist after the drug is cleared from plasma[40]

Adverse effects with a single dose of dexamethasone are probably extremely rare and minor in nature, and previous studies have demonstrated that short-term (<24 hours) use of dexamethasone was safe[43]. In one study, after approximately 2000 intrathecal injections of dexamethasone (8 mg) in 200 patients for treatment of posttraumatic visual disturbance, no neurological disorders were found at 1-month follow up[44]. Nerve injury is a rare complication of dexamethasone injection, and it usually occurs in the context of needle trauma[45].

EPIDURAL STEROID INJECTION(ESI):

ESI have been used to treat backpain (mainly due to nerve root irritation) in patients with a wide variety of spine pathologies including radiculopathy, spinal stenosis, disk-space narrowing, annular tears, spondylosis, spondylolisthesis, vertebral fractures, and postlaminectomy syndrome[46,47]. It has good safety record with few side effects like nerve/spinal cord damage, PDPH, epidural hematoma and abscess, vasovagal reaction. ESI provides an effective alternative to surgical treatment and is best for patients with lumbar disc disease who have not improved after 4weeks of conservative medical therapy[47].

A common regimen is use of 25-50mg triamcinolone or 40-80mg methylprednisolone in a solution containing lidocaine at or near the interspace corresponding to the distribution of pain[47]. Repetitive lumbar ESI have increased efficacy. It is hypothesised that it could be in part related to repetitive steroid uptake from epidural veins in post epidural space as well as from blood vessels in subarachnoid space after steroid passive diffusion across the dura.

The effects of epidurally administered

corticosteroids stem from their ability to inhibit the synthesis of prostaglandins, their antiinflammatory effects, and their ability to inhibit ectopic discharges from injured sensory nerves. Local anesthetics exert their analgesic effects by blocking the conduction in nerves via their effects on Na^+ channels and suppressing the ectopic signal generation in injured nerves. In addition to providing temporary pain relief, local anesthetics may provide prolonged benefits by putatively interrupting the cycle of pain. Although it seems logical that a larger dose of steroids injected around the affected nerve root would provide more effective analgesia than a smaller dose, the ideal dose and type of steroid has yet to be determined[46].

Positive results from epidural steroids vary from 20% to 95% and may depend on route of injection[49]. Lumbar ESI can be accomplished by one of three methods: caudal (C), interlaminar (IL), or transforaminal (TF). Each technique has been reported to be effective for reducing lower extremity radicular pain[50]. The TF route of epidural steroid placement is more effective than the C or IL routes.The potential benefits of a transforaminal approach may include minimal risk for dural puncture, better delivery of medication to the site of pathology, increased spread into the ventral epidural space, and subsequently a reduced amount of medication necessary to produce the desired effect.

Kay et al[51], in there study observed that 80mg triamcinolone lumbar ESI results in acute suppression of HPA axis (↓plasma ACTH and ↓plasma cortisol) 15minute after injection. Median suppresion of HPA axis was <1month and all patients recovered by 3months. So, proper perioperative steroid replacement is required if patient undergoes any stress/illness/surgery[51].

TRAUMATIC SPINAL CORD INJURY:

The use of steroids remains controversial for cord injuries because improvement is minimal and difficult to document[47,49]. A suggested protocol for traumatic cord injury includes the use of high dose methyl prednisolone with an IV bolus of 30 mg/Kg followed by 5.4 mg/kg/hr infusion for 23 hrs. Steroids must be used within 8 hrs of cord insult to be of any benefit[49]. Some of the partial cord syndromes have been reported to respond favorably and prompted the maintenance of steroids through subacute interval of one week followed by weaning.

Patient's may develop adrenal insufficiency (AI) after this protocol[52]. Although a definitive causal relationship between the steroids and AI was not established, their temporal association and the exclusion of other possible etiologies led them to postulate that AI was a complication of the steroid protocol. Clinicians should, therefore, consider AI in patients with spinal cord injury receiving glucocorticoids, a population in whom it may otherwise go undiagnosed and untreated[52].

STEROIDS IN HYPER-REACTIVE AIRWAY:

Steroids by virtue of their anti-inflammatory action leads to decreased mucosal odema and prevention of release of bronchoconstricting substances. They are aslso said to have a permissive role[2,3] for bronchodilator medication that is they enhance the efficacy of bronchodilator. They are useful in both acute as well as chronic hyper-reactive diseases. For this purpose they can be administered orally, parenterally, or in aerosol form. The most commonly encountered hyper reactive states in anaesthetic practice are patients with history of asthma, recent upper respiratory tract infection, difficult airway, multiple intubation attempts, aspiration, foreign body bronchus, airway surgeries and COPD[47,49].

ASPIRATION PNEUMONITIS:

The use of steroids in treatment of aspiration pneumonitis is controversial[47,49]. There is evidence in animals that steroids administered immediately after the inhalation of acidic gastric content, may be

effective in decreasing pulmonary damage[47]. Conversely, other data show no beneficial effect or suggests that the use of steroids may enhance the likelihood of gram negative pnemonia[49]. Despite the absense of confirming evidence that steroids are beneficial, it is not uncommon for the treatment of aspiration pneumonitis to include the emperical use of pharmacologic doses of these drugs.

POST INTUBATION LARYNGEAL ODEMA:

Steroids are commonly given after multiple attempts at intubation to prevent post operative laryngeal odema[49]. Steroids by virtue of their anti-inflammatory action leads to decreased mucosal odema. Dexamethasone 0.1-0.2mg/kg iv is commonly used, though its efficacy in treatment of this condition has not been confirmed[49]. Dexamethasone 0.6mg/kg orally is effective treatment for children with mild croup[53].

SEPSIS AND STEROID

Septic shock has a crude mortality rate of 45%[54] and claims the lives of 90 000 people each year in the USA alone[55]. Some recent approaches have shown promise in prevention or treatment of sepsis and septic shock. They include tight glycaemic control, early haemodynamic goal-directed therapy, infusion of activated protein C, and use of corticosteroids[56].

Patients having severe sepsis or in septic shock were found to have occult or unrecognized adrenal insufficiency, incidence may be as high as 28% in seriously ill patients[54]. Clinically it has been shown that in sepsis with adrenal insufficiency, steroid supplementation was associated with significantly higher rate of success in withdrawal of vasopressor therapy. Corticosteroids have a long history of use in intensive care for septic shock and were extensively used in high dose for a short duration until the mid-1980s when large multicentre trials showed they were of no benefit[57]. This finding was confirmed in subsequent meta-analyses[58]. Their use for septic shock was largely abandoned.

However, since then, small explanatory trials of physiological 'stress' doses of corticosteroids have demonstrated a reduced need for vasopressor drugs to maintain cardiovascular homeostasis, a possible surrogate for improved clinical outcomes[59]. The benefits of steroids may result from suppression of over-exuberant and dysregulated immune responses, suppression of inflammatory responses through a variety of mechanisms, and up-regulation of adrenoreceptor function[60,61]. However, adverse sequelae are well described and effects of corticosteroids on development of nosocomial infection, reactivation of latent infection, hyperglycaemia, bone metabolism, and psychosis as well as intensive care associated paresis must also be considered[62,63].

For patients who have a critical illness such as septic shock, Coursin and Wood[64] have recommended 50-100mg of hydrocortisone every 6-8hrs or 0.18mg/kg/hr as a continuous infusion, together with fludrocortisone 0.05mg daily. The current evidence does not support the use of hydrocortisone doses above 200mg/day8.

Arafah[65] reported that after intravenous boluses of hydrocortisone 50mg had been given 6 hourly, peak plasma cortisol levels were over 100ug/dl(2760nmol/L) and nadir levels remained elevated at 40-50ug/dl(1100-1380nmol/L)[66].

Keh et al[67] showed that , during continuous hydrocortisone infusion(10mg/hr), plasma total cortisol levels were over 3000nmol/L, well above the levels reported in patients with septic shock(mean- 880nmol/L)[68]. Another study found that the majority of cortisol levels were between 552 and 1242nmol/L in intensive care unit patients with severe sepsis or septic shock[69].

While it is evident that the glucocorticoid dose should not exceed 200mg/day[66], the optimal dose for managing septic shock in patients with adrenal

insufficiency has not been evaluated in controlled clinical trials. Mineralocorticoid supplemantation with fludrocortisone is not required in patients with secondary adrenal insufficiency or in those with primary adrenal insufficiency receiving more than 50mg hydrocortisone daily, given its potent mineralocorticoid activity at high doses[66].

STEROIDS AND POST EXTUBATION STRIDOR:

Post ex tubation stridor typically occurs in people who have been intubated for several days, with a prevalence of upto 37% of intubated patients in the intensive care unit[67]. As many as 80% of patients who develop the complication require reintubation-prolonging their stay in the icu and increasing their risk for complications, such as nosocomial pneumonia. Some practitioners use a cuff leak test in adults to decide whether to use steroids before extubation[67]. If there is a small difference between exhaled tidal volume with the cuff inflated versus deflated, inflammation likely is present. In these cases, physicians often use steroids to reduce inflammation before the endotracheal tube is removed. But because steroids have adverse effects, including hypertension and hyperglycemia, they should be avoided unless necessary.

STEROID AND ANAPHYLAXIS:

Anesthesiologists use a myriad of drugs during the provision of an anesthetic. Many of these drugs have side effects that are dose related, and some lead to severe immune-mediated adverse reactions. Anaphylaxis is the most severe immune-mediated reaction; it generally occurs on reexposure to a specific antigen and requires the release of proinflammatory mediators. Symptoms may include all organ systems and present with bronchospasm and cardiovascular collapse in the most severe cases. Management of anaphylaxis includes discontinuation of the presumptive drug (or latex) and anesthetic, aggressive pulmonary and cardiovascular support, and epinephrine. The incidence of anaphylaxis during anesthesia is very difficult to estimate but has been calculated to range from 1 in 3,500[73].

A survey of anaphylaxis during anesthesia demonstrated that cardiovascular symptoms (73.6%), cutaneous symptoms (69.6%), and bronchospasm (44.2%) were the most common clinical features[73]. Muscle relaxants are associated with the most frequent incidence of anaphylaxis, and over the last two decades, natural rubber latex (NRL, or cis-1,4-polyisoprene) has emerged as the second most common cause of anaphylaxis[74].

Corticosteroids can decrease the airway swelling and prevent recurrence of symptoms, as seen in biphasic or protracted anaphylaxis[75]. Hydrocortisone is the preferred steroid because it has a fast onset of action. Glucocorticoids can supplement primary therapy to suppress manifestations of allergic diseases of limited duration like hay fever, serum sickness, urticaria, contact dermatitis, drug reactions, bee stings and angioneurotic odema. In mild forms of allergy anti histaminics are the first choice of drugs. In very severe forms, iv methylprednisolone 125mg, 6th hourly or equivalent dose can be used to obtain relief.

CEREBRAL ODEMA:

Steroids are of value in reduction or prevention of cerebral edema associated with parasitic infections or neoplasms[76]. The mechanism by which steroids influence vasogenic edema are thought include one or more of the following :

- Stabilisation of cerebral endothelium, leading to decrease in plasma filtration.
- Increase in lysosomal activity of cerebral capillaries.
- Inhibition of release of potentially toxic substances such as free radicals, fatty acids and prostaglandins.

- Electrolyte shifts favoring transcapillary efflux of fluid.
- Increase of local and global cerebral glucose use, leading to improved neuronal function.

In the management of patient with malignant brain tumor, it is not uncommon for subjects who are somnolent or stuporous on admission to respond within hours to loading dose of dexamethasone (8-32 mg) and appear alert and without neurological deficits by the following day[76,77].

Controversy exists regarding response to steroid use in closed head injury. As prolonged steroids are associated with various side effects, among them hyperglycemia and increased predisposition to infection which are harmful in head injury patient. So, use of steroids in traumatic brain injury is considered obsolete[49].

CARDIAC ARREST:

Cardiac arrest causes global cerebral ischemia resulting in intracellular cytotoxic edema[47,49]. Dexamethasone(0.5-1mg/kg) is given initially followed by 4-8mg 6th hrly but methylprednisolone may be as effective[78] .The efficacy of steroids in treatment of perifocal vasogenic odema as occurs in intrinsic mass lesions is well established but its value in intracellular cytotoxic edema is still unproved. In deed steroids havenot been shown to improve survival or neurologic recovery rate after arrest and there administration is not recommended[49].

CONCLUSION:

As discussed in the review, there is an increasing application of steroid therapy during perioperative period for various purposes. Because they exert effects on almost every organ system, the clinical use of and withdrawal from corticosteroids are complicated by a number of serious side effects, some of which are life-threatening. Therefore, the decision to institute therapy with corticosteroids always requires careful consideration of the relative risks and benefits in each patient. With improved medical and diagnostic facilities, it is more likely for any anaesthesiologist to encounter patients on long/short term steroid therapy. Patients on long term steroids are a challenge to any anaesthesiologist. It's incumbent on us to become familiar with all aspects of steroid physiology, functions, adverse effects to avoid potentially life threatening situations in the perioperative period.