

Steroids in neurosurgery

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Moderators

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History

- 1855 – Addison's disease
- 1856 – Adrenal glands essential for life
- 1930 – Cortex > medulla
- 1932 – Cushing's syndrome
- 1949 – Hench et al (Steroids in rheumatoid arthritis)
- 1952 – Aldosterone

Synthesis

Stimuli	Part	Principal product
Angiotensin II	Zona glomerulosa	Aldosterone
ACTH	Zona fasciculata & reticularis	Cortisol Adrenal androgens
Sympathetic nervous system	Medulla	Adrenaline & Nor-adrenaline

Basal secretions

Group	Hormone	Daily secretions
Glucocorticoids	<ul style="list-style-type: none"> • Cortisol • Corticosterone 	<p>5 – 30 mg</p> <p>2 – 5 mg</p>
Mineralocorticoids	<ul style="list-style-type: none"> • Aldosterone • 11- deoxycorticosterone 	<p>5 – 150 µg</p> <p>Trace</p>
Sex Hormones	<ul style="list-style-type: none"> • DHEA • Progesterone • Oestradiol 	<p>15 – 30 mg</p> <p>0.4 – 0.8 mg</p> <p>Trace</p>

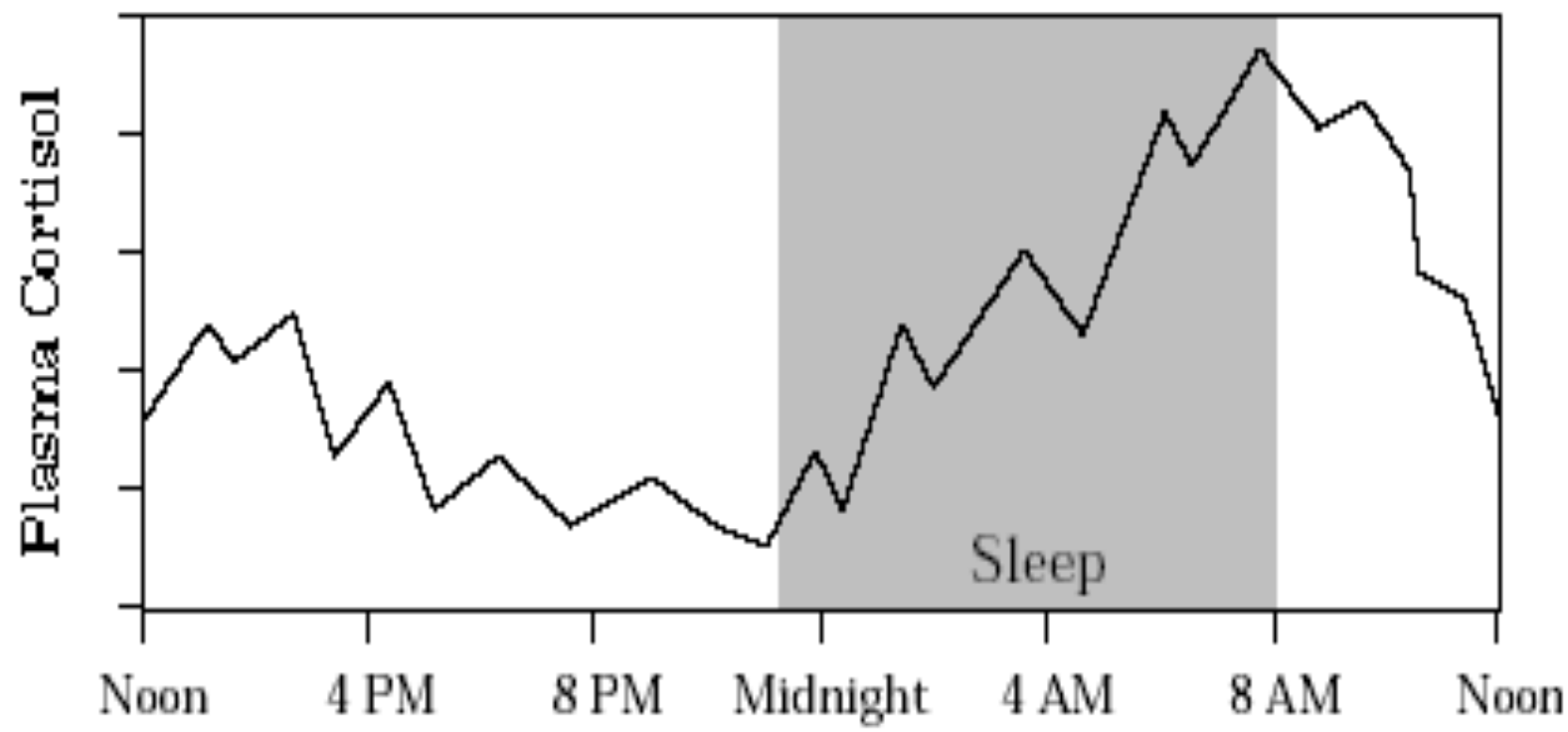
From Essential of Pharmacotherapeutics, ed. FSK Barar. P.351

Preparations

Drug	Anti-inflam.	Salt retaining	Topical
Cortisol	1	1.0	1
Cortisone	0.8	0.8	0
Prednisone	4	0.8	0
Prednisolone	5	0.3	4
Methylpredni- solone	5	0	5
Intermediate acting			
Triamcinolone	5	0	5
Paramethasone	10	0	-
Fluprednisolone	15	0	7

Preparations

Drug	Anti-inflam.	Salt retaining	Topical
Long acting			
Betamethasone	25-40	0	10
Dexamethasone	30	0	10
Mineralocorticoids			
Fludrocortisone	10	250	10
DOCA	0	20	0



Pharmacological Actions

- Direct (Intended) Actions
 - Anti-inflammatory
 - Anti-allergy
 - Anti-immunity
- Permissive Actions
 - Lipolytic effects
 - Effect on bp
 - Effect on bronchial muscles
 - (*e.g.*, sympathomimetic amine)










Pharmacological Actions

- For most clinical purposes, synthetic glucocorticoids are used because they have a higher affinity for the receptor, are less activated and have little or no salt-retaining properties.
- **Hydrocortisone** used for: orally for replacement therapy, i.v. for shock and asthma, topically for eczema (ointment) and enemas (ulcerative colitis).
- **Prednisolone** the most widely used drug given orally in inflammation and allergic diseases.

Pharmacological Actions

- **Dexamethasone**: very potent, w/o salt-retaining properties; thus, very useful for high-dose therapies (*e.g.*, cerebral edemas).
- **Beclometasone, dipropionate, budesonide**: more active when applied topically
- **Triamcinolone**: local joint inflammation (intra-articular inj.).

Actions: **Anti-inflammatory**

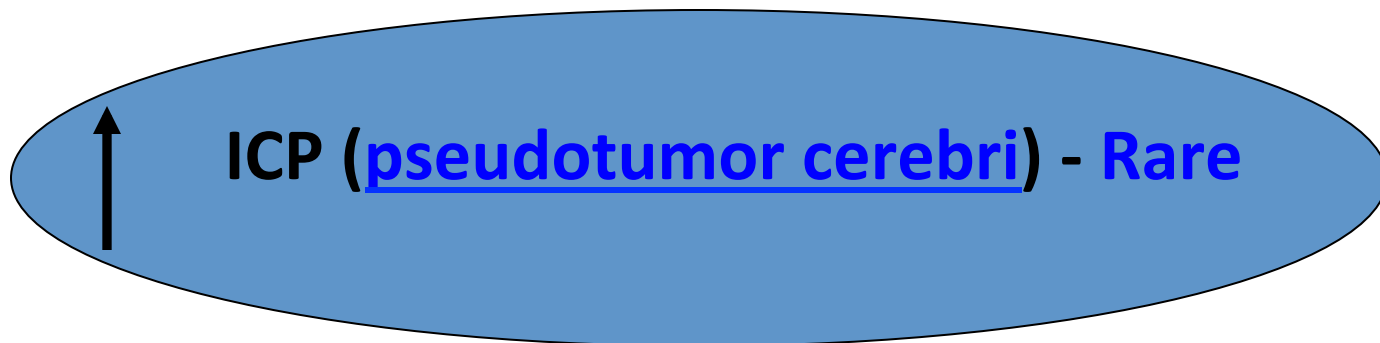
-  Recruitment of WBC and monocyte-macrophage into affected area & elaboration of chemotactic substances
-  Lipocortin
-  ELAM1 and ICAM-1 in endothelial cells
-  TNF from phagocytic cells
-  IL1 from monocyte-macrophage
-  Formation of Plasminogen Activator
-  Action of MIF and  fibroblastic activity
-  Expression of COX II

Pharmacological Actions

1. Carbohydrate
2. Protein
3. Lipid
4. Electrolyte and H₂O
5. CVS
6. Skeletal Muscle
7. CNS
8. Stomach
9. Blood
10. Anti-inflammatory
11. Immunosuppressant
12. Respiratory system
13. Growth and Cell Division
14. Calcium metabolism

Actions: CNS

- Direct:
 - Mood
 - Behaviour
 - Brain excitability
- Indirect:
 - maintain BBB, circulation and electrolyte balance



Role in neurosurgery

Role in traumatic spine and head injuries

Pathophysiology of Spinal Cord injury

Primary mechanisms

Initial crush, shear impingement of cord with the inciting trauma.

Secondary mechanisms

Vascular insults/insufficiency

Edema

Cell toxicity

Apoptosis

Secondary Mechanisms

- Electrolytes
 - Calcium release
- Cell toxicity
 - Glutamate release, arachidonic acid metabolites, free radical generation
- Apoptosis
 - Programmed cell death
- Vascular
 - Dysautoregulation, hypotension, neurogenic shock

Secondary mechanisms

- Numerous mediators of spinal cord damage have been identified experimentally.
- The hope is that through simple pharmacologic interventions, the secondary damage can be limited, or even potentially reversed.
- Unfortunately very little clinical progress has been made to date.

Steroids

- Several studies have reported success with high dose steroid infusions, limiting progression of spinal cord damage in trauma.
 - NASCIS II and III (NEJM 1990, JAMA 1997)
 - Two highly publicized studies demonstrating small but clinically significant improvement with neurologic recovery following administration of high dose methylprednisolone
 - NASCIS II placebo controlled
 - NASCIS III dose varied. Not placebo controlled

NASCIS II(National acute spinal cord injury study)

- Steroid bolus 30mg/kg over 15min in 1st hour, then 5.4mg/kg/hr for 23 hours
 - An average 70Kg patient would receive 23 GRAMS of steroid over 24 hours
- NASCIS II was in fact a negative study.
 - Only on post hoc sub group analysis did steroid yield a “benefit”
 - Only patients who received steroid in the first 8 hours post injury demonstrated a benefit
- What degree of benefit however?

NASCIS II

- Unfortunately, the degree of “statistically significant benefit” has no clinical relevance
- Motor score improvements were 17.2 and 12.0 for steroid and placebo groups respectively (out of a total possible score of 70), which gives a difference of 5.2.
 - A difference of 5.2 simply put could be gained if a patient regained the ability to shrug his shoulders.

NASCIS 3 Results

- Initiation of treatment within the first 3 hours is optimal
- The nonglucocorticoid tirilazad is as effective as 24-hour MP therapy; and
- If treatment is initiated more than 3 hours post-SCI, extension of the MP dosing regimen is indicated, from 24 hours to 48 hours.

Results

- However, in comparison with the 24-hour dosing regimen, significantly more glucocorticoid-related immunosuppressive side effects were seen with more prolonged dosing
- In contrast, tirilazad showed no evidence of steroid-related side effects, suggesting that this nonglucocorticoid 21-aminosteroid would be safer for extension of dosing beyond the 48-hour limit used in NASCIS III.

Suggested Indications for the Use of MP in Acute SCI

- **For acute non-penetrating SCI (<3 h after injury)**, MP should be given as per NASCIS II protocol (i.e. 24 hours of treatment)
- **For acute non-penetrating SCI (>8 h after injury)**, MP should not be used
- **For acute non-penetrating SCI (after 3 h, within 8 h)**, MP should be given as per NASCIS III protocol (i.e., 48 h of treatment)
- **For acute penetrating SCI, MP is not recommended**

Controversies

- Comparison of 24-h vs. 48-h MP dosing
 - No placebo group for comparison
 - Increased rates of wound infection/severe sepsis
 - No significant benefit at 1 year follow up
 - Benefits noticed in post hoc analysis only
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- Fehlings MG. Editorial: recommendations regarding the use of methylprednisolone in acute spinal cord injury: making sense out of the controversy. *Spine* 26[Suppl 24]: 56–57, 2001.

Conclusion

- Bracken et al
- “Given the devastating impact of SCI and the evidence of a modest, beneficial effect of MP, clinicians should consciously consider using this drug despite the well-founded criticisms that have been directed against the NASCIS II and III trials. With great understanding of the biomolecular events contributing to the pathogenesis of SCI, it is hoped that other neuroprotective agents will enter into clinical practice in the next 5-10 years”

Important Papers

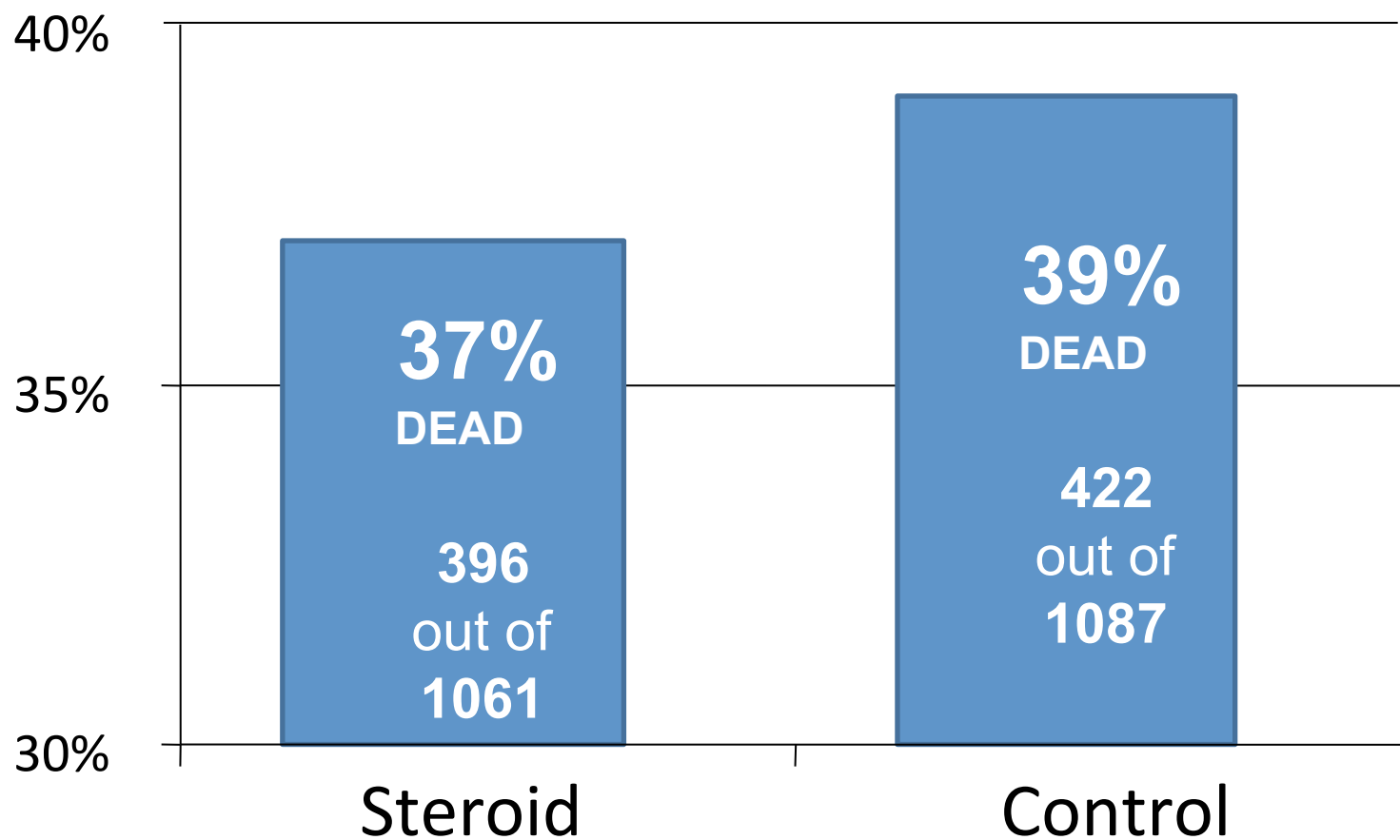
- NASCIS II
 - NEJM 1990 322:1405-11
- NASCIS III
 - JAMA 1997 277:1597-1604
- Revisiting NASCIS II & III
 - J. Trauma 1998 45:6 1088-93
- Methylprednisolone for acute spinal injury....
 - J. Neurosurg (Spine 1) 2000:93:1-7

- Head injury

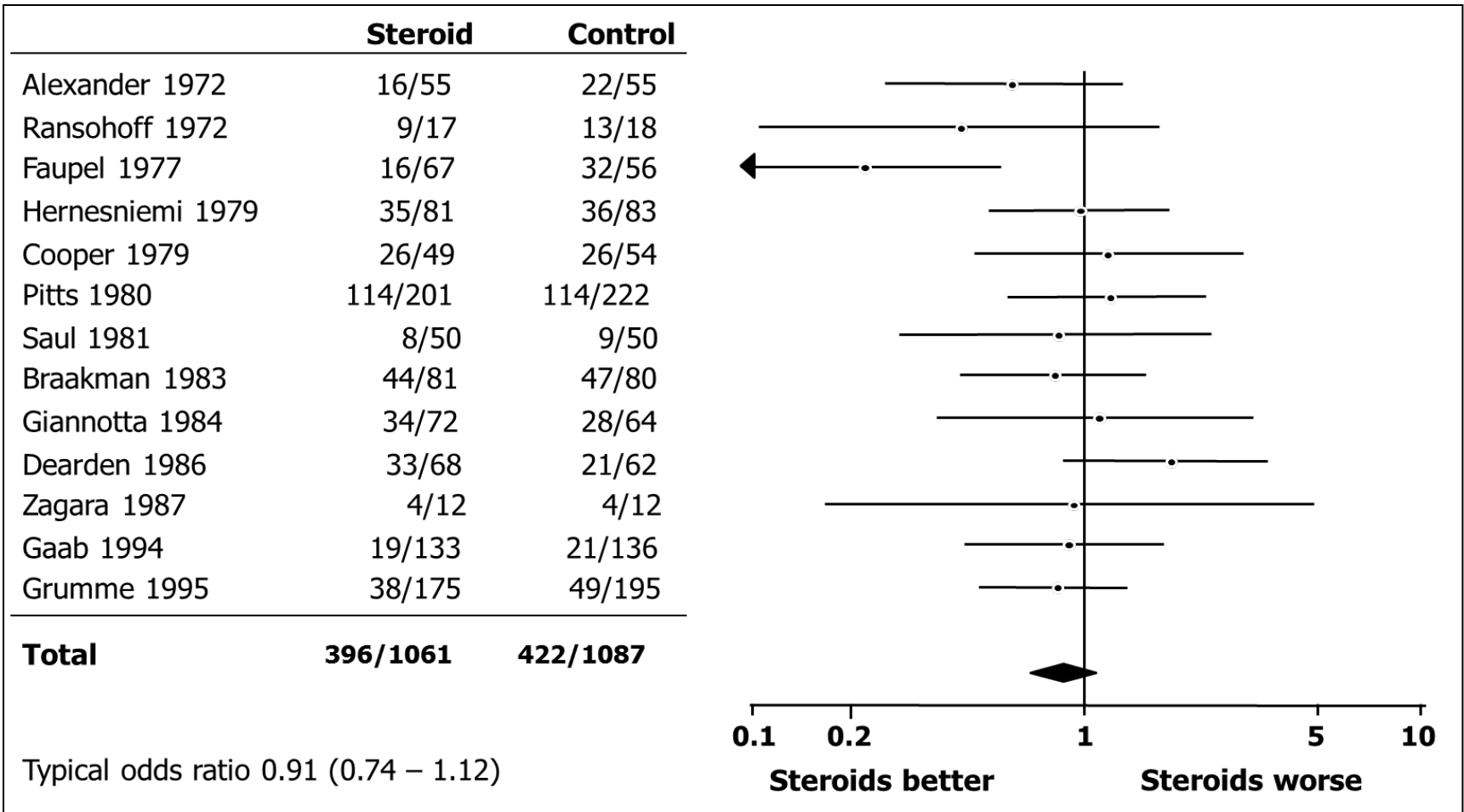
CRASH

- Corticosteroid Randomisation After Significant Head Injury
- A large simple placebo controlled trial, among adults with head injury and impaired consciousness
- Studied the effects of a 48-hour infusion of corticosteroids on death and neurological disability

Aggregate mortality from 13 randomised trials of steroids in head injury



Meta-analysis of mortality in previous trials of steroids in head injury



Aim

- To determine reliably the effects of high dose corticosteroid infusion on:
 - death and disability after head injury
 - risk of infection and gastrointestinal bleeding
- Primary outcomes
 - Death within 2 weeks
 - Death or disability at 6 months

Results

- 10,008 adults with head injury and a Glasgow Coma Scale score of 14 or less, within 8 h of injury
- 48-h infusion of corticosteroid (methylprednisolone) or placebo.
- Data at 6 months were obtained for 9673 (96.7%) patients.

Results

- The risk of death was higher in the corticosteroid group than in the placebo group (1248 [25.7%] vs 1075 [22.3%] deaths; relative risk 1.15, 95% CI 1.07-1.24; $p=0.0001$)
- There was no evidence that the effect of corticosteroids differed by injury severity or time since injury.
- These results lend support to earlier conclusion that corticosteroids should not be used routinely in the treatment of head injury

Discussion

- Landmark for head injury research
- Also a red flag warning clinical trials
- Planned to randomize 20,000 patients
- Powered to detect a drop in mortality from 15% to 13% (?? Only 2%)
- Document relatively small but important improvements in outcome

- CRASH data safety monitoring board asked that the trial be stopped after 10,008 patients from 239 hospitals in 49 countries had been randomized

WHY ??

- No evidence that MPSS improved survival
- Mortality was greater in the MPSS treated patients
- 1248 [25.7%] vs 1075 [22.3%]
- Relative risk 1.15, $p=0.0001$
- Risk could not be attributed to injury

References

- Final results of MRC CRASH, a randomized placebo-controlled trial of intravenous corticosteroid in adults with head injury—outcomes at 6 months. **Lancet** 365: 1957–1959, 2005
- Alderson P, Roberts I: Corticosteroids for acute traumatic brain injury. **Cochrane Database Syst. Rev** 2:CD000196, 2000.
- CRASH (CORTICOSTEROID RANDOMIZATION AFTER SIGNIFICANT HEAD INJURY TRIAL): LANDMARK AND STORM WARNING. *Neurosurgery* 57:1300-1302, 2005

- Brain tumors
 - Pituitary adenoma
 - Gliomas
 - Meningiomas
- Brain edema
- Spinal pathologies
- Vascular

- Pituitary adenoma

Glucocorticoid Replacement in Pituitary Surgery: Guidelines for Perioperative Assessment and Management

- The first decision regarding the perioperative use of glucocorticoid cover depends on the result of the **preoperative screening test**
- **Abnormal ACTH 1–24 (Synacthen) test.**
 - dose regimen, using hydrocortisone, is
 - 50 mg every 8 h on d 0,
 - 25 mg every 8 h on d 1, and
 - 25mg at 0800 h on d 2.
 - alternative regimen, using dexamethasone
 - 4mg at induction of anesthesia,
 - 2 mg at 0800 h on d 1, and
 - 0.5 mg at 0800 h on d 2.
 - Dexamethasone has the advantage of a long half-life, which allows for single daily administration.
- Individuals with normal HPA axis function show a return to baseline cortisol secretion within 48 h of major surgery, providing that no complications have occurred*

* Salem M, Tainish RE, Bromberg J, Loriaux DL, Chernow B 1994 Perioperative glucocorticoid coverage. A reassessment 42 years after emergence of a problem. Ann Surg 219:416–425

Pre-operative

- **Normal ACTH 1–24 (Synacthen) test.**
- If neurosurgeon is able to perform a selective adenectomy
 - no perioperative glucocorticoid cover be given
 - There is good evidence that such an approach is safe in this patient group
- If selective adenectomy is not possible and surgery is more extensive
 - Glucocorticoid cover for 48 h and proceed as above.
 - Otherwise, we recommend measuring 0800 h plasma cortisol on d 1–3 postoperatively. Again, the subsequent use of glucocorticoid replacement depends on these results

Post-operative

- Many patients show a rapid increase in pituitary hormone secretion after pituitary adenectomy
- Result of increased flow of hypothalamic releasing hormones through the hypothalamic-hypophyseal portal system
- A patient with hypopituitarism may gain full functional recovery, providing that viable normal pituitary tissue remains *in situ*.
- Prolonged treatment with high doses of glucocorticoids postoperatively may result in adrenal suppression and mask those who spontaneously recover function.

Post-operative

- Provided there are no postoperative complications, **glucocorticoid supplementation should be withdrawn, after 48 h**
- **Measurement of 0800 h plasma cortisol levels performed daily between d 3 and 5 postoperatively**
- Daily clinical assessment of the patient, together with these cortisol results, will determine the subsequent use of glucocorticoid replacement therapy

ACTH-secreting adenomas

- Cushing's disease presents a special situation, with all patients requiring perioperative glucocorticoid cover.
- The presence of low plasma cortisol levels postoperatively implies a surgical cure
 - Patients with low plasma cortisol levels after pituitary surgery for Cushing's disease require physiologic hydrocortisone replacement therapy with ongoing review of their HPA axis.
 - The aim is to gradually withdraw the hydrocortisone as the ACTH suppression recovers.

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ACTH-secreting adenomas

- Normal plasma cortisol levels postoperatively usually indicate ongoing autonomous ACTH secretion.
- The clinician must decide whether early repeat surgery is indicated or use an alternative modality of treatment, such as radiotherapy or adrenolytic therapy.
- Estimation of 24-h urinary free cortisol may help in this decision and may be performed while receiving physiological replacement with dexamethasone (0.5–0.75 mg daily).
- The CRH test and ACTH 1–24 test have also been used in this setting but, overall, seem to add little to the basal measurements of plasma and urinary free cortisol in predicting relapse.

Early postoperative assessment

- Based on the levels of 0800 h plasma cortisol in the early postoperative period
- These measurements should be made on
 - d 1–3 in patients not treated with glucocorticoids and
 - d 3–5 in patients covered with glucocorticoids for the initial 48 h

Guidelines for the further investigation and management of glucocorticoid replacement based on postoperative 0800 h plasma cortisol levels

0800-h Cortisol level (nM)	Glucocorticoid replacement (hydrocortisone equivalent)	Definitive test required
< 100	15–30 mg/d (maintenance)	No—deficient HPA axis
100–250	10–20 mg, single morning dose	Yes
250–450	Stress only, safe to withhold routine replacement until definitive test	0800 h cortisol d 7 Yes if 350 nM
> 450	No	No—normal HPA axis

Who needs a definitive test of their HPA axis?

- Only in those patients with postoperative 0800 h cortisol levels between 100 and 350 nm
- A repeat 0800 h plasma cortisol at 4–6 wk, 24 h after the last dose of hydrocortisone, may provide additional evidence for normal HPA function.
- Plasma cortisol levels more than 350 nm are highly suggestive of normal pituitary-adrenal function
- Those patients with levels less than 100 nm are likely to remain ACTH deficient

Who needs a definitive test of their HPA axis?

- There are cases where late recovery of the HPAaxis is documented
- A repeat 0800h cortisol, 24 h after the last dose of hydrocortisone, performed between 4 and 6 wk postoperatively, should identify such patients.
- Those still less than 100 nm require lifelong replacement therapy, whereas those whose cortisol levels have risen to more than 100 nm should undergo a definitive test and replacement

Pituitary apoplexy

- **Steroid therapy in pituitary apoplexy**
- Patients with pituitary apoplexy, who are hemodynamically unstable
- Empirical steroid therapy.
- **In adults hydrocortisone 100-200mg as an intravenous bolus is appropriate, followed by**
- 2-4mg per hour by continuous intravenous infusion or 50-100mg six hourly by intramuscular injection, after drawing blood samples for random cortisol, FT4, TSH, PRL, IGF1, GH, LH, FSH, testosterone in men, oestradiol in women, electrolytes, renal function, liver function, full blood count and clotting screen;
- Indications for empirical steroid therapy in patients with pituitary apoplexy are
 - haemodynamic instability,
 - Altered consciousness level,
 - reduced visual acuity and
 - severe visual field defects
- Patients who do not fulfil the criteria for urgent empirical steroid therapy should be considered for treatment with steroids, if their 09.00 serum cortisol is less than 550nmol/l;

Orders in neurosurgery

- Pre-op
 - T eptoin
 - T decdac
 - T rantac
 - C b/c
- Post-op
 - Inj phenytoin
 - Inj clox
 - Inj amikacin
 - Inj wymesone
 - Inj rantac
 - Inj ketonov

History of use of steroid in tumor

- Steroids were introduced into the care of brain cancer patients nearly 50 years ago based on their powerful effects on tumor-induced edema
- Ingraham pioneered the use of cortisone to treat postoperative cerebral edema in neurosurgical patients in 1952
- Kofman first used prednisone for peritumoral edema from brain metastases in 1957
- More than 40 years ago, Galicich demonstrated that dexamethasone effectively alleviated cerebral edema due to brain tumors

- Dexamethasone, which was first synthesized in 1958, to date has remained the most favorable drug for brain cancer patients.
- Dexamethasone has become the drug of choice in neuro-oncology, in part owing to its long half-life, low mineralocorticoid activity, and a relatively low tendency to induce psychosis.
- Subsequently, prednisone, prednisolone, dexamethasone and methylprednisolone have demonstrated antineoplastic effects against malignancies

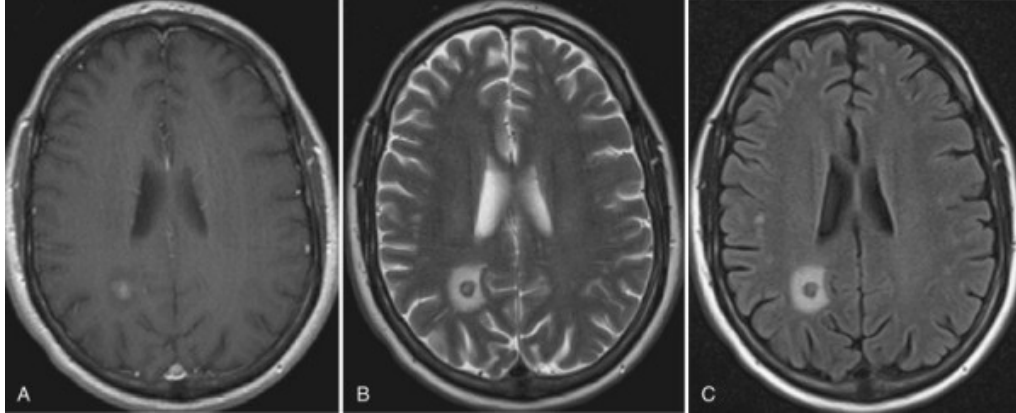
Disruption of the BBB

- Trauma
- Inflammatory and autoimmune disease,
- Infection
- Cerebrovascular disease
- Neurodegenerative disease
- Epilepsy, and
- Neoplasia

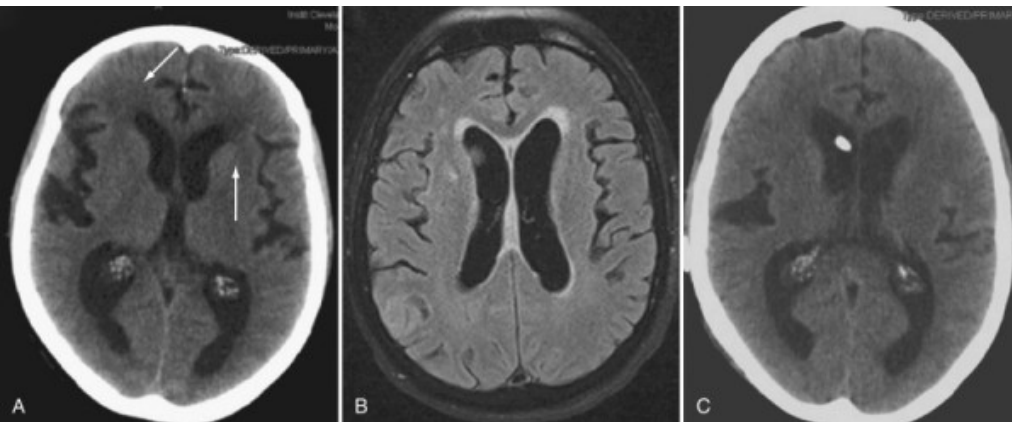
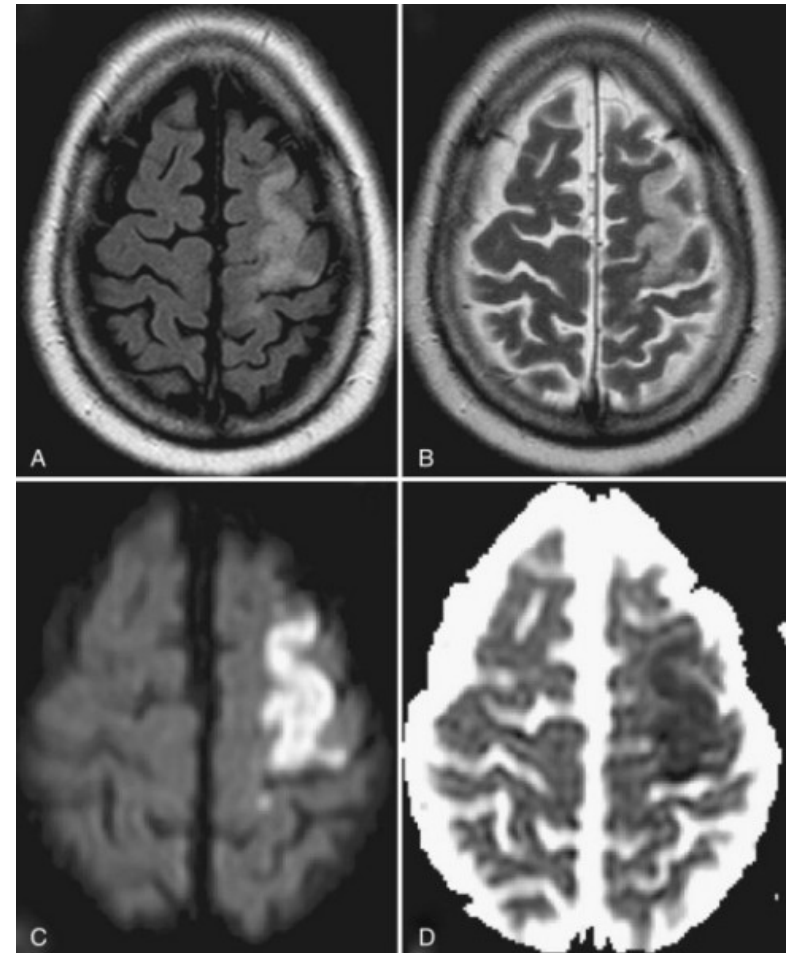
Disruption of the BBB

- Klatzo first characterized brain edema as a cytotoxic versus a vasogenic process
- BBB remains intact in cytotoxic edema
- Corticosteroid therapy after brain tumor surgery helps re-establish the BBB integrity.
- Cairncross JG, Macdonald DR, Pexman JH, et al:
[Steroid-induced CT changes in patients with recurrent malignant glioma.](#) *Neurology* 1988;
38:724-726

Cerebral edema



1. Vasogenic edema
2. Cytotoxic edema
3. Interstitial edema



Steroids

- Glucocorticoids primarily reduce vascular permeability of vessels rather than reduced VEGF production
- This inhibition of the effects of VPF/VEGF on the vasculature is associated with interference of VEGF's action on vessels and requires the glucocorticoid receptor
- Thus, the effects of steroids in tumor-induced vasogenic edema is to restrict permeability of the BBB to macromolecules
- By contrast, steroids are not effective when the BBB is not functional

Heiss JD, Papavassiliou E, Merrill MJ, et al:

[Mechanism of dexamethasone suppression of brain tumor-associated vascular permeability in rats. Involvement of the glucocorticoid receptor and vascular permeability factor. *J Clin Invest* 1996; 98:1400-1408.](#)

. Merrill MJ, Oldfield EH: [A reassessment of vascular endothelial growth factor in central nervous system pathology. *J Neurosurg*](#)

Cranial surgery: Brain tumor

- Several days before surgery when there is **symptomatic brain edema or mass effect**
- Typical dose is 16 mg/day or 4 mg four times a day
- In brain edema, there appears to be a dose threshold that must be surpassed before symptomatic benefit is derived
- Therefore, dexamethasone should be started at a high dose in patients with large amounts of brain edema and then reduced after neurological improvement has occurred.

Pre-op

- Patients respond within 24 hours of beginning steroid treatment
- Decrease tumor capillary permeability and tumor blood volume
- In patients undergoing more elective surgery, such as those with schwannomas and meningiomas, may be given steroids beginning 3 days before surgery
- However, if there is only minimal edema, steroids are typically first given at time of surgery

Post-op

- Patients at risk for the development of significant postoperative edema are:
 - deep intrinsic tumors in which only minimal resection was possible
 - infiltrating tumors involving a large amount of white matter, and
 - extensive edema before surgery.
 - progressive cerebral edema (can also be treatment related)
- Maximal postoperative swelling occurs between days 1 and 5 after surgery

Post -op

- Regardless of the cause of the cerebral edema, management is similar
- These patients should receive maximal medical treatment of the edema and elevated ICP
- Steroids should be given at maximal doses (20 mg every 4 hours).

Yamada K, Ushio Y, Hayakawa T, et al: [Effects of methylprednisolone on peritumoral brain edema. J Neurosurg 1983; 59:612-619.](#)

Leenders KL, Beaney RP, Brooks DJ, et al:

[Dexamethasone treatment of brain tumor patients: effects on regional cerebral blood flow, blood volume, and oxygen utilization. Neurology](#)

Metastasis

- Believe to exert their effect by reducing cerebral edema surrounding tumor, and to lesser extent there may be direct oncolytic effect on tumor cells
- Ameliorate sign and symptom in 2/3rd of patients
- Benefit evident within 24 hrs.
- Minimum effective dose is not known but in practice loading dose equivalent to 10 mg of dexamethasone intravenously, followed by 16 mg orally/IM (divided in 3-4 doses)
- Steroids tends to stabilise patient ,after which more definitive treatment (surgery/radiotherapy) may be undertaken. May be continued through initial phase and tapered and finally discontinued after 1-2 weeks.
-
- Relief is only temporary. Patient tends to relapse despite continued steroid administration

Renaudin, J., Fewer, D., Wilson, C. B., Boldrey, E. R., Calogero, J., and Enot, K. J. Dose dependency of Decadron in patients with partially excised brain tumors. *J. Neurosurg.*, 39: 302–305, 1973.

Brain metastasis

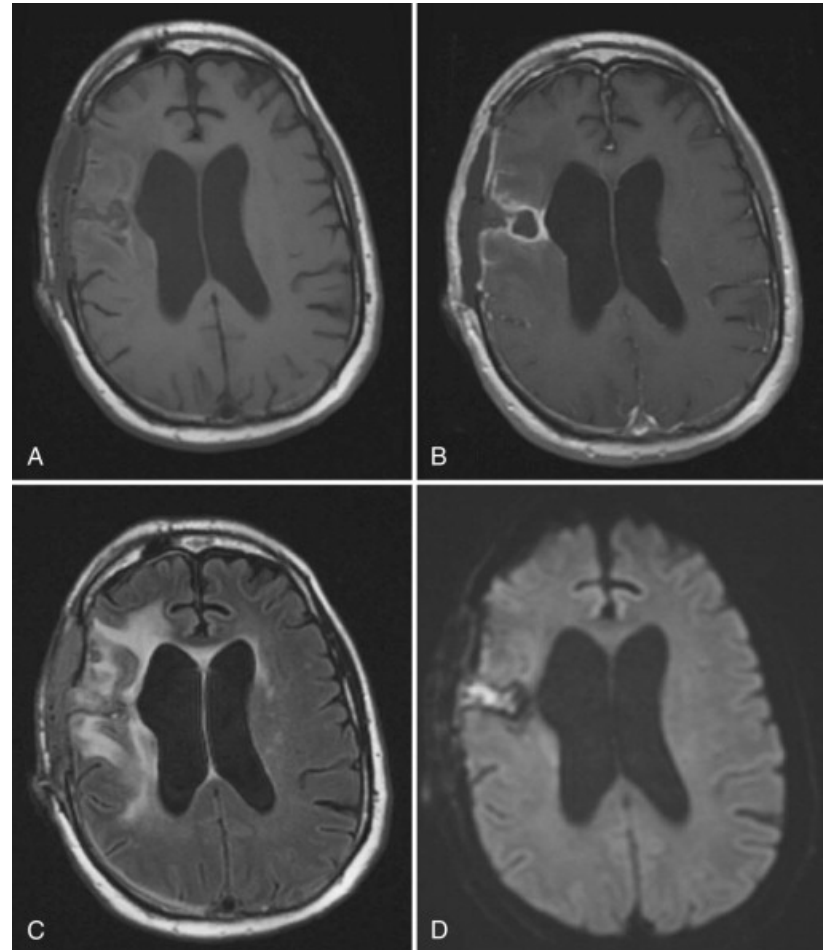
- All patients should receive steroids.
- Rapid improvement within 6-24 hours.
- Maximum effect by 3-7 days.
- Benefit is temporary.

Dose of Steroids

- Most common dose dexamethasone 4 mg qid.
- Doses as low as 4 mg qid if not symptomatic.
- Up to 100 mg per day if symptoms are progressing.
- Taper as tolerated.

Brain abscess

- Concurrent treatment with corticosteroids, radiation therapy, and chemotherapy may alter the radiographic progression of abscess development
- Corticosteroids have been shown to reduce the thickness of the abscess capsule and the extent of contrast enhancement on both CT and MRI
- [Cavusoglu H, Kaya RA, Turkmenoglu ON, et al: Brain abscess: analysis of results in a series of 51 patients with a combined surgical and medical approach during an 11-year period. *Neurosurg Focus* 2008; 24\(6\):E9](#)



Spinal Indications

- Control of pain in low back ache :Epidural injection in lumbar pain
- Epidural metastasis (acute phase)
- Neurological deterioration after implant migration and /or compression
- Rheumatoid arthritis

Miscellaneous

- Carpal syndrome
- Bell's palsy
- Cranial n. damage
- Chronic SDH
- Dexamethasone suppression test
- Neurodegenerative ds.
- Meningitis

Meningitis

- Reported in the 1950s
- Individuals with bacterial meningitis treated with antibiotics plus steroids within 12-24 h of admission had better outcomes
- Interestingly, these benefits were not observed if steroid treatment was delayed until 5 days after admission.
- Bacterial lysis induced by antibiotics causes inflammation in the subarachnoid space, and this response is attenuated by steroid treatment, thereby improving the outcome
- Hoh TK and Mong CT (1962) The treatment of purulent meningitis with adrenal cortico-steroids. *SingaporeMed J* 3: 73-77

Current Guidelines and Recommendations

- Should be administered just before or in conjunction with antibiotic therapy.*
- Dexamethasone (10 mg) should be administered intravenously every 6 h for 4 days in adults
- At present, children in low-income countries do not seem to derive appreciable benefit from steroid use (??)
- In most childhood studies, a 4-day regimen of dexamethasone (0.4 or 0.6 mg/kg per day) divided into four daily doses was used.
- We suggest that clinicians consider administering treatment with this 4-day regimen, as most published studies have used this duration of treatment without observing increased adverse effects relative to the shorter durations.

*van de Beek D and de Gans J (2006) Dexamethasone in adults with community-acquired bacterial meningitis. *Drugs* 66: 415-427

Meningitis: Tubercular

- **Current Guidelines and Recommendations***
- Stratified dexamethasone treatment regimen
- Patients with a Glasgow Coma Scale score of less than 15 or who have a focal neurological deficit are treated with
 - intravenous dexamethasone for 4 weeks
 - 0.4 mg/kg per day in week 1
 - 0.3 mg/kg per day in week 2
 - 0.2 mg/kg per day in week 3, and
 - 0.1 mg/kg per day in week 4, followed by
- Taper of oral dexamethasone(4 mg/day, 3 mg/day, 2 mg/day and 1 mg/day, each for a period of 1 week)

*Prasad K *et al.* Steroids for treating tuberculous meningitis. *Cochrane Database of Systematic Reviews* 2006, Issue 1. Art. No.: CD002244.doi:10.1002/14651858.CD002244.pub2

*Kumarvelu S *et al.* (1994) Randomized controlled trial of dexamethasone in tuberculous meningitis. *TuberLung Dis* 75: 203-207

Meningitis: Tubercular

- Patients with a normal mental status and no neurological findings receive
 - intravenous dexamethasone for 2 weeks
 - 0.2 mg/kg per day in week 1, then
 - 0.1 mg/kg per day in week 2, followed by
- The same oral taper as described above.
- steroid treatment should start as soon as possible after initiation of appropriate first-line anti tuberculosis drugs.

Conclusion

- Corticosteroids have varied roles in the pathologies of the brain and spine especially in neurooncology.

Thank you