

TIVA IN NEUROANAESTHESIOLOGY

GOALS IN NEUROANESTHESIOLOGY

- Haemodynamic stability
- ICP control
- Maintaining Cerebral perfusion
- Neuroprotection
- Providing optimal conditions for surgery
- Smooth emergence
- Rapid awakening

Ideal anaesthetic agent for neuroanesthesia

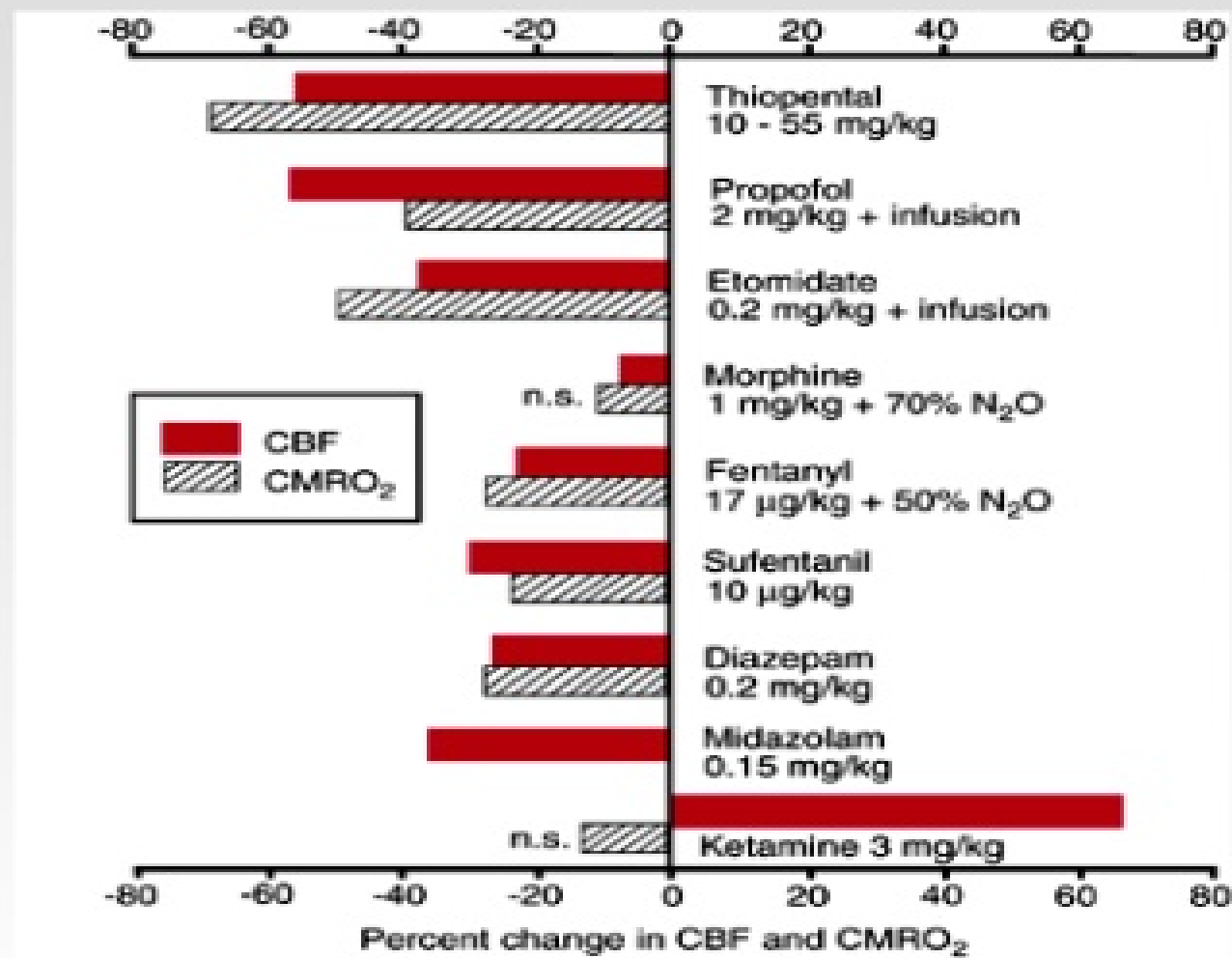
- Maintains CBF without altering the autoregulation.
- Minimizes or if not doesn't itself causes an increase the ICP.
- Preserves reactivity of cerebral arterioles to PaCO₂ changes.
- Decreases the CMRO₂ with cerebral protective effects
- Lacks seizure causing potential
- Lacks arrhythmogenicity

Ideal IV anaesthetic drug- pharmacodynamics

- Wide therapeutic ratio
- Minimal cardiorespiratory or motor side effects
- Rapid, predictable and smooth onset
- Painless and non irritant
- Stable at room temperature
- Rapid recovery (no rebound or emergence effects)
- No adrenal or immunosuppression

Low potential of anaphylaxis

Changes in (CBF) and the (CMRO₂) caused by I V agents



10/30/14

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TIVA IN NEURO- The Introduction

- Total intravenous anesthesia (TIVA) employs a sedative-hypnotic anesthetic combined with an analgesic agent (typically an opioid) .
- Intravenous (IV) adjuvants such as ketamine, dexmedetomidine, or lidocaine may be used in some patients to replace or minimize the total propofol or opioid doses
- Hence avoiding their side effects (eg, hypotension due to higher doses of propofol or postoperative nausea and vomiting [PONV] due to opioids).

- Propofol is most commonly selected as the sedative-hypnotic component of a TIVA technique
- Owing to its rapid onset and recovery; beneficial antiemetic, bronchodilatory, and anticonvulsant properties.
- Propofol is infused at 75 to 150 mcg/kg/minute, with titration according to individual requirements, the degree of noxious surgical stimulation, and coadministration of other anesthetic agents.
- An opioid is invariably employed as the analgesic component of a TIVA technique.

Advantages of TIVA

- Easily titratable
- Superior recovery profile
- Portable delivery systems(TCI)
- Lowered OT pollution
- Minimal risk of Malignant hyperthermia
- Less PONV
- Preserves HPV.
- Improves V/Q mismatch
- Preserves cerebral autoregulation.

A propofol-based TIVA technique may contribute to postoperative analgesia

- In a 2016 meta-analysis of 4520 patients undergoing noncardiac surgery in 31 trials, intraoperative use of propofol-based TIVA was associated with generally lower postoperative pain scores at rest and lower requirements for supplemental opioid analgesia compared with any inhalation-based anesthesia with a potent volatile agent

Feasibility for neuromonitoring

- Most of the IV agents have less effect on evoked potentials than potent volatile inhalation agents or N₂O.
- In particular, motor-evoked potentials (MEPs) are very sensitive to inhalation agents, while somatosensory-evoked potentials (SSEPs) are moderately affected and BAEPs are resistant to the effects of inhalation anesthetics.
- TIVA regimes help maintain the level of anesthesia during these critical monitoring periods in order to avoid confounding the interpretation of changes

Disadvantages of TIVA

- Blood concentrations of IV agents are not easily obtained (Vs inhalationals)
- While technology such as target-controlled infusions (TCI) may allow the prediction of propofol and opioid concentrations in either the plasma or at the effect site (ie, the brain)

However, these methods are not easily available in the developing world.

- Greater risk of intraoperative awareness.

Edge of TIVA over Inhalational

- Superior recovery profile
- Portable delivery systems(TCI)
- Lowered OT pollution
- Minimal risk of Malignant hyperthermia
- Less PONV
- Preserves HPV.
- Improves V/Q mismatch
- Preserves cerebral autoregulation

So , is TIVA superior to the gases??

- TIVA was widely used in neuro anaesthesia on the pretext that all the known anesthetic gases altered cerebral haemodynamics at therapeutic concentrations.
- With the advent of various studies suggesting that sevoflurane doesn't alter cerebral haemodynamics significantly at therapeutic doses for anaesthesia
- Led to a role reversal

The resurgence of Sevoflurane

- **Kaisiti et al** studied via PET study that the cerebral blood flow increase by a Sevoflurane of MAC 1.5 was comparable to the cerebral blood flow in patients receiving propofol in healthy volunteers. *Anesthesiology*. 2002 Jun;96(6):1358-70
- **Matta BF et al** studied the vasodilatory effects of sevoflurane and Isoflurane.
- They found that although both agents increased blood flow velocity in the middle cerebral artery at 0.5 and 1.5 MAC, the increase was significantly less during sevoflurane anesthesia. *Anesthesiology*. 1999 Sep;91(3):677-80

- **Holmstorm A et al** studied in animal models and concluded that Desflurane increases intracranial pressure more and sevoflurane less than isoflurane in pigs subjected to intracranial hypertension **J Neurosurg Anesthesiol. 2004 Apr;16(2):136-4**
- **JASON CHUI et al** in 2014 after metaanalysing 14 studies comprising 1819 patients concluded
“Propofol-maintained and volatile-maintained anesthesia were associated with similar brain relaxation scores, although mean ICP values were lower and CPP values higher with propofol-maintained anesthesia. There are inadequate data to compare clinically significant outcomes such as neurological morbidity or mortality.”

Can J Anesth/J Can Anesth (2014) 61:347–356

- **G. Magni et al** studied Emergence Time and Early Cognitive Function Between Sevoflurane–Fentanyl and Propofol–Remifentanyl in Patients Undergoing Craniotomy for Supratentorial Intracranial Surgery and concluded “ *there is no patient benefit of using total intravenous anesthesia with an ultra-short-acting opioid over the conventional balanced volatile technique in terms of recovery and cognitive functions.*”

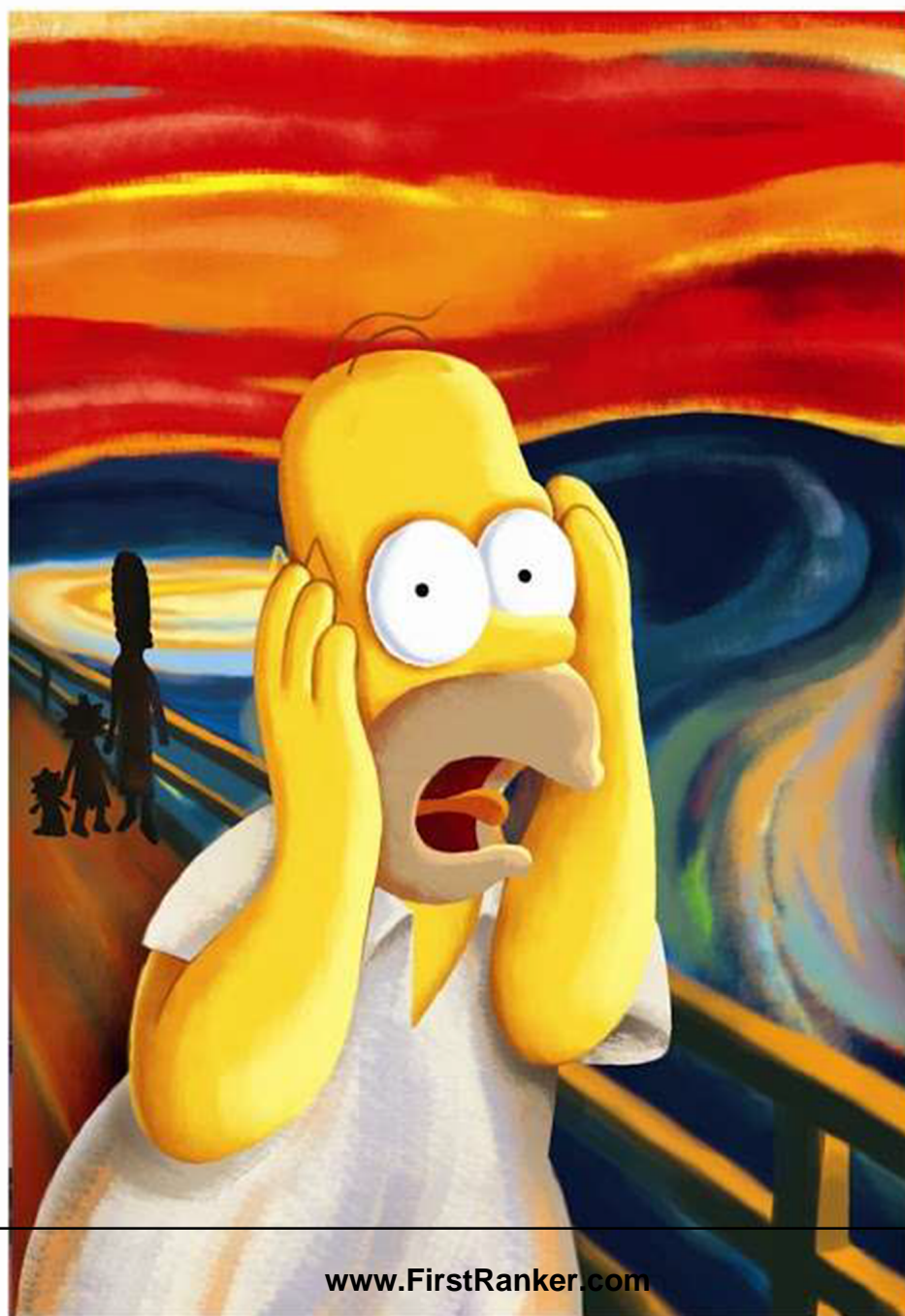
J Neurosurg Anesthesiol 2005;17:134–138)

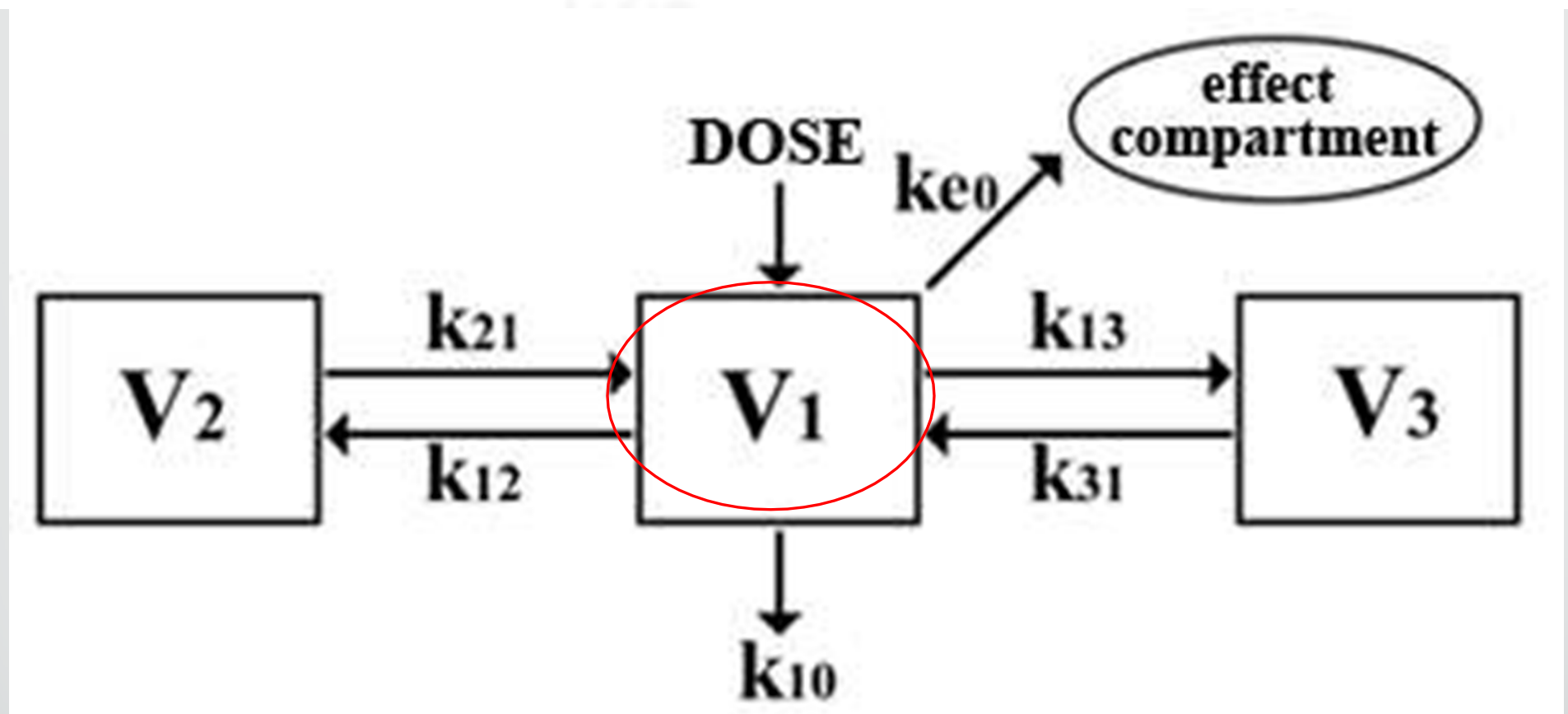
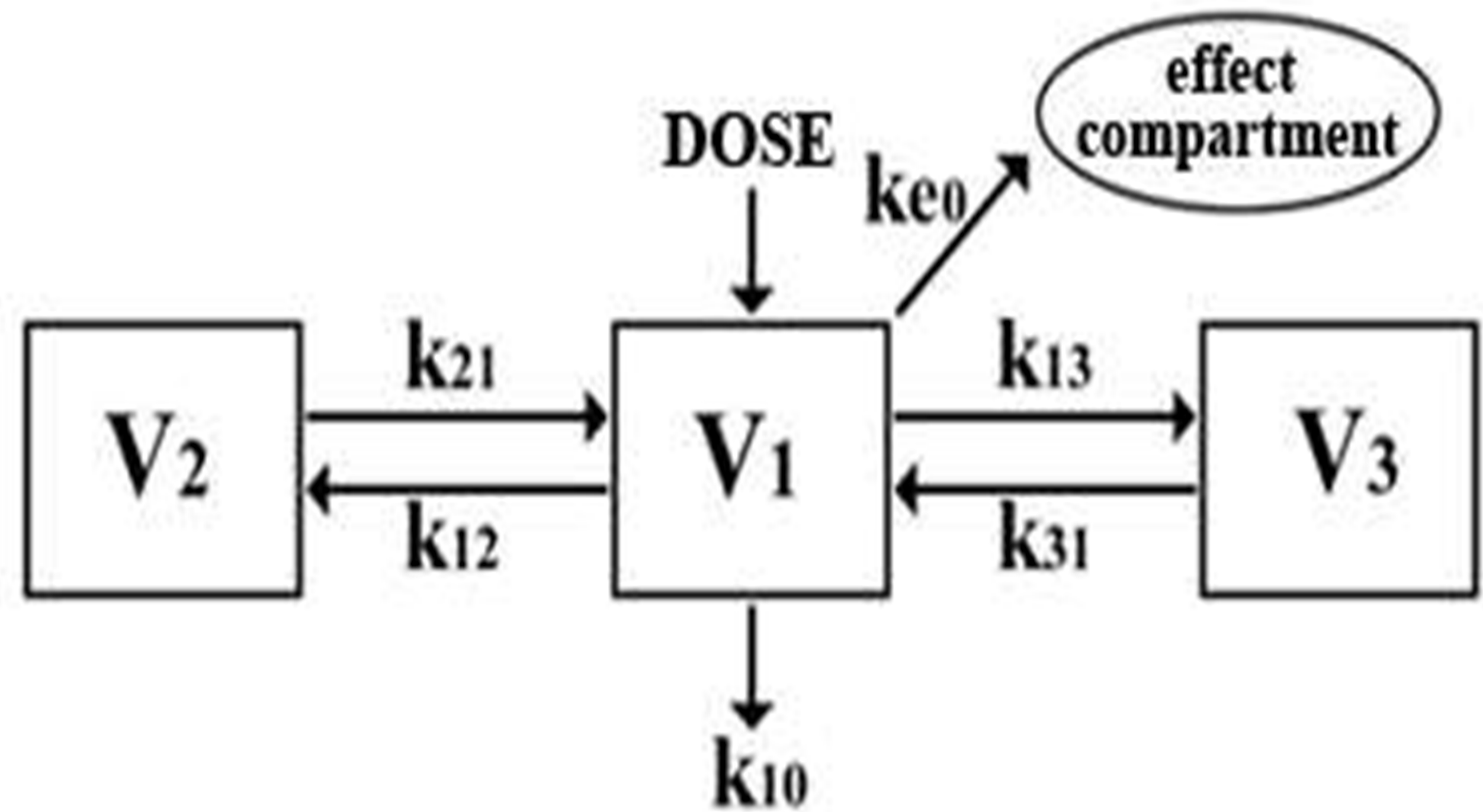
- Hence at this level in those patients with normal to mild raise in ICP , CBF increase by sevoflurane at therapeutic levels may not be as pronounced and clinically relevant as previously believed to be.

TIVA anaesthesia requirements

- Rapidly achieve an appropriate blood and brain concentration of the drug
- Maintain that concentration.
- Adjust the level as required (clinically / or if using a neuromonitor)
- Can use manual or automated infusions.

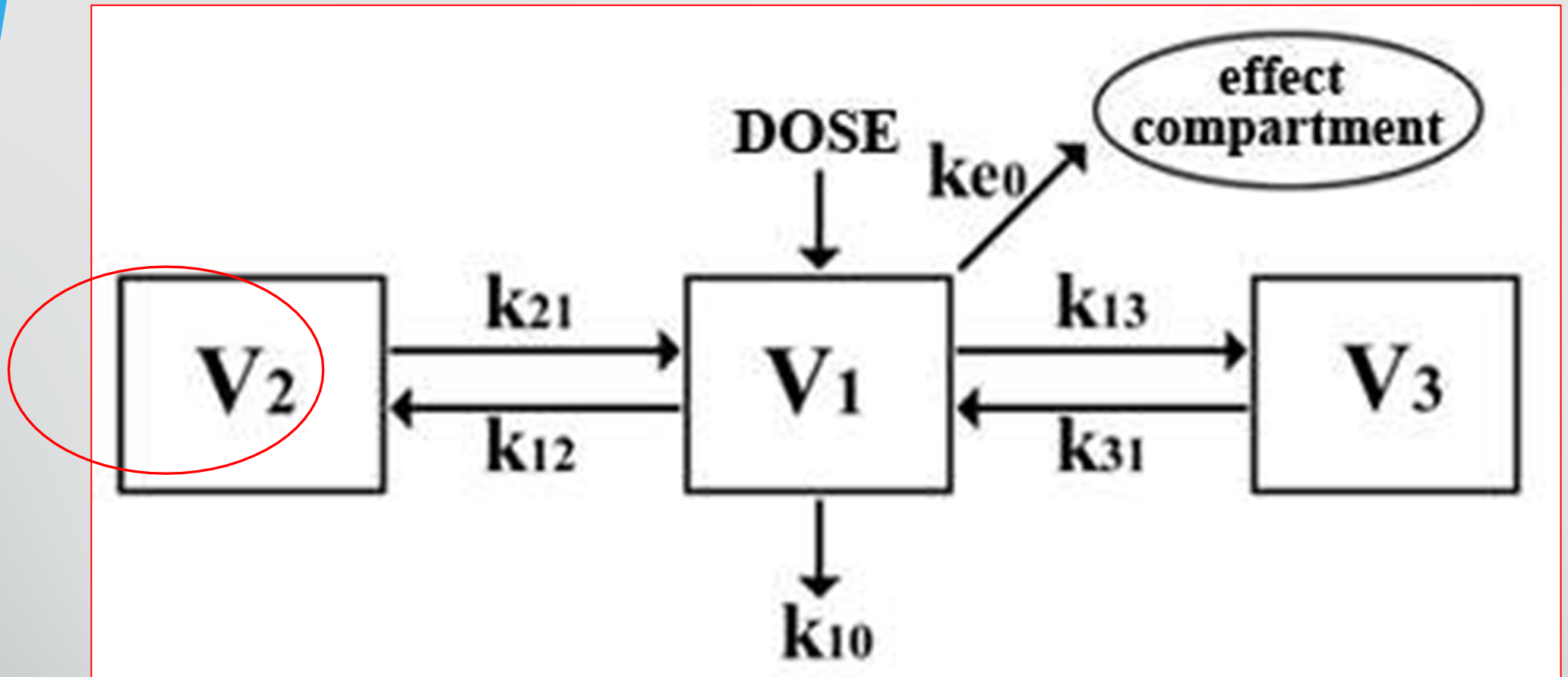
Pharmacokinetic principles





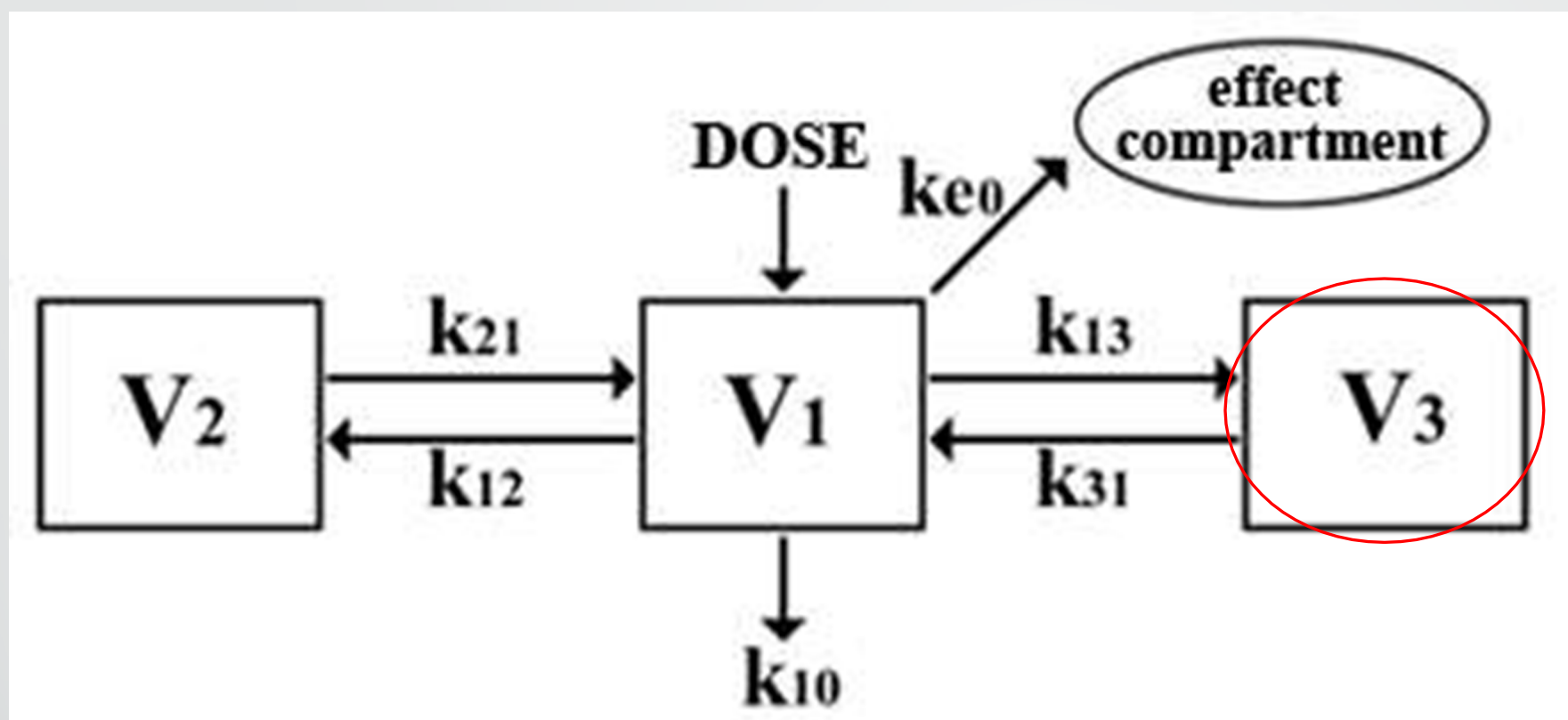
Drug injected into Central compartment V_1

- Initial volume of distribution
- Comparable to 'plasma'



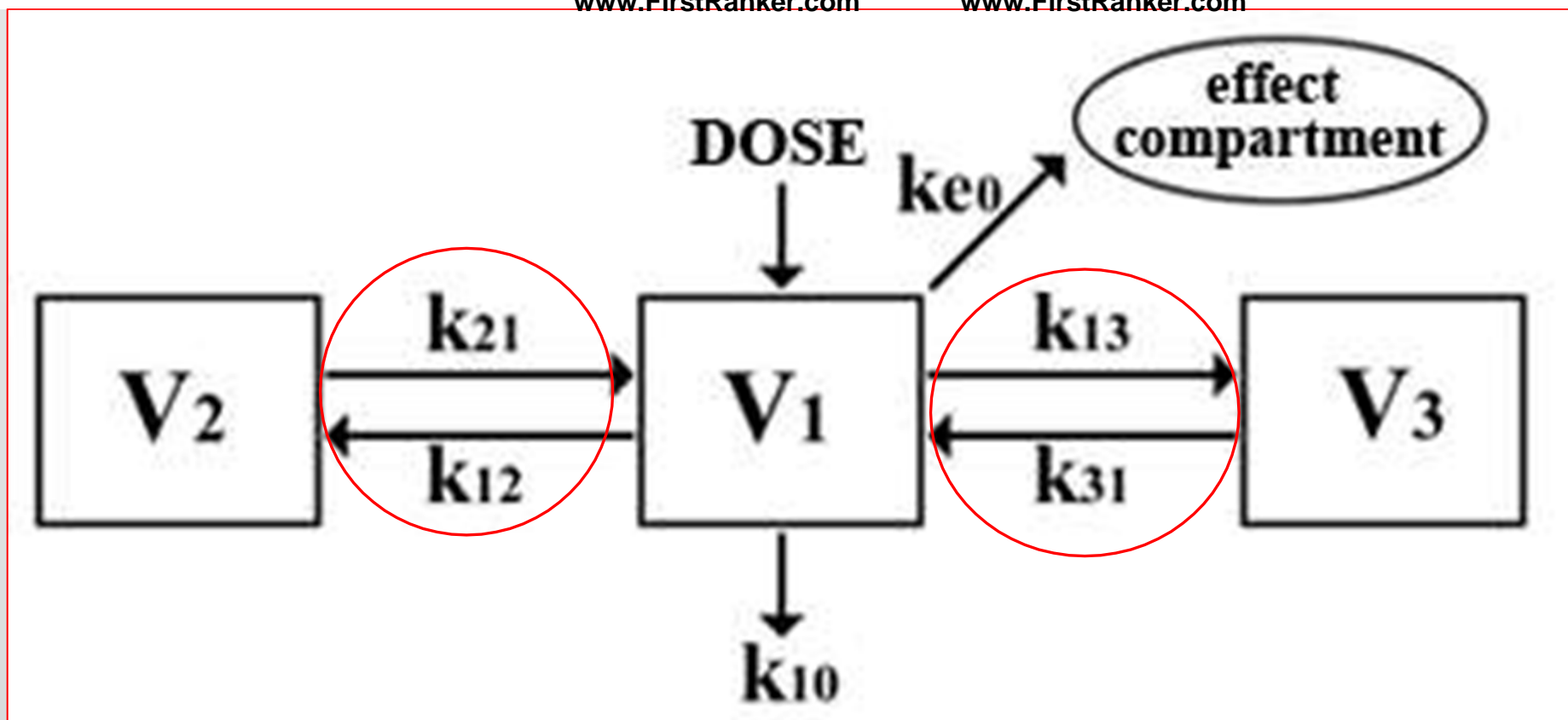
Redistribution into second compartment (V_2)

- "vessel-rich" or "fast"

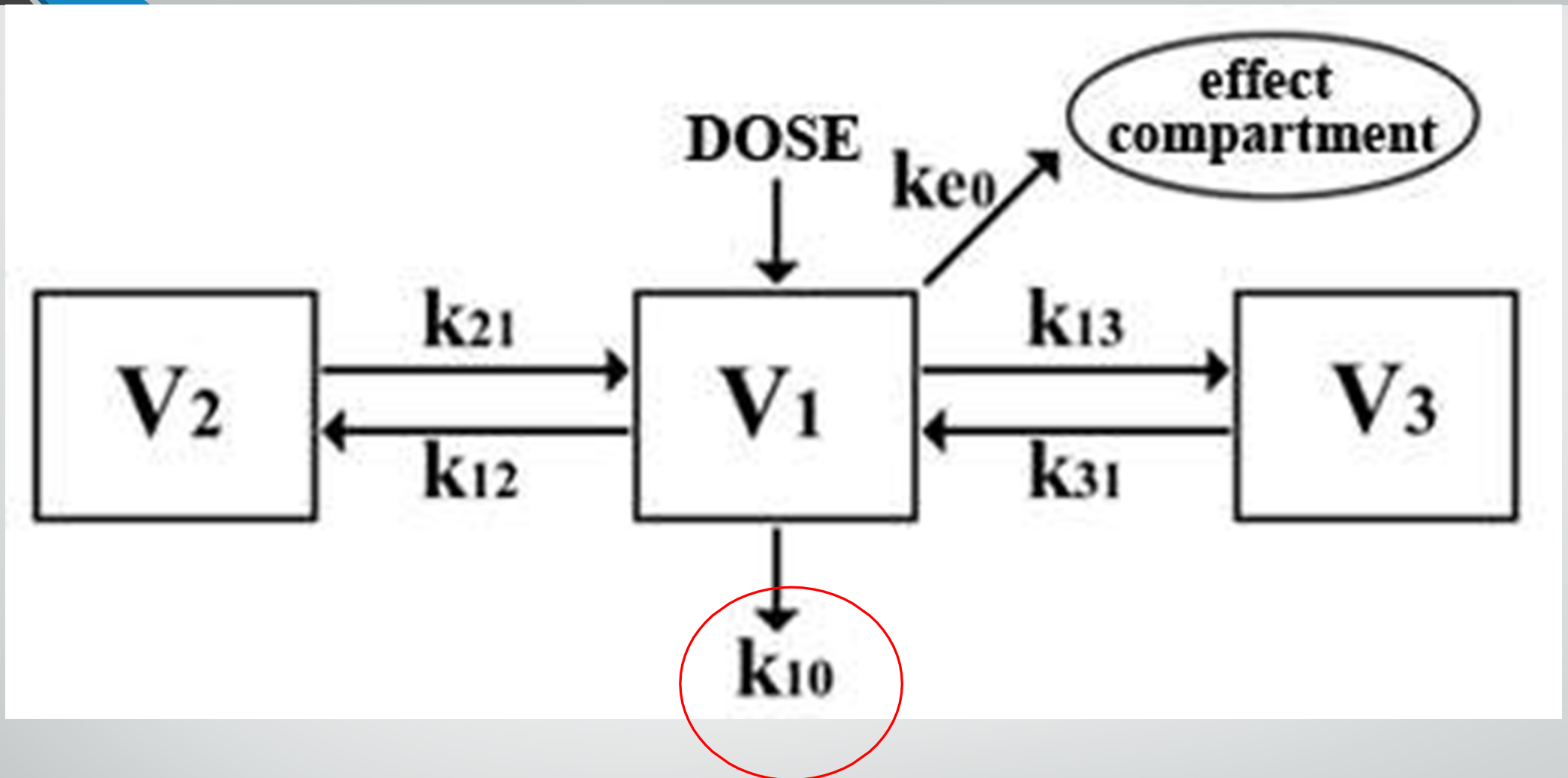


Redistribution into third compartment (V_3)

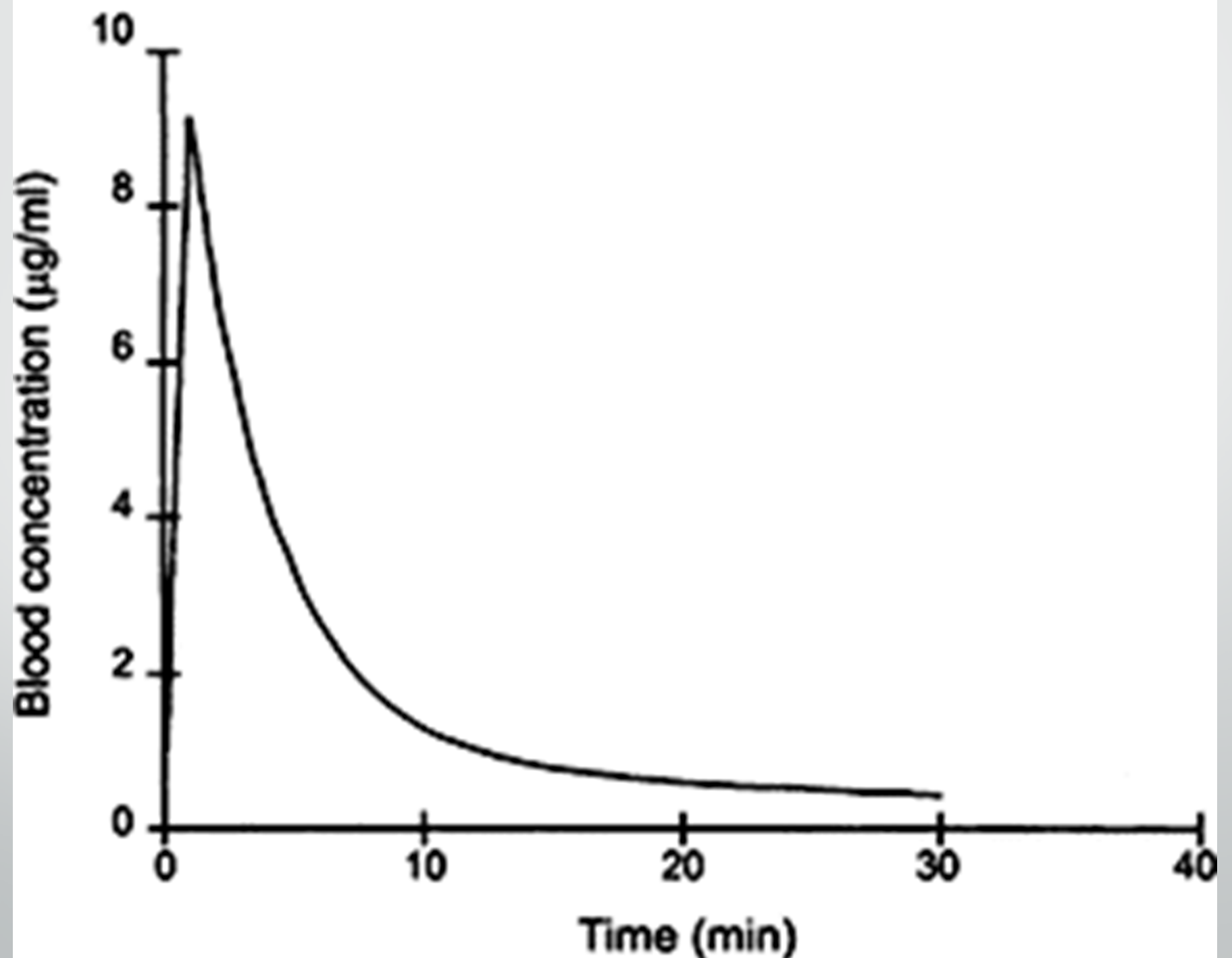
- "vessel-poor" or "slow"



- Governed by rate constant / concentration gradient.
Exponential process



Elimination - Fixed rate



Achieving a constant plasma level

- initial bolus = concentration desired x vol of distribution
- maintaining however can get tricky
- needs to match the rate of decline of plasma propofol level
- initially high rate de to a rapid redistribution
- reduces overtime as V_2 and V_3 fill up
- ultimately just matches the elimination

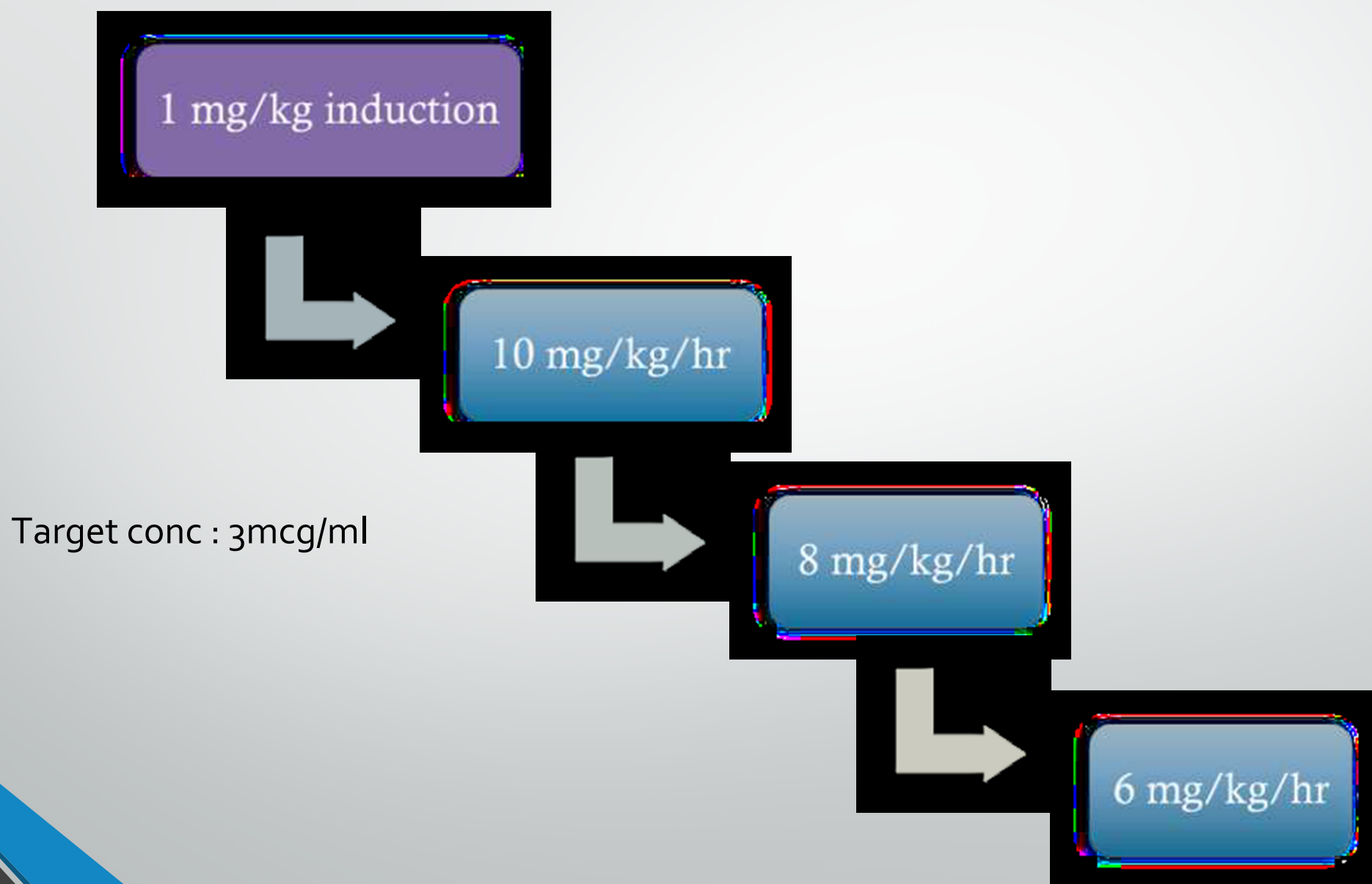
Manual infusions

- Inaccurate regimes,
- 'shooting in the dark'
- No control over the exact amount of drug concentration at any level.
- a thorough understanding of the pharmacokinetics of the drugs being used is necessary
- risk of under- or over-dosage

MANUAL INFUSION SCHEMES

	Anesthesia		Sedation or Analgesia	
Drug	Loading Dose ($\mu\text{g/kg}$)	Maintenance Infusion ($\mu\text{g/kg/min}$)	Loading Dose ($\mu\text{g/kg}$)	Maintenance Infusion ($\mu\text{g/kg/min}$)
Alfentanil	50–150	0.5–3	10–25	0.25–1
Fentanyl	5–15	0.03–0.1	1–3	0.01–0.03
Sufentanil	1–5	0.01–0.05	0.1–0.5	0.005–0.01
Remifentanil	0.5–1.0	0.1–0.4	†	0.025–0.1
Ketamine	1500–2500	25–75	500–1000	10–20
Propofol	1000–2000	50–150	250–1000	10–50
Midazolam	50–150	0.25–1.5	25–100	0.25–1
Methohexital	1500–2500	50–150	250–1000	10–50
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Bristol regime



- But:
- Changes to infusion rate will not lead to changes in blood concentration for some time
- Manual boluses have to be given to rapidly change depth
- Size of bolus has to be 'guestimated'
- May result in excessive side effects or awareness
- TCI systems automate the whole process

Target controlled infusions (TCI)

- Target Controlled Infusions
- Computer driven infusions to achieve a preset plasma concentration
- Multi-compartment pharmacokinetic models used to calculate infusion rate required to achieve the target concentration.
- “open-loop” systems
- Comprised of a user interface, a microprocessor and an infusion device

DOSES OF OPIOIDS FOR TIVA

	Loading Dose ($\mu\text{g/kg}$)	Maintenance Infusion Rate	Additional Boluses
Alfentanil	25–100	0.5–2 $\mu\text{g/kg/min}$	5–10 $\mu\text{g/kg}$
Sufentanil	0.25–2	0.5–1.5 $\mu\text{g/kg/hr}$	2.5–10 μg
Fentanyl	4–20	2–10 $\mu\text{g/kg/hr}$	25–100 μg
Remifentanil	1–2	0.1–1.0 $\mu\text{g/kg/min}$	0.1–1.0 $\mu\text{g/kg}$

Alaris Asena® PK (Alaris Medical Systems)



Base Primea (Fresenius)



How it works

- Models have sizes and rate constants for the various compartments programmed which allows the pump to calculate rate of Propofol redistribution and elimination at a given time
- Initial bolus given to achieve rapid rise in plasma level
- 3 superimposed infusion rates are present to match the rate at which drug is being removed from the central compartment
- When one wants to increase the plasma level then pump will calculate and give a bolus
- When one wants to decrease the level then the pump will stop and allow the level to fall before restarting

Effect site equilibration

- The lag time between achieving a specific plasma concentration and observing a particular clinical response.
- Mathematical or temporal relationship between the conc in the plasma and the clinical response observed – time taken to equilibrate is described as a rate constant (K_{eo})
- This is different for each drug.

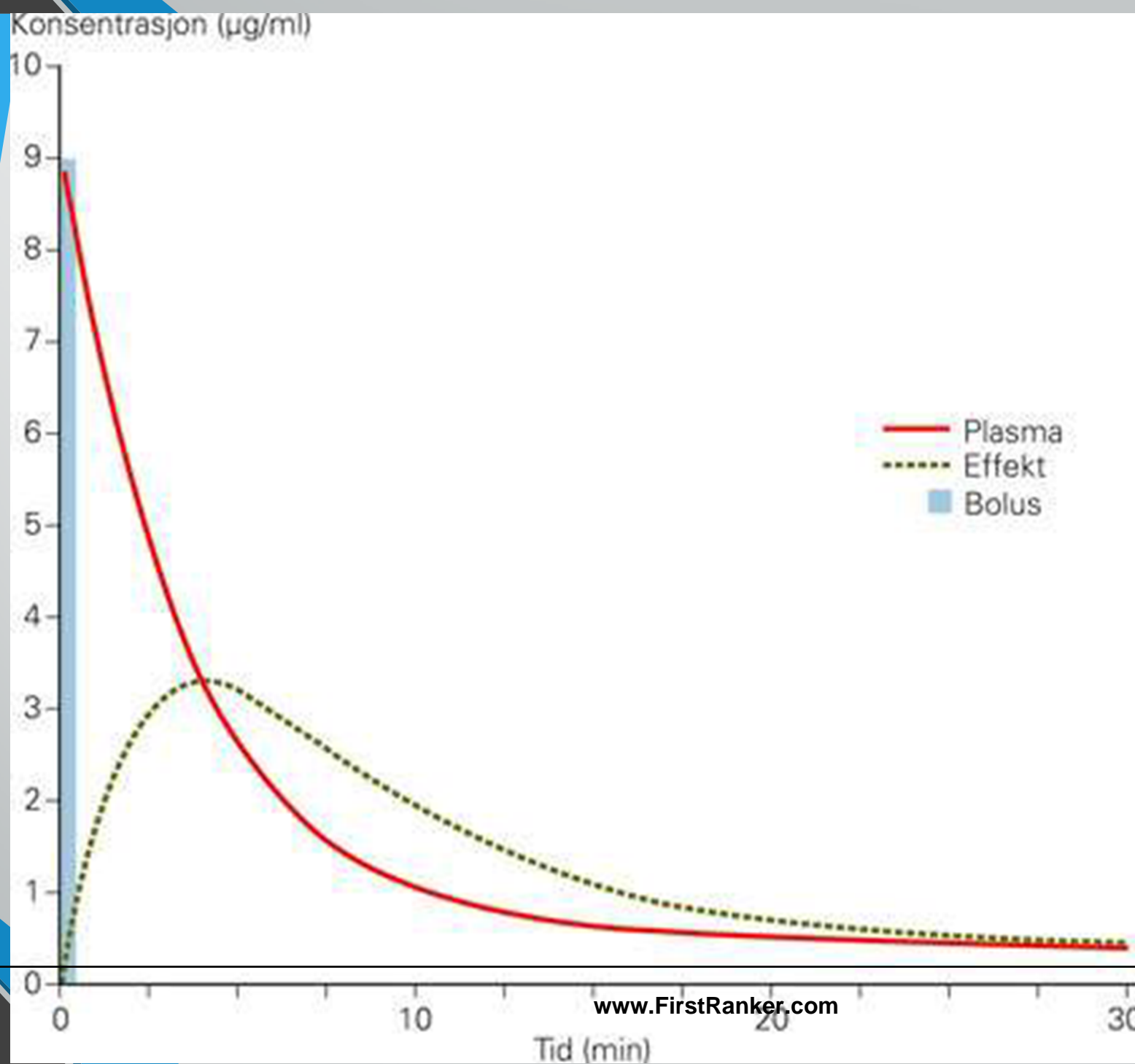
Plasma vs Effect site Targeting

- The clinical effect of Propofol is related to brain concentration = *effect site*
- With plasma targeting there is a lag between achieving the plasma level and the brain level catching up.
- Therefore the lag in induction and lag in changing depth of anaesthesia

- Equilibrium between blood and effect-site depends on several factors:
 - Rate of drug delivery to effect-site
 - Pharmacological properties of the drug
 - Mathematically described by Keo time constant
 - Concentration gradient
- Only factor we can control is the concentration gradient

Time to peak effect (TTPE)

- After a bolus, maximum effect-site concentration occurs at the point where the blood and effect-site concentration curves cross.
- Time delay between bolus and this point is known as the “time to peak effect” TTPE
- Independent of size of bolus
- Propofol TTPE is 1.6 minutes



- By knowing the Keo and TTPE it is possible to 'target' the effect site concentration
- Nomenclature of TCI:

Ce = Effect site concentration

Cp = Plasma concentration



Which to use ? Manual or TCI

- Use TCI if its available !!!!!
- Cochrane review in 2008

Looked at results of 20 poor quality trials

1759 patients patient pool were studied

No significant difference in quality of anaesthesia or adverse outcomes

- Hence,they Couldn't recommend one over the other

- *Małgorzata Witkowska et al* Compared the target controlled infusion and total intravenous anaesthesia with propofol and remifentanyl for lumbar microdiscectomy and concluded "There are no clinically important differences in haemodynamic variables, depth of anaesthesia, time to recovery and doses of propofol/remifentanyl between manually controlled and target-controlled infusion of propofol and remifentanyl."

Anaesthesiology Intensive Therapy 2012, vol. 44, no 3, 138–144

Propofol TCI Models : Marsh vs. Schnider

Marsh Model

- First Published in 1991
- Model employed in the original Diprifusor®
- Based on study of 3 groups of 6 patients
- Weight is the only limitation
- Age entered but has no effect on model
- Unless its an age of < 16 in which case pump wont run
- A 'modified' Marsh model was published by Struys et al in 2000
- Results in less overshoot and undershoot when using Marsh effect-site targeting
- Model used in most of the modern TCI systems.

- Uses total body weight (TBW)
- Will tend to overdose in obesity
- Ideal Body Weight (IBW) best for induction
- But....
- Maintenance infusion rate is TBW

Schnider Model

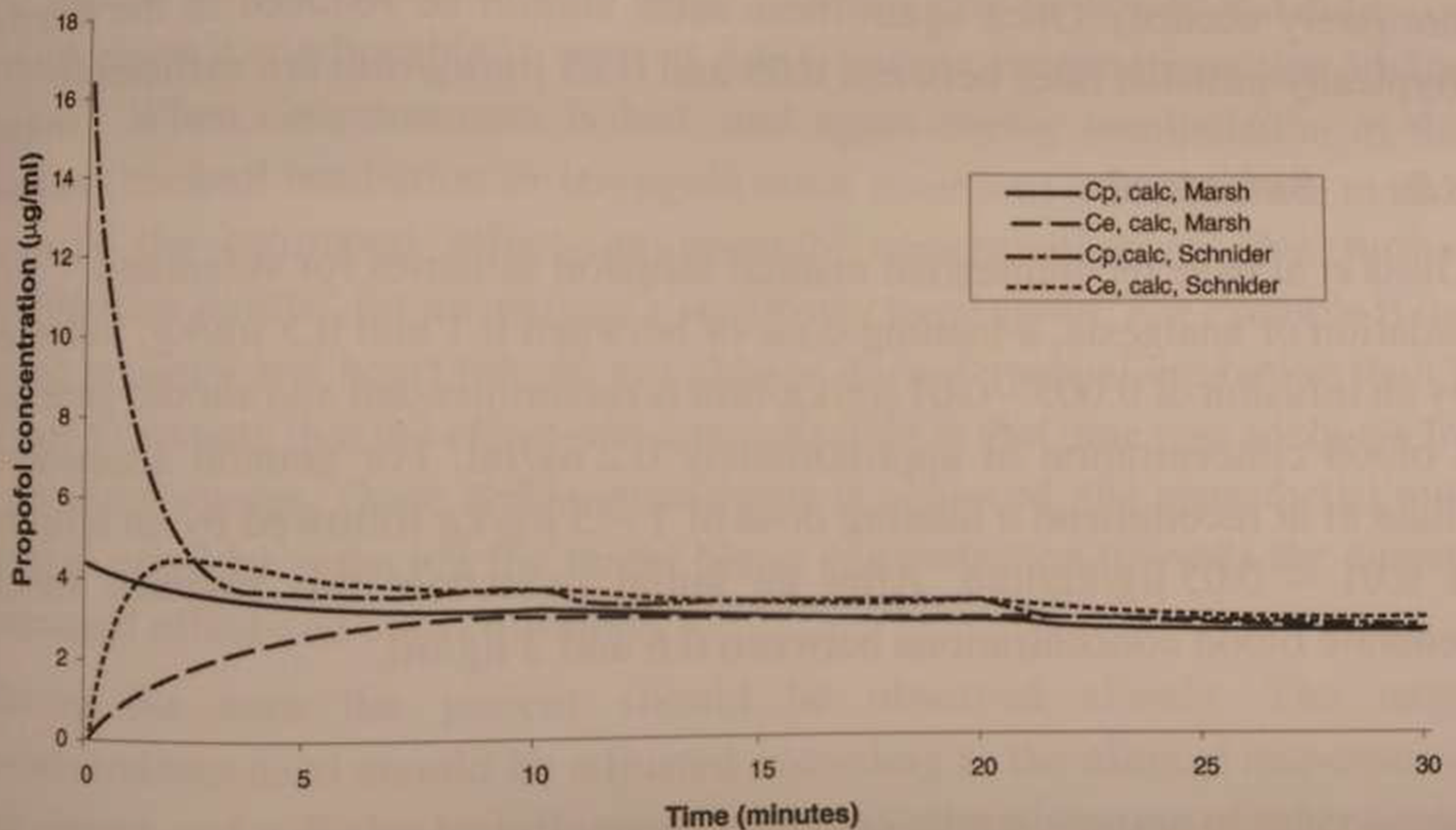
- Published in 1998 Based on 24 volunteers (11 women, 13 men)
- Uses age, height, weight, age and gender
- V_1 fixed - 4.27 L
- V_3 fixed - 238 L
- V_2 variable of age
- Elimination uses weight, height & LBM
- Uses a TTPE of 1.6 minutes and calculates a K_{eo} for each individual patient

- Uses lean body mass (LBM)
- User enters TBW and pump calculates LBM
- $LBM = 1.1 \times \text{weight} - 128 \times (\text{weight}/\text{height})^2$
- Accurate up to BMI of 42 in men and 37 in women - then get paradoxical decrease in LBM

Marsh Vs Schnider

1. Time To Peak Effect

- Schnider model has a faster TTPE (1.6 vs 4.5 min)
- Less 'overshoot' and 'undershoot' with Schnider effect-site targeting than with Marsh
- Net effect is less Propofol administered with Schnider vs. Marsh in effect-site targeting
- Probably safer in elderly and compromised patients



2. Size of central compartment

- Schnider has fixed V_1 (4.27 L)
- Marsh is a function of weight (15.9 L for 70kg)
- Striking differences in estimated plasma and effect-site concentrations in first 10 minutes after the bolus

- One minute after bolus:
- Marsh $C_p = 4 \text{ mcg/ml}$ $C_e = 0.9 \text{ mcg/ml}$
- Schnider $C_p = 8.2 \text{ mcg/ml}$ $C_e = 3.6 \text{ mcg/ml}$
- Differences less significant after 10 minutes
- After 30 minutes both estimate the same levels
- Net effect is Schnider administers less Propofol

3. Age

- Volume of central compartment reduces with increasing age
- It decreases by 50% from 25 to 75 years
- Marsh model doesn't account for age
- Schnider does

Typical target concentrations in routine practice

- Target concentrations are individually determined based on patient characteristics, other drugs administered, and the expected magnitude of surgical stimulus.
- If a relatively rapid induction of anaesthesia is required, initial plasma (Marsh model) or effect-site (Schnider model) propofol target concentrations of 4-6 $\mu\text{g}\cdot\text{ml}^{-1}$ are typically used in healthy young or middle-aged patients.
- During maintenance of anaesthesia, target concentrations of 3.0-6.0 $\mu\text{g}\cdot\text{ml}^{-1}$ (without opioids) or 2.5-4.0 $\mu\text{g}\cdot\text{ml}^{-1}$ (with opioids) are typical

- Other TIVA models used in neurosurgery :
 - i. Remifentanil in neuroanesthesia with the *Minto model*,
 - ii. TCI administration of sufentanil infusion in the *Gepts model*
 - iii. Older pharmacokinetic models for Dexmedetomidine (**Dyck and Talke**) were widely used which tended to under predict the plasma concentration at higher concentrations.

Hannivoort has recently published a new combined PK model for DEX

- Errors during TIVA can lead to failure to deliver the intended drug, under-dosing, over-dosing or other complications
- the two commonest causes of accidental awareness during TIVA were failure to deliver the intended dose of drug and poor understanding of the underlying pharmacological principles.

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- Where more than one infusion is given through a single i.v. cannula (or central venous catheter lumen) an anti-reflux valve should be present to prevent backward flow of drug up the infusion tubing.
- Drug and fluid lines should join together as close to the patient as possible to minimise deadspace in which a drug may accumulate rather than entering the vein
- The infusion line through which TIVA is delivered should have as few potential sites for leakage as possible.
- A continuous line from syringe to cannula is ideal, without additional connections or three-way taps

- Particular caution should be exercised if a cannula is inserted in a vein in the antecubital fossa, where inadvertent subcutaneous administration may be difficult to detect
- Previous guidance has recommended that the i.v. cannula through which TIVA is delivered should be 'visible at all times' , although this has been modified in more recent publications to specify 'visible whenever practical'
- Whenever , its not possible to keep an eye at all times , anaesthetists should have a higher index of suspicion for problems with the infusion and periodically inspect the cannula site, if possible.

- Pumps must be charged before use and, where practical, mains-powered during use to prevent failure due to battery depletion.
- Infusion pumps should only be programmed after a syringe containing the drug to be infused has been placed in the pump
- Drug labels should be attached to syringes only when the intended drug is drawn-up.
- Propofol must be drawn up using precautions to reduce the risk of contamination.
- Syringes should be prepared just shortly before use
- All vascular access devices used for TIVA should be flushed with at least twice the deadspace volume of the device at the end of the procedure

Monitoring in TIVA

- Use of an EEG monitor is recommended when TIVA is underway especially with manual infusions.
- Efforts to prevent awareness should, mostly focus on patients who receive a neuromuscular blocking drug.
- large majority of cases of self-reported awareness that were identified occurred in patients who had received a neuromuscular blocking drug (AAGBI 2017)
- Processed EEG monitoring should commence before administration of the neuromuscular blocking drug

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Other applications of TIVA in neuroanesthesia

TIVA in Brain trauma

- IV anesthetics can be administered for maintenance of anesthesia as part of a balanced anesthetic that includes inhalation agents, or as TIVA.
- Most commonly, TIVA includes an infusion of propofol along with infusion of a short-acting opioid
- Propofol infusion – Propofol infusion causes reduction in CMR, CBF, CBV, and ICP, while CO₂ responsiveness and autoregulation are maintained.
- Opioids – When administered as part of IV anesthesia with controlled ventilation, opioids have minimal, clinically irrelevant effects on cerebral physiology

- The vasoconstrictive property of *dexmedetomidine* may be of concern in patients at risk for regional cerebral ischemia or compromised flow metabolism coupling (eg, traumatic brain injury [TBI], subarachnoid hemorrhage, intracranial lesions);
- Data regarding Dexmed in TBI is very limited barring few animal studies
- **Drummond JC et al** studied Brain tissue oxygenation during dexmedetomidine administration in surgical patients with neurovascular injuries and found that there was no significant reduction in CBF as postulated popularly. (*J Neurosurg Anesthesiol.* 2010 Oct;22(4):336-41.)

Electrophysiological monitoring and TIVA

- Electrophysiological monitoring is applied during cranial and spine surgery for monitoring and for mapping
- EEGs are usually monitored during craniotomy for cerebral aneurysm clipping, during carotid endarterectomy, cardiopulmonary bypass, extracranial–intracranial bypass procedures, and pharmacological depression of the brain for “cerebral protection.
- The use of TCI allows a constant level of anesthetic effect which can help to avoid misinterpretation of EEG depression caused by boluses or rapid changes in anesthetic level from true physiologic/pathologic insults to the cortex.

- Inhalational agents and muscle relaxants, are confounders for motor evoked potential (MEP) monitoring as they have deleterious effects on the amplitude of the waveform signal.
- (TIVA) with no intraoperative muscle relaxants following intubation has been suggested as the preferred anaesthetic technique for these surgeries.
- However balanced anaesthetic technique with a low dose inhalational and an adjunct IV regimes have been recently established.

(Royan NP, Lu N, Manninen P, Venkatraghavan L. The influence of anaesthesia on intraoperative neuromonitoring changes in high-risk spinal surgery. J Neuroanaesthesiol Crit Care 2017;4:159-66)

TIVA IN PEDIATRICS

- Compartment volumes in children are about twice the size of those in adults in comparison with their body weight.
- This difference gradually reduces from around 12 years of age, reaching adult values at 16 yr.
- Thus, to achieve a given plasma concentration, children require larger propofol bolus doses and initial infusion rates relative to body weight than adults
- During prolonged infusions of propofol in children aged < 12 yr, drug accumulation in the peripheral compartments occurs to a greater extent than in adults.

- Therefore, when the infusion is stopped it typically takes longer in a child for the propofol concentration to decline to a level at which consciousness is regained than in an adult
- Propofol requirements can be reduced, and speed of emergence improved, by remifentanyl (or other opioid) co-administration, and the use of other drugs such as nitrous oxide, ketamine and α_2 agonists.
- Most children regain consciousness at an estimated propofol plasma concentration of approximately $2\mu\text{g.ml}^{-1}$, but this can vary considerably from $1\text{--}3\mu\text{g.ml}^{-1}$ depending on inter-individual differences and the use of adjunctive drugs

- The two widely available and validated paediatric models which target plasma propofol concentration are Kataria [11] for ages 3-16 yr and Paedfusor [12] for ages 1-16 yr.
- Effect-site targeting has not been implemented in paediatric TCI systems
- For an average length procedure in a young child, both models administer approximately 50% more propofol than in an adult using the Marsh model, which is why adult models should not be used in this age group

- Limited use so far due to the due to the original weight restrictions on target controlled infusion devices
- Modified schnider models have been advocated in children >5years.(ped anaesthesia 2010)
- Propofol use, at induction and as maintenance of anaesthesia, has been seen to reduce the risk of Emergence Delirium in comparison with sevoflurane anaesthesia. Costi D , Cyna AM, Ahmed Set al. . Effects of sevoflurane versus other general anaesthesia on emergence agitation in children. Cochrane Database Syst Rev 2014: CD007084)
- The incidence of PONV in children over 3 yr is double that of adults. Propofol reduces early PONV significantly. (Creeley C , Dikranian K, Dissen G, Martin L, Olney J, Brambrink A. Propofol-induced apoptosis of neurones and oligodendrocytes in fetal and neonatal rhesus macaque brain. Br J Anaesth 2013; 110(Suppl. 1): i29–38)

- Remifentanil infusion has been tried in children widely
- A remifentanil infusion commenced at the induction of anaesthesia can readily be titrated to response and avoids the hypotension and bradycardia associated with boluses of remifentanil in children.
(Krane EJ, Phillip BM, Yeh KK, Domino KB. Smith RM, Mototyama EK, Davis PJ. Anaesthesia for paediatric neurosurgery, Smith's Anaesthesia for Infants and Children , 20067th EdnPhiladelphia Mosby(pg. 651-84)
- Remifentanil usually obviates the need for repeated doses of neuromuscular blocking agents significantly in children

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Strategy of conscious sedation in awake Sxs

- Administer sedation with a combination of dexmedetomidine with propofol, as follows
 1. Administer midazolam 1 to 2 mg intravenous (IV), and fentanyl 25 to 50 mcg IV (no midazolam if electrocorticography is planned).
 2. Administer loading dose of dexmedetomidine 1 mcg/kg IV, dose adjusted for patient factors, followed by infusion dexmedetomidine 0.3 to 0.7 mcg/kg/hour, titrated to level of sedation. Add propofol infusion as necessary (start at 25 mcg/kg/min, titrate to level of sedation (25 to 75 mcg/kg/min)).
 3. For skull pinning, administer propofol boluses until patient is unarousable with tactile stimulation (propofol 10 mg IV boluses, total usually 30 to 40 mg IV). Surgeon infiltrates pin sites with 1 or 2% lidocaine, then places skull pins. Administer fentanyl 25 to 50 mcg IV if necessary to tolerate pinning.
 4. When patient awakens, check that head, neck, and shoulders are comfortable, and readjust position if necessary and as possible. The surgeon infiltrates the scalp with up to 40 mL of 0.25% bupivacaine with epinephrine 1:200,000
 5. Additional boluses of fentanyl 25 to 50 mcg IV, with or without propofol 10 to 20 mg IV may be administered, or infusions increased, for pain as needed during skull pinning, scalp infiltration, or during painful portions of surgery (eg, temporalis muscle dissection, dural opening or closure).
 6. Stop propofol infusion after bone flap is removed, and wait for the patient to wake up for cortical mapping. Stop or reduce dexmedetomidine infusion at the same time, depending on the depth of sedation. For some patients, dexmedetomidine can be continued during mapping.
 7. Restart sedation after mapping, with propofol bolus 10 to 20 mg IV, followed by infusion as before mapping.
 8. During scalp closure, administer ondansetron 4 mg IV, discontinue propofol and dexmedetomidine infusions, and administer fentanyl 25 mg IV, repeated as necessary for pain.

Strategy for asleep-awake-asleep technique

- For patients who require an asleep-awake-asleep technique, we prefer to use total intravenous anesthesia (TIVA) with propofol and remifentanyl, and to manage the airway with a laryngeal mask airway, as follows:

- **Asleep portion -**

1. After pre-oxygenation, induce general anesthesia with propofol (2 to 2.5 mg/kg IV) and fentanyl (0.5 to 1 mcg/kg IV). Test and note the degree of difficulty with mask ventilation before inserting a laryngeal mask airway (LMA).

2. Maintain anesthesia with TIVA using propofol (100 to 150 mcg/kg/min) and remifentanyl (0.05 to 0.1 mcg/kg/min). Maintain spontaneous ventilation if possible; controlled ventilation may be required to reduce PaCO₂ for brain relaxation.

3. After skull pinning, position the patient carefully, avoiding extreme neck rotation and/or flexion, and ensuring access to the face for airway manipulation.

If there are concerns about the airway after the head fixation, before finalizing positioning, remove the LMA, verify the ability to ventilate by mask with an oral airway in place, and that the LMA can be reinserted easily. If necessary, adjust the head position.

Awake portion — Call for assistance for awakening.

1. Assure spontaneous ventilation, then turn off propofol, reduce remifentanyl to 0.03 to 0.05 mcg/kg/min, and administer 100 percent oxygen.
2. Warn the surgeon that the patient might cough, and gently suction the oropharynx.
3. Extubate or remove the supraglottic airway when awake.
4. If necessary, continue remifentanyl infusion (0.03 to 0.05 mcg/kg/min) for analgesia during awake procedure.

Asleep portion — Induce general anesthesia with propofol and fentanyl as before, and reinsert the LMA. Maintain anesthesia with TIVA, as before, for the rest of the procedure.

IN CONCLUSION

- Currently, there are no consensus guidelines or recommendations suggesting any one as the best anesthesia technique for neurosurgical procedures
- However, it's wiser to choose a balanced technique keeping in mind the nature of surgery, condition of the patient, risks and benefit for choosing a technique and above all the acquaintance and knowledge pertaining to the said technique.-



THANK YOU