Drug	Induction dose	MOA	ONSET	Special features and Effects	notes
Propofol	1.5– 2.5 mg/Kg	Increases binding affinity of GABA with GABAA receptor.	30-60 secs after administration >> "arm brain" circulation time	**-Potent cardiovascular and respiratory depressant** **-Decreases BP by decreasing cardiac contractility, SVR and preload (inhibition of sympathetic tone and direct vascular smooth muscle effect).** **-The most profound cardio depressant of all induction agents** **-Has antipruritic and antiemetic properties – used in TIVA (total intravenous anesthesia) and as background infusion to prevent PONV (post op nausea & vomiting)** **-pain on injection**	**-Produced in an 1% egg lecithin emulsion, glycerol and soybean oil (relevant to patient allergies to egg white – not contraindicated with egg allergy).** **-Formulation can support bacterial growth ,need for good sterile technique** **-Highly lipid soluble only administered intravenously** **-Rapid hepatic metabolism to water soluble compound and removed by kidneys** **-Co-administration of 1% lidocaine can lessen the pain.**
Barbiturates	3 – 5 mg/kg adults	Enhance GABAA receptor transmission	30-60 secs after administration >> "arm brain" circulation	-Leads to prolonged cognitive effects: Decreases (CMRO2 (CBF) (ICP).	**Most commonly used are thiobarbiturates (thiopental) and oxybarbiturate (methohexital)** **-Highly alkaline (pH ~ 10) at 2.5% solution** **-Undergo terminal elimination via hepatic metabolism.** **-Should not be used in patient with porphyria will stimulate porphyrin formation and lead to acute crisis**
Etomidate	0.2 – 0.3 mg/kg	Acts through binding to GABAA receptors	-------	**decreases CMRO2, CBF and ICP while maintaining good CPP.** **-Superior hemodynamic stability**	**-irritation and pain on injection** **-Lidocaine pre-administration will help**

				(does not cause vasodilation or myocardial depression) -PONV is common. -adrenal suppression.(nhibits 11-B-hyroxlase),	
ketamine	1to2 mg/kg IV -The IM induction dose is 4 to 6 mg/kg.	Mechanism through NMDA (N-Methyl-D-aspartate) receptor antagonism.	----------	**Causes analgesia by blocking pain signals at spinal cord but also disassociating the signal between thalamus and limbic system. -Dissociative amnesia -Stimulates sympathetic nervous system -Has minimal respiratory depression -Causes potent bronchodilation -Increases CBF, CMRO2, ICP -Direct myocardial depressant but indirectly increases catecholamines resulting in increased blood pressure, heart rate and cardiac output.**	**-Several routes of administration: IV, IM, Oral, Rectal, epidural and intrathecal -Relative contraindication in patients with space-occupying CNS lesions**
Dexmedetomidine	--	Highly selective alpha-2 adrenergic agonist		**-Sedation and analgesia without much respiratory depression -Has sedative, analgesic, sympatholytic, and anxiolytic effects**	**-Sedation for awake fiberoptic intubation, regional anesthesia or as an adjunct to general anesthesia. In ICU, to wean patients off ventilator**
Benzodiazepines	0.1 – 0.2 mg/kg IV	Bind to same GABAA receptors as barbiturates but at different site on receptor		**Produce mild respiratory, cardiovascular and upper airway reflex depression -All benzos have anxiolytic, amnestic, sedative, hypnotic, anticonvulsant properties. NOT analgesic.**	**-Commonly used benzos are: Midazolam , Diazepam and Lorazepam -Usually given as premedication, sedation and anxiolysis before GA -sedation can be reversed by administration of flumazenil – specific competitive antagonist**