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Delayed Recovery after Anesthesia

Thesis

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INTRODUCTION

DEFINITION OF RECOVERY

It has been generally accepted that cortical arousal and focus of attention elicited by afferent sensory stimuli are mediated by the reticular activating system (RAS). This multisynaptic pathway is known to be early depressed by barbiturates, (French, et al., 1953), and certain metabolic disorders as hypoglycaemia and hypoxia (Arduini and Arduini, 1954). Although selective depression of the reticular activating system may be of some significance in the delayed awakening in certain patients following anaesthesia, many other cortical and subcortical neural pathways are also involved. Recent evidence in humans and experimental animals has shown that general anaesthesia is produced by a variety of neurophysiologic mechanisms. For example, ether produces early cortical depression, and ketamine results in neural excitation both at cortical and subcortical levels (Darbinjau, et al., 1971, and Ferrer et al., 1973). Thus the neurophysiologic mechanism of prolonged narcosis following anaesthesia is drug dependent and may involve neural stimulation or depression at cortical or subcortical levels.

Post operative coma during recovery may be attributed to post anaesthetic respiratory depression which is dependent upon several risk factors as, the

patient's age, it is more pronounced at the extremes of age, any associated disease, the preoperative status, whether the operation was an emergency, and the duration of the procedure (Tiret, et al, 1986). Post operative coma was also related either to a cerebrovascular accident or to post anoxic encephalopathy, also some patients are related to delayed symptoms in relation to anaphylactoid reactions.

The prognosis of post anaesthetic coma appears to relate to the availability of post anaesthetic recovery room (Tiret, et al, 1986) Before recovery, the patient's vital signs should be stable, evaluated by the responsible physician to that individual. The course and post operative recovery room nurse clearly delineates the patient's perioperative course and the post operative recovery. Many scoring systems are used to assist in the discharge decision. An ideal scoring system would be sensitive, reliable and easily used.

General guide lines for recovery room discharge include:

- 1- Recovery from anaesthesia so that the patient is easily arousable and coherent.
- 2- Stable vital signs, particularly integrity of cardiovascular system and pulmonary system.
- 3- No surgical problems such as bleeding.

The individual responsible for discharge should determine the number and skill levels of personnel required to transfer a given patient safely. High risk individuals may require a physician in constant attendance during transfer to another intensive care unit (Burnell, et al., 1983)

For evaluation of post operative recovery, awakening time was recorded from end of administration of anaesthesia to the time required to follow simple commands. Orientation time was recorded from the end of administration of anaesthesia to that time when the patient could recall his(her) name, date of birth, and present location (Martin, et al., 1988).

The degree of post anaesthetic recovery was assessed using a modification of the recovery room score developed by Steward, (1975).

	upon reversal of muscle relaxant	5 min. after turning off N ₂ O	upon passing suction catheter in trachea	upon suctioning oropharynx	upon extubation
<u>How Awake</u>					
2: Fully awake	2				
1: Arousable	1				
0: Not responding	0				
<u>Ventilation</u>					
2: Can cough	2				
1: Breathing regularly	1				
0: Not breathing	0				
<u>Movement</u>					
2: Moving purposefully	2				
1: Moving involuntarily	1				
0: Not moving	0				
<u>Time in Minutes :</u>	A	B	C	D	E

Post anaesthetic recovery score that allows progressive assessment of recovery of consciousness, ventilation and movement . The highest total score possible at each recovery period is six. (Steward, 1975).

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The modified Steward coma scale emphasises the maintenance of a clear airway and the awake state, reflecting the essentials for patient safety. Assessment was performed at one minute interval for 15 minutes following the termination of anaesthesia (Wolff, et al., 1986).

* <u>Consciousness</u>	Score
- Fully awake, eyes open , conversing.	4
- Lightly asleep, eyes open intermittently.	3
- Eyes open on command or in response to name.	2
- Responding to ear pinching.	1
- Not responding.	0
* <u>Airway</u>	
- Opening mouth or coughing or both on command.	3
- No voluntary cough, but airway clear without support.	2
- Air way obstructed on neck flexion , but clear without support on extension.	1
- Air way obstructed without support .	0
* <u>Activity</u>	
- Raising one arm on command .	2
- Non purposeful movement .	1
- Not moving .	0

Modified Steward coma scale. A score of 9 indicates complete recovery to the awake state.

Gustafson, (1985), defined the organic brain syndrome scale (OBS), which was used to detect and follow confusion. The (OBS) scale consists of 2 parts, the dysorientation subscale and the confusion subscale. The disorientation subscale describes the patient's orientation as to time, place and own identity,. The confusion subscale describes different cognitive perceptual, emotional and personality changes, physical and practical disabilities and fluctuation of the clinical state. The items are rated on a four point scale. A non disturbed behaviour or correct answers result in a zero (0). Most disturbed behaviour or a totally incorrect answer is assigned three points, as in no answer given.

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CAUSES OF DELAYED RECOVERY

Prolongation of drug action is attributed to many factors including:

I OVER DOSE

Delayed awakening after general anaesthesia is most commonly by anaesthetic over dose. Such over dose can occur when anaesthetics are administered for the wrong reason. For example, elevated perfusion pressure during cardio-pulmonary by pass. The cardio pulmonary by pass produces marked changes in drug pharmacokinetics. Elimination half life is prolonged due to a large volume of distribution and decreased hepatic blood flow (Hug, et al., 1983). Plasma protein binding is decreased (due to dilution), and although total plasma drug concentration is reduced, decreases in free concentration will be buffered by large peripheral compartment stores. Decreased tissue (skeletal muscle) perfusion during by pass or bypassed tissue (lung) will affect drug kinetics, and decreases in hepatic perfusion and body temperature will slow hepatic drug clearance and metabolism (Hug, et al., 1983).

Intra operative hypertension due to catecholamine secreting tumour might be treated with large doses of barbiturates in an attempt to deepen anaesthesia. Failure to employ specific vasodilator or adrenergic blocking agents in such condition can lead to

anaesthetic over dose and delayed recovery. (Denlinger, 1983)

Drugs that compete with barbiturates for common binding sites increase barbiturate sleeping time by displacement of barbiturates from plasma protein. For example, administration of sodium acetrizate (a radiographic contrast material) increases the duration of pentobarbital narcosis by way of this mechanism (lasser, et al., 1963). Thiopental concentration in the brain and heart following intra venous administration is markedly increased by pretreatment with sulfadimethoxine, a drug that undergoes extensive protein binding (Ghoneim, et al., 1976). Hypoproteinemia may prolong the duration of barbiturate anaesthesia by a reduction in the delivery of barbiturate to the liver (Said man, 1974).

The most popular technique for narcotic anaesthesia used in the past was bolus injection producing initially supratherapeutic blood levels and relative albeit safe over dose. This has often led to prolonged effects during the post operative period. The use of pharmacokinetic principles and the theoretic ability to produce known plasma concentrations of drugs has led to minimize this side effect. The use of alientanil which is relatively more predictable than