

May excessiveness of renalase enzyme be one of the underlying biochemical and endocrinal mechanisms of late reanimation from anaesthesia?

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Short Communication

Anaesthesia is inhibiting the pain of the patient with the help of various drugs for a painless and safe operation. Healthcare providers performing this work are anaesthetists. These people make evaluations about presence of drug use, drug allergies or any other allergies, general health conditions, information about previous diseases and surgery if present, and surgical methods and anaesthesia techniques if patient previously underwent an operation before applying anaesthesia [1]. Most common problem after application of anaesthetic substances may be late or early reanimation or early reanimation during the surgery [1,2]. Even though occurrence of these kind of situations is generally linked with the genetical properties of the patients or plasma cholinesterase enzyme deficiency [3], we presume (as a biochemist and clinicians) in this editorial that it may be linked to the amount of Renalase enzyme which is responsible for the catabolism of dopamine [4]. It is known that low level of dopamine was detected in the patients who reanimate lately [5]. Low levels of dopamine may either be caused by excessiveness of Renalase enzyme which metabolizes dopamine (in the presence of excessive Renalase enzyme per each unit of substrate) or deficiency of dopamine synthesis for any reasons (deficiency of tyrosine enzyme limits the synthesis of catecholamines). L-DOPA (this molecule passes the brain-blood barrier and converts into dopamine molecule) application [6] may even be applied for early reanimation of treatment of late reanimating patients. As we review the synthesis and release of Renalase enzyme, even it is synthesized from kidneys, it is reported that it is released from various other biological tissues including brain [7]. Principal duty of this enzyme is to remove catecholamines (dopamine, epinephrine and nor-epinephrine) by metabolizing them. It is foreseen that amount of Renalase in brain causes late reanimation from anaesthesia rather than the amount of Renalase which is synthesized peripherally (for example Renalase released from kidneys). Because Renalase is an enzyme weighing 3.8 kDa and it cannot pass brain-blood barrier. If Renalase synthesizing regions in brain increase their capacity (such as hypertrophy), late reanimation of anaesthesia will occur due to deficiency of dopamine as catabolism of catecholamines

increases [4,7]. The reason of differences in catabolism of anaesthetic substances among individuals may be caused by schizophrenia, hyperactivity and attention deficit and Parkinson's disease. For instance, while individuals with schizophrenia and hyperactivity may reanimate earlier because of dopamine (probably deficiency of Renalase in brain), patients having diseases such as Parkinson which may cause deficiency of dopamine may also reanimate lately. If we know the effect of dopamine related diseases on anaesthetical applications in individuals, we can consider redosing of anaesthesia. Current anaesthesia options such as general, spinal, epidural, nerve block and local anaesthesia may give anaesthesia science a new point of view by revealing the amounts of catecholamines and Renalase. One day, determination of the relationship between plasma cholinesterase (deficiency), dopamine and Renalase enzyme may be irreplaceable routine laboratory exercises in order to remove late reanimation problem. This may remove the concerns of doctors (especially during long-term operations such as cardiac and eye surgery) and relatives of the patients by revealing the relationship as well as may increase the social comfort.

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