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Replacing the illogic of anesthesia by the logic of aneuronia

Sir,

May we be allowed to declare at the outset that the word anesthesia is illogical and is in need of being replaced by a more logical, comprehensive, etymologically correct term. Skinner in his the origin of medical terms expands on anesthesia as follows: “Greek -an privation, plus aesthes, sensation; plus suffix ia indicating a condition in which there is absence of sensation. So defined in 1721 by Bailey, for a defect in sensation observed in certain paralyzed individuals.” It should be obvious that the taken-for-granted term anesthesia was born in permanently paralyzed humans and for reasons best unknown, has been extrapolated to mean a vital discipline of medicine. Some analysis and a cogent synthesis are called for.

The term anesthesia is beset by another problem, namely the fact that aesthesia and aesthetics connote refined taste, sense of cultural values and courteous and decent living. The increasing absence of aesthetics in the expanding metropolises of the world would lead one to declare that they are all suffering from anesthesia. Further anesthesia has usually aimed at reversible inhibition not only of sensation but also of movement and consciousness. Surely we need to replace the illogic of anesthesia by a logical concept.

A medical dictionary of the third millennium lists 61 modes of anesthesia, the cardinal feature of each of them being reversible inhibition of some neurones in central craniospinal axis or in the peripheral nerves which are nothing but dendritic/axonal extensions of central neurones themselves. The concept of nerve fibre creates an illusionary dichotomy between the bodies of the nerve cells in the craniospinal axis and their reach to the farthest periphery of the body through the so called nerves. An act as simple as the instillation of anesthetic drops into the eyes to measure ocular pressure or a brachial plexus block basically affect nothing but neurones, albeit away from the main cell bodies. One can safely conclude that the target cell in all the 61 forms of anesthesia is the neurone. The logical step is to allow the new term aneuronia to replace the illogical term anesthesia. Needless to say aneuronia is a mother term to allied words such as aneuronist, aneuronology, aneuronic, and so on. If a cytologist deals with cells, an aneuronist deals with neurones in effecting their reversible inhibition to facilitate a procedure.

It comes as a surprise to realize that the science of aneuronology (anesthesiology) doesn’t engage itself in either diagnosis, investigation or treatment of any medical condition, So where do we put this global discipline?

We need to recognize aneuronology as ‘facilitatory medicine’ that assists diagnosis (examination under anesthesia) investigation (CT scan /MRI of a child) and of course surgery. Advances in aneuronology have facilitated the advances in surgery, but it is not the other way round. The only invasion that an aneuronist inflicts on the human body is a prick into a vein or the spinal theca, a catheter in the epidural space or intubation into tracheobronchial tree.

We submit that the 9-lettered aneuronia in comparison with the 10 lettered anesthesia is more evocative, elegant, euphones, etymologically correct and educative. It directly indicates the neuronocentric basis of the aneuronal science. The new term ushers in eusemantics11 in a branch which in the last 150 years has matched its progress with another discipline namely radiology.

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Sir,

Quinine has been available for centuries and is commonly used for the treatment of leg cramps. However, it can cause a variety of adverse reactions including fever, skin rash, hematological abnormalities and organ failure. We present a patient with drug rash with eosinophilia and systemic symptoms (DRESS) syndrome due to quinine.

A 58-year-old man was admitted to the hospital with a one-week history of jaundice. He had been unwell with myalgia, fever, chills and rigors for two weeks and had noticed a generalized, pruritic, skin rash two days before admission. He had a past history of well-controlled hypertension and type 2 diabetes and was taking insulin, ramipril 5 mg, amlodipine 5 mg and aspirin 75 mg for a few years. His general practitioner had commenced him on 300 mg of quinine for symptoms of leg cramps 10-12 weeks before admission. He was a non-smoker and consumed about six units of alcohol per week and was not taking any herbal medicines. There was no history of any travel abroad.

On examination he had no signs of chronic liver disease, was febrile (38ºC), jaundiced and had generalized maculopapular rash (progressed to exfoliative dermatitis). Central nervous system, cardiovascular, respiratory and abdomen examination was unremarkable. With a possible diagnosis of cholangitis and hepatitis he was commenced on antibiotics and insulin sliding scale. Quinine and all other drugs were stopped. The blood picture showed hemoglobin (Hb) 14.5 g/dL, platelet (Plt) 137 x 10^9/L, white cell count (WCC) 15.44 x 10^9/L, eosinophils 2.47 x 10^9/L (0-1), C-reactive protein (CRP) 38, bilirubin 210 umol/L, alanine transaminase (ALT) 173 IU/L and alkaline phosphatase (ALP) 230 iu/L. Serology for hepatitis (A, B, C), brucella and leptospira was negative. Stool was negative for ova, cyst and parasite. Blood and urine cultures were sterile. Autoantibody and vasculitis screen was negative. A computed tomography scan of abdomen showed normal gall bladder, common bile duct, liver, spleen and pancreas. Liver biopsy [Figure 1] revealed portal tract fibrosis, cholestasis, a mixed infiltrate with prominent eosinophils and presence of non-caseating epitheloid granulomas. A diagnosis of drug-induced granulomatous hepatitis and DRESS syndrome was made. He gradually made a good recovery with supportive treatment. His blood counts returned to normal levels at four weeks with Hb 14.1g/dL, Plt 177 x 10^9/L, WCC 9.69 x 10^9/L, eosinophils 0.30 x 10^9/L, CRP 6, bilirubin 16 umol/L, ALT 45 iu/L and ALP 65 iu/L. He is back on his regular medication (insulin, ramipril, amlodipine and aspirin) and remains well at six months follow-up.

DRESS syndrome sometimes also known as hypersensitivity syndrome is a serious and potentially fatal adverse drug reaction which starts within eight weeks of initiation of the offending drug. The characteristic features include fever, dermatitis, internal organ involvement, hematological abnormalities (eosinophilia > 1.5 x 10^9/L) and lymphadenopathy (> 2 cm). Various drugs such as antiepileptics, nonsteroidal anti-inflammatory drugs, allopurinol, sulphonamides and antibiotics have been associated with DRESS syndrome. DRESS syndrome is associated with significant morbidity and a mortality of about 10%. Treatment is symptomatic and supportive and early recognition and withdrawal of the drug is essential for better prognosis.

Our patient presented with fever, skin rash and jaundice; and had eosinophilia, thrombocytopenia and granulomatous hepatitis. The symptoms started within 8-10 weeks of initiation of quinine, improved after withdrawing quinine and there was no other identifiable cause for his condition. On Naranjo scale it scored 5, classifying it as a probable adverse drug reaction. Hematological abnormalities associated with quinine are due to quinine-dependent-antibodies, however, eosinophilia has not been reported in the past and granulomatous hepatitis is not common. Quinine remains an important and lifesaving drug (falciparum malaria), however, physicians should be aware of the possibility of DRESS syndrome and quinine should be added to the list of drugs associated with DRESS syndrome.

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