

LETTER TO THE EDITOR

REVIEW OF HANDBOOK OF ACUTE MEDICINE

Dear Sir

We write to you regarding the review of Handbook of Acute Medicine⁽¹⁾ by Dr Vernon Oh that was featured in the December issue of SMJ. We have taken note of some of his valid comments and are heartened that he has at least enjoyed several chapters of the Handbook.

It is however a surprise to us the reviewer mentioned that the book does not declare its objectives clearly at the outset. We believe we have set out our objectives concisely in the "Preface to the Fifth Edition". We are also somewhat incredulous that he also reviewed the weight of the book as well as the potential reader whom he describes as "somewhat ignorant, sometimes diffident ... harrassed or all three," the relevance of which we will leave to the reader to decide.

In our opinion, a credible reviewer should read a book carefully from cover to cover before writing his comments. We therefore take serious umbrage of the fact that he has either not read or misread the following texts. Specifically:

1. The reviewer indicated that "a note should warn against pralidoxime in Carbamate poisoning". The warning is there on Page 93 para 5, item (e) - Pralidoxime (PAM) ... contra-indicated in carbamate poisoning.

2. The reviewer asks "How is salicylate an antidote for anti-cholinergic toxicity?" The antidote for anti-cholinergic compounds according to the American Hospital Formulary Service, Drug Information 1992 is Physostigmine salicylate which is the salicylic acid derivative of physostigmine (page 87 - Table II). This is the same mistake that a very harrassed house-physician made when she misread the antidote as Physostigmine and salicylate. One would have expected the learned reviewer to know better.

Current medical literature also does not support some of the reviewer's comments. He mentioned cardiogenic shock as "clinically hopeless". Some papers report on the efficacy of emergency coronary artery bypass grafting and percutaneous transluminal coronary angioplasty in reducing mortality in certain patients in cardiogenic shock^(2,3). He also considered amrinone as a useless inotrope and restricted the role of aminophylline in pulmonary edema to those intolerant of opiates. This is contrary to the American Heart Association's latest Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiac Care drawn up in February 1992⁽⁴⁾. In these guidelines, amrinone was to be considered for patients with pump failure and acute pulmonary edema resistant to first and second line therapies. Aminophylline has been found to be effective in those with acute bronchospasm associated with

severe heart failure (cardiac asthma).

The reviewer also questioned the role of H₂ blockade in those with anaphylactic reactions. We have reserved H₂ blockade as a therapeutic modality to be considered in patients with severe anaphylactic reactions refractory to standard treatment. Studies have shown that pretreatment prior to Histamine release with both H₁ and H₂ antagonists could prevent or blunt adverse cardiopulmonary responses⁽⁵⁾.

We have no pretensions that our Handbook rival or replace larger texts published in Oxford mentioned by the reviewer. We contend that at 2.00 am in the morning it is difficult for the house-physician to think in terms of "mass units/kg body mass" let alone to quote Thomas Henry Huxley 1877.

Finally, readers may wish to know that we have gathered valuable feedback not only from your reviewer but several of our enlightened readers. These will be incorporated in a reprint which is due in the middle of 1993.

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ANSWER TO ELECTROCARDIOGRAPHIC CASE

Diagnosis: Pericardial effusion

DISCUSSION

This 12-lead electrocardiogram shows low QRS voltage in all the limb and precordial leads with flattened or inverted T wave. Several definitions have been used to define low voltage. These include: (i) the sum of QRS amplitudes in the six standard leads <30 mm and <60 mm in the precordial leads or (ii) QRS amplitude of 5 mm or less in each of the standard leads or <10 mm in each of the precordial leads⁽¹⁾. The differential diagnosis of these electrocardiographic changes include pericardial effusion, chronic constrictive pericarditis, myxedema heart disease, diffuse myocardial disorder (eg. haemochromatosis, amyloidosis, scleroderma), pleural effusion and chronic obstructive lung disease⁽²⁾. Reduced voltage occurring in the frontal leads only may however be due to a normal variant⁽³⁾. Reduced voltages in the precordial leads only may be due to obesity or a left-sided pneumothorax or pleural effusion⁽³⁾. It is however important to exclude understandardisation as a technical error for the cause of low voltages. The low voltages in pericardial effusion is however a weak predictor of the amount of effusion and is sensitive only in patients with large effusion⁽¹⁾.

The electrocardiographic changes in this patient was due to cardiac tamponade occurring in a patient with hypothyroidism. In general, pericardial effusion and tamponade is uncommonly due to hypothyroidism^(4,5). One important feature of pericardial effusion associated with myxedema is the presence of bradycardia. Patients with large pericardial effu-

sion and tamponade usually have tachycardia instead⁽⁶⁾. Myxedema should however be suspected when flattening or shallow inversion of many T waves are seen without comparable ST segment displacement. Its cardinal characteristics include low to inverted T waves in all or many leads, low voltage, sinus bradycardia and prolonged QT⁽⁷⁾. Pericardial effusion is a common cardiac manifestation of myxedema but a recent paper suggests that it may not be as common as previously thought⁽⁸⁾. It is rarely associated with cardiac tamponade⁽⁹⁾. The management of pericardial effusion depends in part on the aetiology. In this patient pericardiocentesis was performed to relieve the cardiac tamponade. Thyroxine replacement therapy was given once the diagnosis of hypothyroidism was confirmed.

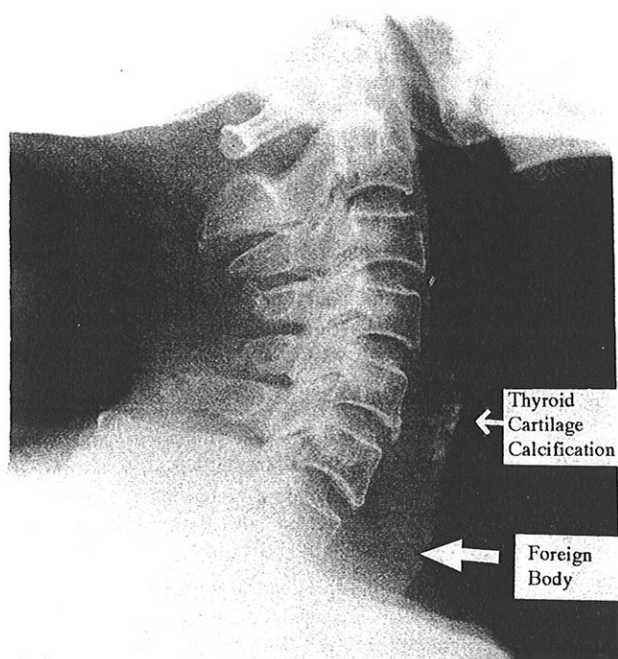
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CORRIGENDUM

MIGRATORY FISH BONE IN THE THYROID GLAND⁽¹⁾

Fig 1 - The plain lateral neck radiograph showing a linear opacity of a foreign body at the C6 - C7 level that appears outside the upper oesophagus.



In Fig 1, the calcified laminae of the thyroid cartilage has been erroneously labelled as the foreign body. The corrected Fig 1 is reproduced herewith.

We apologise for the oversight.

Our sincere thanks to Dr Wilfred C G Peh, Dept of Diagnostic Radiology of the University of Hong Kong for bringing this error to our attention.

-The Editor

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