ABSTRACTS


A mixture of pure amino acids in 8 per cent solution has been given an extensive clinical trial parenterally. Few toxic effects have been noted with this solution. The incidence of nausea and vomiting, reduction in blood carbon dioxide content and rise in urea nitrogen, and the excretion of unchanged amino acids is recorded. The case report of one death of obscure origin, possibly from acidosis, is presented.

A rate of administration of 400 cc. per hour appears to avoid toxicity except in rare cases of nausea and vomiting. Lowering of blood carbon dioxide content and rise in urea nitrogen occur with more than 2,000 cc. of 8 per cent solution per day. There is no evidence of renal damage.

An infusion of 1,500 cc. of this solution provides the average daily requirement of 15 Gms. of available nitrogen, and about 350 calories is furnished.

Emaciated patients who cannot afford to lose from their fat stores can only be treated for short periods by this technic unless some calories are taken by mouth. Mention is made of the importance of evaluation of cardiac reserve. Even patients on the border of failure can tolerate 400 cc. per hour of amino acids. Supplementary glucose is given by eysis so that it will not be so readily absorbed, should signs of heart failure supervene. Amino acids should not be given when there is evidence of renal damage in view of the acidosis and the rapidity of urea nitrogen accumulation in the blood, which results even with normal kidney function. 17 references.


A clinical study has been made of the effect of postoperative treatment with a mixture of pure amino acids on the recovery from partial gastrectomy of 21 ulcer patients, as compared with 32 similar cases handled in the same way, but not given the amino acid mixture.

One-fifth of the controls appear to do less well than the amino acid treated cases. However, the results in the two groups are similar enough to raise the suspicion that routine, unconsidered postoperative therapy with hydrolysates or amino acids may be in large part unnecessary.

The criteria of clinical benefit used are evaluated, as are the indications for high nitrogen intakes before and after operation.

M. F. P.


Intravenous oxygen was administered to 2 patients in shock and to 4 patients who were not in shock, and were considered to be in relatively stable cardiorespiratory condition. Clinical observations alone were made on the last 2 patients. Clinical and laboratory studies were done on the first four.

One experiment was done in vitro to approximate the amount of oxygen absorbed by the venous blood during the passage of the gas bubbles from the intravenous needle to the lungs.

No definite clinical changes were noted in the first patient who was in
shock. There was possible clinical improvement that could be attributed to the oxygen in the second patient in shock. Moreover, there was no clinical evidence of pulmonary embolism nor of systemic hypoxia. In the other 4 patients there was a marked and consistent decrease in the oxygen saturation of the arterial blood. This decrease was evidenced by actual arterial oxygen saturation determinations, symptoms of systemic hypoxia and by electrocardiographic changes. Three of the last 4 patients had clinical evidence of pulmonary embolism.

Studies on the passage of oxygen bubbles through a sample of venous blood, at rates comparable to intravenous administration, failed to show any appreciable absorption of the gas by the blood.

The amount of oxygen absorbed by the venous blood from the time of its introduction into the vein until its passage to the smaller pulmonary arteries, is insignificant. The gas bubbles coalesce in the veins. Therefore, they are of considerable diameter upon their arrival at the smaller pulmonary arteries and occlude these vessels.

The resulting gaseous pulmonary embolism causes decreased pulmonary circulation, arterial hypoxemia, and systemic hypoxia. These effects are probably intensified by reflex pulmonary spasms and possibly also bronchospasm.

It is possible that some of the ill effects of pulmonary embolism and thereby arterial hypoxia may be alleviated by the administration of atropine and papaverine before intravenous oxygen is given. Likewise a state of shock or of anesthesia may diminish these reflexes and thereby permit the oxygen to be given.

In its present status the value of intravenous oxygen as a therapeutic measure is doubtful. Because of pulmonary embolism it is definitely hazardous. 11 references.

M. F. P.


These studies have evolved the picture of a complete neuromotor pattern for reflex stimulation of respiratory centers during surgery. Respiratory response to surgical trauma has been demonstrated to be segmentally neuromotor and to occur whatever the anesthetic agent used. In general, selective reflex stimulation of either inspiratory or expiratory neurons and intensity of response are directly related to the neurologic segment and the autonomic plane or structures within which the stimulus originates.

Reflex response of these accessory muscles of respiration is of both a tonic and a superimposed rhythmic type of contraction. The first is completely independent of and dissociated from the rhythmic respiratory cycle. The second is a succession of intermittent rhythmic, forceful contractions of accessory muscles during the expiratory phase of respiration. There may be a tonic contraction of accessory muscles of respiration both during the induction phase of anesthesia and also as reflex response to surgical trauma.

The intensity of respiratory response to trauma reveals an increased gradient of activity from the periphery of the body toward the central zone. This is true in both somatic and visceral planes. Reflex response of respiration to trauma as noted in these studies is dependent on stimulation of end organs which lie in relation to nerve endings in areas having repre-