LEUCOCYTE ASCORBIC ACID LEVELS IN PATIENTS WITH MULTIPLE INJURIES

Sir,—It is known that ascorbic acid is concerned in the binding of collagen. In its absence there can be delay in wound healing and healed wounds can break down. This is true of bone and cartilage as well as soft tissue (Booth and Todd, 1970; Editorial, 1969; Schwartz, 1970). Ascorbic acid is concentrated in healing wounds and the maximum effect of any deficiency becomes noticeable between 5 and 14 days after the injury (Antonowicz and Kodicek, 1968; Kirchheiner, 1969; Schwartz, 1970). In cases of multiple trauma, especially those severe enough to require ventilation, there is a great deal of tissue damage. It is possible, therefore, that the intake of ascorbic acid might be deficient in the early stages, resulting in impaired or delayed healing. Deficiency is especially likely when nutrition has to be maintained by the administration of fluids and electrolytes intravenously during the early period.

In a pilot study we have tried to discover any drop in ascorbic acid levels and assess the contributing factors in traumatized patients using the leucocyte ascorbic acid (LAA) level. This is a much more accurate guide as to the body stores of ascorbic acid than the serum level (Booth and Todd, 1970; Denson and Bowers, 1961; Schwartz, 1970) and, indeed, the leucocytes can show adequate stores even when there is total absence of ascorbic acid in the serum.

The LAA was measured by the method of Denson and Bowers (1961) in 12 patients (11 males and 1 female) admitted to the artificial ventilation unit of the Edinburgh Royal Infirmary with crushed chest injuries. None were suspected of having pre-existing ascorbic acid deficiency. Nevertheless, on admission 6 were found to have LAA values below the normal range (20-57 \( \mu g/10^8 \) WBC); 3 were in the range 10-20 \( \mu g/10^8 \) WBC and 3 were below 10 \( \mu g/10^8 \) WBC, the lowest being 3.8 \( \mu g/10^8 \) WBC in a 66-year-old male.

In 6 patients further estimations of LAA were made 7 and 14 days after admission: 4 showed a drop in LAA levels over the first week; the 2 who showed rises had the lowest values on admission (6 and 6.1 \( \mu g/10^8 \) WBC) and in both the values after the first week were still below normal.

During the second week the results were more variable, 3 cases showing a rise of LAA over the level after 1 week, 1 remaining unchanged and 2 cases continuing to fall reaching 11.1 and 6.4 \( \mu g/10^8 \) WBC respectively.

The ascorbic acid requirement might be expected to be proportional to the severity of the injury, but there are many modifying factors. Pre-existing deficiency of ascorbic acid is not unlikely in the elderly (Brocklehurst et al., 1968) and in patients with chronic disease, malignant or non-malignant (Booth and Todd, 1970; Editorial, 1969; Windsor et al., 1972). Even after injury a number of factors may increase the ascorbic acid requirements including surgical operations, severe burns (King, 1968), oxygen therapy (Shanklin et al., 1967) and infection (Editorial, 1969). Drug therapy, including tetracycline (Windsor et al., 1972) and barbiturates (Booth and Todd, 1970), has also been incriminated as a cause of low LAA levels.

The patients studied by us had all suffered severe trauma and the additional complicating factors of emergency surgery, infection, antibiotic therapy, raised inspired oxygen levels were present in varying degrees, causing an increased requirement for ascorbic acid. Gastrointestinal ileus resulting in impaired intake of ascorbic acid was also frequently present. These factors varied so greatly in duration and severity that no assessment of ascorbic acid requirements could be made.

The 2 patients who showed a continuing drop in LAA values over the second week were the 2 with the greatest severity of trauma. However, both patients had intestinal ileus throughout the period, whereas the 4 with no drop in LAA over the second week had regained normal intestinal motility by that time. The results, while suggestive of a relationship between LAA depletion and the severity of trauma, may merely indicate that nutritional normality was restored in the 4 patients but not in the 2 who showed the fall.

These results suggest that ascorbic acid deficiency may be more prevalent than suspected among patients in an intensive care unit. Considering how easy it is to correct any deficiency we believe further work is indicated to elucidate the problem more fully.

Acknowledgements. We would like to thank Dr Percy Robb and the staff of the Department of Clinical Chemistry of Edinburgh Royal Infirmary, for carrying out the estimations of leucocyte ascorbic acid.

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REFERENCES


TEST OF A FUEL CELL OXYGEN ANALYSER

Sir,—We read with great interest the recent article by Drs Torda and Grant (Brit. J. Anaesth., 1972, 44, 1108) about the use of a fuel cell oxygen analyser. In the Binnengasthuis, Amsterdam, we have been using a Teledyne oxygen meter coupled with a Capnograph carbon dioxide analyser for the continuous gas monitoring in a circle system.

Gas samples are taken as close to the patient as possible via a narrow-bore tube inserted into the top of a Cobb's connector. The sample passes first through the Capnograph oxygen meter and is finally returned to the circle immediately prior to the carbon dioxide absorber.

The use of the oxygen meter coupled with a Capnograph is of great advantage to the anesthesiologist, since the oxygen and carbon dioxide levels can be continuously monitored and the oxygen requirement of the patient can be calculated by the use of the equation given by Meehan and colleagues (Anesthesiology, 1964, 25, 704).
Because of the slowness of recording of the oxygen meter, the readings do not show the variations in the oxygen concentrations of the inspired and expired gases but rather give a mean value of the oxygen concentration. Water vapour does not cause any problems as the gas is dry before reaching the oxygen meter.

The main advantage of using this system is that when low gas flows are used in a circle system one has an extra check that the patient is receiving a sufficiently high oxygen concentration in the inspired gases.

Two main types of retard are produced commercially: (1) variable orifice type; (2) diaphragm and spring type.

The variable orifice type produces a constriction which impedes the expiratory flow, thus prolonging the time necessary to deflate the lungs. Inspiration occurs before the lungs are completely deflated and thus the end-expiratory pressure is kept at a positive value. The pressure on either side of an orifice is proportional to the degree of compression of the spring. During expiration the spring exerted by the spring expiration occurs freely until the airway pressure falls below the spring pressure and then expiration ceases.

A serious disadvantage of this type is that flow is unidirectional and if inserted the wrong way round expiration cannot be incorrectly fitted. The retards produced by Philips and the latest model produced by Cape Engineering Co. are of this type. The variable orifice and the diaphragm and spring type produced by Cape Engineering Co. are identical in appearance on the outside and as the flow through the former can occur in either direction it could be used either way round depending on the type of connection on the ventilator.

If the expiratory limb from the patient is inserted into a British Standard female connection on the ventilator the latest Cape model would work satisfactorily. However, the inspiratory and expiratory connections can be interchanged and thus it is possible to have a British Standard male connection at the expiratory port. In these circumstances, the Cape retard could only be inserted in reverse and might well prove fatal.

The connections at either end of the Philips retard are interchangeable but one would have to be aware of the danger of reversing the direction of flow before thinking of changing the connections.

This very real danger could be solved by designing a retard with two diaphragms and springs in parallel, each allowing flow in a direction opposite from the other.

Sir,—In reply to the letter from Drs Gamble and Coppell, I make no observations upon their description of the two types, but would like to refer you to our application sheet and to the letter by Dr John Zorab and myself (Brit. J. Anaesth. (1968), 40, 918); perhaps they will help to explain their different functions and use.

Their comments that both the Cape Expiratory Resistance Valve and the Cape Positive End Expiratory Pressure control valve are identical in shape and size is correct; however, both are clearly labelled and have different coloured control knobs; black for the former and grey for the latter.

With regard to their observations that they could be incorrectly fitted to Cape ventilators, this is not so. We have been very careful to ensure that these retards cannot be incorrectly fitted to either the Cape Ventilator Mk II, the Cape-Waine Anaesthetic Ventilator Mk II and IIA, the Cape-Waine Multipurpose Ventilator or a modified Cape Ventilator Mk I or Cape-Waine Anaesthetic Ventilator Mk I fitted with inlet and outlets in accordance with BS 3849, providing the modifications were carried out by this company or their accredited agents.

Their comments that the inspiratory and expiratory connections could be interchanged on Cape machines is incorrect. For these machines or the retards that is at fault, the fault and the responsibility lies with the person using the retards incorrectly.

I hope that the foregoing will, in some way, answer their comments and that users of both retards will not feel they have a hazard on their hands.

Before leaving this subject, I should like to point out that the retards can be used on the Cape Bristol Ventilator System. It is intended that the retards be fitted on the outlet side of the expiratory filter.

The retards will fit both the inspiratory and expiratory ports on the sterilizable heads as both the inlet and outlet are 22-mm male tapers to BS 3849. However, they have special recesses to take the locking female connectors attached to the breathing tubes. It will be found that if the retards are fitted on either the inlet or the outlet tapers, the female locking connector will not engage with the retard and therefore cannot be incorrectly fitted.
I would like to qualify the terms "expiratory resistance" and "end expiratory pressure" (PEEP). The following is a simple explanation of the two terms:

1. Expiratory resistance is achieved by introducing an obstruction to the expiratory flow in the form of a variable orifice.

   The effect is to slow down the expiratory flow and the size of the orifice can be adjusted so that flow is maintained at a reduced rate throughout the expiratory period.

   With this method it is not possible to hold a positive pressure during the latter part of the expiratory period but the control can be adjusted so that pressure does not return to atmospheric (zero on the pressure gauge) at the end of the expiratory period. This is known as maintenance of a residual positive pressure during expiration.

2. End expiratory pressure is achieved by introducing an obstruction to the expiratory flow in the form of a spring-loaded valve with variable tension on the spring.

   A certain expiratory pressure is required to lift the valve and, when this pressure is no longer reached, the valve closes and positive pressure is then held to the end of the expiratory period in the form of a plateau. This has come to be known as PEEP—positive end expiratory pressure.

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MATERNAL HYPOCAPNIA AND THE FETUS AT SECTION

Sir,—I would like to offer some criticisms of the paper by Drs Peng, Blanato and Motoyama on this topic (Brit. J. Anaesth. 1972, 44, 1173 (November)).

The technique of ventilation used (4 l. total gas flow. Ventimeter/Ventilator set to provide a minute volume of approximately 10 l.) was such that the anaesthetists could not have known the proportion of oxygen in the inspired mixture. The failure has possible relevance to the fact that the mean value of P02 in the control patients was 14 mm Hg less than that in the group receiving carbon dioxide—accepted that the difference was not significant, it is surely suggestive, especially as the difference in the mean values of P02 in umbilical artery and vein blood respectively was of the order of 5 or 6 mm Hg. Expressed another way, it could be claimed that the major factor which promotes a difference between the two groups in respect to the transplacental gradient of oxygen tension (maternal artery minus umbilical artery) is the level of the oxygen tension in maternal blood. Carrying this argument one stage further, application of the Hellegers and Schuefer (1961) nomogram to the data presented in table II reveals that the difference in content of oxygen (per 100 ml blood) in the umbilical vein blood and that in the umbilical artery blood is about identical in the two groups. Thus it could be said that if the mean rate of umbilical blood flow is the same in the two groups the mass transfer of oxygen across the placenta is the same.

Now the authors claim to have demonstrated that the umbilical blood flow in the hypocarbic group is approximately 30% lower than that in the CO2-rebreathing group. This contention is based upon two others: firstly, their derivation of the "coefficient of foetal oxygen utilization"; a figure which is obtained from a knowledge of the oxygen content of umbilical vein and artery blood (the authors provided us with data respecting only oxygen tension). As I have pointed out, the means of the arteriovenous differences (CUV—CUA) are the same in the two groups, so any difference in the coefficient (which is expressed as (CUV—CUA/CUV X 100) must be reflective solely of the differences in oxygen content of umbilical vein blood, and this in turn could well be a function simply of the relatively higher maternal artery Po2 in the CO2-rebreathing group. Secondly, the authors' contention that there is a relatively low umbilical blood flow associated with maternal hypocapnia is based upon the assumptions that "there is no oxygen lack in (the CO2-rebreathing group) and that the oxygen consumption is equal in both groups". I find the latter difficult to understand as surely it is the authors' thesis that in the presence of maternal hypocapnia the foetus is rendered hypoxaemic.

The reference to the effect of blood pH upon the maternal oxyhaemoglobin dissociation curve is surely irrelevant to the discussion: the Po2 in maternal arterialized blood, even at the placental site, would, I suggest, be unlikely to be less than 100 mm Hg, and thus the possibility of induced shifts in the dissociation curve requires little consideration. Further—and possibly more germane—to the matter of the effect of a shift in the oxyhaemoglobin dissociation curve, one could make the point that as the pH of cord blood was higher in the hypocarbic group than in the CO2-rebreathing group, the ability of foetal blood—in cases in the former group—to carry oxygen at a defined Po2 is increased and thus there is a potential for total oxygen transfer across the placenta to be greater in the hypocarbic group.

Finally, I consider it worth drawing attention to the fact that the mean differences in base deficit between the hypocarbic and the hypercarbic groups, as observed in cord blood assays (UV difference 1.9 mequiv/l.; UA difference 2.3 mequiv/l.) were of the same order as that recorded between the mean values obtained from maternal arterial samples (1.1 mequiv/l.). Incidentally, I used the term "hypercarbic group" with intent, as I do not accept that a carbon dioxide tension of 39.3 mm Hg is normal for the pregnant patient, although I grant that it is nearer to the normal value (usually accepted as being 32-34 mm Hg) than is 23.0 0 mm Hg.

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REFERENCE