necropsy a characteristic finding is disease of small blood vessels of the sort we've seen in the patient we are now discussing. Why the red cells are destroyed is really anybody's guess, but one hypothesis is that they are destroyed in some way by contact with the damaged vessels. Whether this is a result of contact with necrotic tissue, or as the result of intimate contact with fibrin deposited in the vessels, we don't know.

Professor BOOTH: Does this imply that when you see crenated and damaged red cells in the peripheral blood there is fibrinoid necrosis, and that you're therefore dealing with severe hypertension, or accelerated hypertension, or malignant hypertension, or whatever you call it?

Professor DACIE: We believe this is so, but there are many other causes of this blood picture than malignant hypertension. One such cause is thrombotic thrombocytopenic purpura—or Moschcowitz's disease—where the blood-vessel changes are widespread and not confined to the kidneys. We see, too, a similar picture in cases of carcinomatosis where carcinoma cells are growing in the blood vessels or in the spleen or bone marrow.

Dr. DOLLERY: May I ask a question of our visitors? What do they think is the difference between benign and accelerated hypertension. If you compare two people with similar pressure levels, one of them may destroy his small arterioles and die with uraemia, while the other goes on for years without suffering severe damage to small blood vessels.

Professor DOYLE: This is a very difficult question to answer. There is a good deal of evidence to suggest that the higher the blood pressure the more likely you are to find fibrinoid change and thrombosis in the blood vessels. Another factor is the speed at which these changes develop; changes rather like this occur rapidly in acute nephritis and in eclampsia. The more rapidly the disease process progresses the more likelihood there is of vascular damage. There is some evidence that it is possible to render blood vessels more sensitive to the changes of high blood pressure. Asscher and his colleagues³ irradiated the arteries of rats and found increased sensitivity. I don't think an autoimmune factor is beyond the realms of possibility. Evidence for this at the moment is not strong.

Professor BOOTH: Well, I think that's a very clear statement of the present problem.

We are grateful to Professor J. P. Shillingford and Dr. E. D. Williams for assistance in preparing this report, and to Mr. W. Brackenbury for the photomicrographs.

REFERENCES

- ¹ Kincaid-Smith, P., McMichael, J., and Murphy, E. A., *Quart. J. Med.*, 1958, 27, 117.
- ² Kline, I. K., Kline, T. S., and Saphir, O., *Amer. Heart J.*, 1963, 65, 446.
- ³ Asscher, A. W., Wilson, C., and Anson, S. G., *Lancet*, 1961, 1, 580.

NEW APPLIANCES

A Means of Speaking for Patients with Cuffed Tracheostomy Tubes

Dr. R. M. L. WHITLOCK, formerly registrar, Acute Respiratory Unit, Auckland Hospital, Auckland, New Zealand, writes: Cuffed tracheostomy tubes are used to protect the lower airways in conditions complicated by bulbar paralysis and to facilitate prolonged intermittent positive-pressure respiration (I.P.P.R.) in the treatment of respiratory failure. Patients with these conditions are often unable to write because of paralysis or limb injuries and few people are skilled at lip reading. Because of the need for another

means of communication a simple tracheostomy-tube attachment has been developed.

The attachment (Fig. 1) consists of an F.G. 8 or 10 disposable plastic suction catheter through which air or oxygen is passed to blow on to the vocal cords. The catheter passes through a hole bored in the lower edge of the flange of a Portex plastic tracheostomy tube (Portland Plastics, Great Britain) which has a detachable inflatable cuff at the distal end. It then runs below the tube until it curves round one side to direct the tip upwards towards the vocal cords. Its curved portion is held in position by the edge of the cuff. With the catheter in the correct position (Fig. 2) a gas flow of 5 litres per minute is sufficient for distinct speech in the average adult without discomfort. The supply can be from a small air compressor or an oxygen cylinder with a pressure-reducing valve. To avoid drying of the mucosa and discomfort to the patient it is preferable to warm and humidify the gas, especially if oxygen is used. A pressure-relief valve somewhere in the supply line is a desirable safety measure. The gas supply should not run continuously but only when the patient wishes to speak. If a small electrical air compressor is used the flow rate can be pre-set and the compressor switched on by a microswitch which can be operated by any movable part of the patient's body. Alternatively a nurse may switch on the gas supply.

This attachment is cheap and of simple construction, and does not interfere with tracheostomy management or I.P.P.R. It allows speech throughout the whole of the respiratory cycle, does not rely on I.P.P.R., and has no moving parts. (A specially constructed tracheostomy cannula that uses the patient's exhaled air through a sliding valve for speech has been described elsewhere,¹ but it can be used only for patients on a Bird respirator.)

A delay of three days after tracheostomy is recommended before attempting to use the attachment. This interval allows time for the tracheostome to become sealed by granulation tissue, thus preventing escape of air around the tracheostomy tube or, worse, into the tissues of the neck and mediastinum.

Clinical trials on seven patients have been encouraging with six successes, though one of these found the flow of air too uncomfortable after a successful trial. The failure occurred in a patient with a tracheostome larger than

FIG. 1

FIG. 2

usual which allowed the air to flow out round the tracheostomy tube more readily than through the vocal cords.

This speaking-aid not only makes communication easier but also relieves the patient from the frustration and fear of not being able to make his requirements known. There have been no complications from its use.

I thank Dr. M. Spence, medical officer in charge, Acute Respiratory Unit, Auckland Hospital, for permission to develop this device and for advice on its use.

REFERENCE

- ¹ Hessler, O., Rehder, K., and Carveth, S. W., *Anesthesiology*, 1964, 25, 719.