The instrument consists of a light plastic face piece with two 0.28 square inch Monel® metal screens (400 mesh) lying parallel over the breathing aperture. An annular manifold for sampling the pressure drop across the outer screen was placed between the two. As in any conventional pneumotachograph, a screen (in this case the outer) was provided to create a slight fixed resistance to the flow of respired air. Varying the angle at which gas impinged on this screen was found to produce inconsistent calibration for known flows. The inner screen was added to convert turbulent expired air into a laminar pattern before presentation to the outer recording screen. With this innovation, a linear response for flows well beyond those encountered in the infant could be obtained regardless of the incident angle of the gas stream.

The dead space has been reduced to less than 4 cc. by employing a very shallow mask and using a silicone plastic material (Sili Putty®) as the mask seal, instead of an inflated rubber ring. This substance proved excellent protection against leaks, held the mask closely to the skin without pressure being applied, and removed the necessity of fixing the mask with head straps. The effect of the dead space could be further reduced by introducing a steady flow of air or oxygen of up to 1000 cc./minute through a side tap in the mask. The volume of inspiration and expiration was not changed by this added steady flow, the effect being merely to change the baseline of the record. The unit as described had a resistance of 0.21 mm. H_2O/liter/minute. At an 8 liter-minute flow rate, the resistance was 1.7 mm. H_2O, a flow rate above that usually encountered in the infant at rest. For comparison, the resistance of a laboratory wet test gas meter, a type which has been used for measurement of infant respiration, was 15 mm. H_2O at the same flow rate.

This pneumotachograph mask has been designed to permit measurement of tidal volume and velocity of air flow during studies on neonatal respiration and infant resuscitation. The design can be modified for use in anesthesia circuits by the addition of a second manifold and valves for inspiration and expiration. Low resistance will be sacrificed by the use of valves, but the small dead space will not be appreciably altered.

Acute Effect of Low-Flow Extracorporeal Circulation on Cerebral Physiology.

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The simplified bubble oxygenator system developed for direct vision intracardiac surgery has been used to perfuse more than 140 patients at blood flow rates significantly less than normal cardiac output. When the electroencephalograms of these patients were compared with those reported in the literature, they appeared similar to patterns observed during hypocapnia, hypoxia, hypotension, and analagous circumstances. This investigation was begun to determine if these conditions exist during low-flow extracorporeal circulation.

Only data collected from 27 acyanotic patients with ventricular septal defects will be presented. This group includes patients between 8 months and 38 years of age. Following cyclopropane or thiopental-flaxedil® induction, anesthesia was maintained with thiopental-flaxedil®-nitrous oxide until the perfusion began. No anesthetic was given during the perfusion. Occasionally 50 per cent nitrous oxide was required to facilitate chest closure. Electroencephalograms were recorded periodically before the perfusion, continuously during the perfusion, and intermittently thereafter.

After performing a bilateral, sternal splitting thoracotomy, the surgeon isolated the left subclavian artery, and sacrificed the vertebral artery on that side. The scalp electroencephalogram electrodes usually were placed on the left side of the head. The subclavian artery was catheterized to provide an inflow tract from the oxygenator to the aorta. At this point the electroencephalogram indicated that, irrespective of age, there was an early stimulation and then a decrease in the frequency and the amplitude of fast (a and b) activity. Slow wave forms (c and d) appeared in tracings that previously had

not shown them, or were increased in amplitude when present before catheterization. These changes were correlated with the clinical anesthetic level and with a slight decreases in blood pressure. However, they were related most directly to the degree of hyperventilation intentionally produced by the anesthesiologist. Arterial samples taken from the subclavian catheter before perfusion indicated that the *p*H varied between 7.44 and 7.78, the $PaCO_2$ between 39.5 and 17.0 mm. Hg, and the oxygen saturation between 97 and 100 per cent.

When the vena cavae had been catheterized to provide for the return of blood to the oxygenator, partial perfusion was begun. No vessels were occluded. Pump flow rates ranged between 34.6 and 60.0 cc./Kg.wt./minute. While this volume was exchanged the heart continued to function normally. The blood thiopental concentration probably was reduced considerably by the volume of blood in the pump. Nitrous oxide was lost immediately through the oxygenator into the atmosphere. Blood pressure fluctuated little during this interval. The electroencephalogram changes previously noted now increased in magnitude. Fast activity decreased as slow activity increased in dominance. This was probably produced as a result of the differences between the blood of the patient and that of the oxygenator. Samples taken from the arterial limb of the pump at the time perfusion began indicated that the *p*H of the pump blood ranged from 7.46 to 7.68 and the $PaCO_2$ between 28 and 14 mm. Hg. The blood was 94.5 to 100 per cent saturated with oxygen. Venous pressure was increased slightly probably as a consequence of catheterization of the cavae and reduction in the rate of venous return by the controlled cross circulation.

Significant changes appeared in the electroencephalogram when total perfusion began at the time of caval occlusion. In the tracing which still contained fast waves, this activity disappeared at a more rapid rate than previously. Slow waves of amplitude and frequency suggestive of pathologic changes appeared immediately. When the cardiotomy was performed an even more severe change occurred. Maximum discharges of energy in the slow frequency range often reached amplitudes exceeding the limits of measurement. This pattern usually was followed by a reduction in amplitude and frequency of all wave forms until a classical pattern of severe depression appeared. Only slow low voltage waves could be seen in many of the tracings during this interval. These findings were correlated with the sudden change from normal cardiac output to the output rate of the pump, with the loss of ventricular blood volume, and loss of coronary blood flow into the wound or into the suction loop which returned into the pump. This consistent reduction in volume available for cerebral perfusion was accentuated if an unsuspected anatomical shunt, such as a patent ductus, was present. Venous pressure increased as the time complete control of venous return into the oxygenator began. The blood pressure probably reached its lowest levels when these variables operated synchronously.

The electroencephalogram patterns of each patient varied considerably during the period required to repair the ventricular defect. Many patients showed evidences of recovery of some fast activity. In others there was a greater depression of slow activity and in some the patterns disappeared and alternated with bursts of complete electrical silence. These changes probably were produced by fluctuations in the variables just reviewed, by the development of metabolic acidosis, or by the duration of perfusion at reduced flow rates. At the time the defect was closed the arterial system contained blood with a *p*H which ranged from 7.33 to 7.62, a $PaCO_2$ from 30 to 19.5 mm. Hg; and an oxygen saturation between 95 and 97 per cent. Electroencephalogram activity returned toward more normal levels after repair of the defect, or during closure of the cardiotomy wound, release of caval occlusion, or termination of the by-pass. Recovery appeared to be correlated with the severity of the change produced, the duration of maximum change and the alterations present in blood volume, blood pressure, cardiac efficiency, and with the degree of compensation from metabolic acidosis and hypocapnia. Most patients recovered a portion of their fast activity patterns, but all retained an increased amount of slow activity in comparison to their preperfusion control values.