the adrenalectomized animal, more liver glycogen is deposited than when either is administered alone(16).

These findings suggest that the stimulation of secretion of adrenal cortical hormone by exogenous glucose, in the normal individual, allows more of the administered glucose to be taken up by the liver and stored as glycogen, thus enabling the blood sugar level to return rapidly to normal. On the other hand, the inhibition of the adrenal cortex caused by hypoglycemia should tend to restore the blood sugar level to normal, probably by allowing glycogenolysis to take place.

There is an alternative explanation of the lymphocytosis after insulin, based on the finding that initially epinephrine administration causes lymphocytosis(17). This explanation is, however, partially negated by the fact that adrenodemedullated animals exhibit a normal insulin tolerance curve(18), and it is therefore unnecessary to assume that moderate hypoglycemia elicits epinephrine production.

Summary. The intravenous administration of 25 g of glucose in 50% solution was accompanied by a significant lymphocytopenia which reached its lowest level at ½ to 1 hour in 5 normal subjects and at 2 hours in the sixth. The intravenous administration of 0.075 unit of regular insulin per kilo of body weight evoked a significant absolute lymphocytosis which reached its peak at 45 to 60 minutes in 7 normal persons, and at 120 minutes in the eighth. The lymphocytopenia after glucose administration is inferred to be a result of adrenocortical stimulation caused by the change in the blood sugar level. The absolute lymphocytosis after insulin is similarly assumed to result from adrenocortical inhibition due to the moderate hypoglycemia. The discrepancy between our findings and those of others, who noted a decrease of lymphocytes following insulin, is explained by the small amounts of insulin used by us, thus avoiding shock and concomitant adrenocortical stimulation. This study suggests that the response of the adrenal cortex to hyper- and hypoglycemia serves to maintain homeostasis.

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A Small Bore Polythene Shunt to Prevent Mechanical Shock after Prolonged Cross-Clamping Thoracic Aorta.* (17930)

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Prolonged cross-clamping of the patent thoracic aorta distal to the left subclavian artery is uniformly followed by severe neurological and visceral damage(1,2,3,4,5). Initially, the injury is not dependent upon cardiac failure, but upon the effect of diminished circulation to those susceptible structures distal to the cross-clamped area. Later effects are consistent with a progressive reduction in an effective circulating blood volume as this fluid pools in the anoxic, hypotensive region(1). It is therefore necessary

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to deal satisfactorily with the problem of hypotension and tissue anoxia before extensive, time consuming surgery, on the thoracic aorta becomes feasible.

Experimentally, Carrel diverted the flow through a paraffined glass tube temporarily placed within the aorta or by a tube from the left ventricle to the descending aorta(3). Gross and Hufnagel, by refrigeration of the backs of dogs, lowered the metabolic rate of the tissues, and thus avoided neurologic damage in three dogs in which the aorta was clamped 45 minutes(4). Clinically, Crafoord has limited his operating time on the clamped aorta to about 30 minutes(5). Potts, Smith, and Gibson have recommended the use of a partially occluding clamp on the aorta(6). While all methods mentioned are appropriate solutions to a particular problem, none present the surgeon with an unencumbered field in which he can carry out prolonged procedures on the circumference of a bloodless thoracic aorta.

Experiments. Thirty-one mongrel dogs weighing 6-20 kg were anesthetized with 30-35 mg I.V. sodium pentobarbital, intubated and connected with a standard anesthesia machine delivering a high concentration of O_2. In an aseptic field the left chest was opened through the bed of the fourth rib supplemented when necessary by cutting the third and fifth ribs. The aortic arch, descending aorta and subclavian artery were widely dissected with division of the superior 2 or 3 pairs of intercostal arteries and the ligamentum arteriosum. A silicone coated polythene tube 12-15 cm long, with an inside diameter of .062″ for dogs under 10 kg or .085″ for dogs over 10 kg was then fashioned into a suitable form by molding in hot water. In some instances a flange of polythene had previously been affixed to the proximal end prior to use. The proximal funneled or flanged end of the tube was then inserted into the clamped subclavian artery, the aorta was then clamped and the turned up distal end of the tube passed between 2 sutures through a puncture wound into the distal aorta. Upon removal of the proximal subclavian clamp blood swelled the distal aorta. In 5 dogs continuous mean pressure recordings were obtained in the carotid and femoral arteries. The femoral pressures ordinarily dropped to levels of 20 to 30 mm Hg upon cross-clamping. The carotid pressure rose to 180-200 mm Hg. Upon opening the shunt the femoral pressure rose to 50-70 mm Hg and the carotid pressure fell to about 160-180 mm Hg. Over a period of 2 to 5½ hours the carotid pressure gradually fell to near normal levels of 120-140 mm Hg. The femoral pressure fell to 40-50 mm Hg.

In 26 dogs segments of aorta were removed and autogenous grafts implanted or "notches" made in the aorta and the remnant banded to produce a coarcted area(7). In no instance did the siliconed shunt thrombose. No supportive therapy in form of blood transfusions or stimulants was administered.

Results. In the control group weighing 8-17 kg, in which the aorta was clamped more than 30 minutes, 10 of 13 dogs had permanent spastic paralysis or died within 12 hours of mechanical shock.

In the "shunted" group weighing 6-20 kg, all clamped more than one hour, one 20 kg dog with a .085″ shunt in place died immediately with uncontrollable tachycardia, ventricular fibrillation and cardiac failure after slowly removing the aortic clamps.

This remains an unexplained phenomenon since it was a solitary occurrence among all the experiments performed. In the remaining 14 animals there were no instances of mechanical shock, or spastic paralysis. Four dogs showed slight stiffness of the hind legs which lasted less than 72 hours.

Discussion. The deleterious effect of cross-clamping of the thoracic aorta may be due to one or many factors. Shorr *et al.* demonstrated that vasodepressor substance is released by skeletal muscle and liver cells in response to anoxia(8). Fine *et al.* have pre-

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vented the irreversibility of hemorrhagic shock by ingenious cross perfusion of the liver(9). It is not our purpose to attempt to evaluate the factors involved in the development of spastic paralysis or irreversible shock, but rather to make another practical use of the fact that tissue damage due to arterial hypotension is related to the product of the degree of hypotension and the time that it persists.

In this instance one can by an inexpensive, readily procurable tube, which can be fashioned into the desired form at the operating table, make a temporary channel to divert blood into a temporarily hypotensive and anoxic area. Although such a small shunt does not return the deprived circulation to normal it enables one to bargain for the time necessary to accomplish a definitive procedure.

The use of similar devices to provide for diversion around smaller essential vessels, such as the coeliac, superior mesenteric or common femoral or hepatic artery, which are undergoing reconstruction might be indicated.

Conclusions. A small polythene shunt used to divert a portion of the blood around the cross-clamped thoracic aorta will temporarily prevent mechanical shock in dogs.

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Use of a Glass Edge in Thin Sectioning for Electron Microscopy.* (17931)

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The use of a steel microtome knife in cutting sections for electron microscopy has certain disadvantages, especially in the achievement and maintenance of a sufficiently sharp edge, which involve a considerable expenditure of time. The search for a knife material having homogeneity and hardness without excessive brittleness led to the development of a glass cutting edge. With it, we have been able to cut thin sections more consistently, easily, and rapidly than with steel microtome knives. Since the glass "knives" are obtained simply by breaking them from a strip of glass, the tedious and uncertain sharpening procedures necessary with steel knives are eliminated. Inspection with the light microscope reveals a smoother and, we believe, sharper edge. Fewer grooves or scratches are found on the face of a block of tissue after it has been cut with the glass edge. The glass knives are also quite inexpensive. The knives are made by breaking a strip 1½″ wide and about 12″ long from a sheet of plate glass approximately 3/8″ thick. Since the edges thus produced will form clearance facets, this fracture should be as smooth and straight as possible. A series of straight parallel scorings at 45° to the long axis are then made on each strip, 1″ apart, and on the opposite surface from the first scoring. The parallelograms thus outlined are broken off, producing a set of glass blocks, each of which has two cutting edges 3/8″ long, formed by faces meeting at a 45° angle (Fig. 1,A). Any competent glass cutter with standard equipment can produce these knives from stock plate glass for something less than 25 cents each. Straighter edges, however, can be more regularly secured with a diamond-point scriber having constant spring tension (designed by Robert C. Jackson of the Department of Biology Machine Shop), utilizing a controllable pressure device, such as an arbor press, to break off the knives.

The knife holder, as shown in Fig. 1, was

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