and peripheral stimulation. (2) Thyroxin potentiation of adrenalin pattern cell receptor relationship in the specific tissue or conducting mechanism of the heart. (3) The action of thyroxin itself on the heart. The action of ergotoxine and ergotamine should be useful in minimizing the action of the first two with little, if any, effect on the third. In addition, the vagal potentiation of ergotamine plays a definite role in the control of the tachycardia. This follows from the ergotamine-cholinesterase relationship, resulting in enzyme inhibition and consequent prolongation of acetylcholine action.

"... The tachycardia of thyrotoxico sis is not a compensating reaction but appears to be rather a fortuitous and undesirable event. Clinical evidence would seem adequately to substantiate this, as most observers concur that its presence, particularly if long continued, adds greatly to the gravity of the condition. In any event, regardless of the mechanism responsible for the beneficial results observed with this therapy, the present investigation brings substantial clinical evidence that this tachycardia can be advantageously influenced during the surgical case of thyrotoxicosis by the ergot alkaloids, ergotoxine and ergotamine."

R. B. S.


"In the treatment of shock, it should be the aim to restore vasculare tone and vascular volume to a normal state as rapidly as possible. The introduction of fluids such as saline, Ringer's solution and glucose, have been a notorious failure in maintaining such a condition. It, in most instances, is at best of only transitory value and is rapidly de-
and a consequent diffusion of plasma fluids into the surrounding tissue spaces. . . Fixed cells also give up water to their environment, and . . . there is a general disturbance of intra- and extra-cellular equilibrium of electrolytes as well as fluids.

"The shift of fluids causes a more or less rapid decrease in the venous return to the heart. It is believed that there is an attempt to compensate for this by constriction of the arterioles, probably from adrenalin action, and the blood pressure is temporarily maintained or increased by this means. It is, however, a vicious circle because the capillary blood flow becomes progressively slower and anoxia is added still further to increase cellular permeability. The best index of this series of events is the hemoglobin percentage." The hematocrit shows decreased plasma in proportion to cells except possibly in severe hemorrhage, vomiting, and diarrhea.

Cannon and Dale suggest a toxic histamine-like substance as the basis for changed permeability. Weil and Browne have studied the relation of the adrenal glands to shock because of the observation that adrenal atrophy or Addison's disease had some features like shock, particularly hemococoncentration and falling blood pressure.

Bram, Rose and Brown have shown that an adrenalectomized animal has little resistance to histamine as to toxic or physical insults.

"Through a single severe or repeated injury or infection the protective power of adrenalin is stimulated, and if this compensation is sufficient it maintains cellular permeability even in the presence of excessive histamine; if not, it permits the histamine or histamine-like substances released from macerated or otherwise damaged organs, or that present in the blood and normal tissues, so to affect the permeability of the capillaries and the cells of a certain organ (and this in man is, firstly, the intestinal tract, and secondly, the lungs) that large amounts of plasma and cellular fluid migrate into the pericellular spaces, leading not only to a reduction of blood volume and its increased concentration, but also to what may be called cellular dehydration which affects many tissues but particularly those mentioned above. There is also dilatation of capillaries and venules with stasis, and thus increased areas to be filled by a diminished volume of blood, anoxia, and all the other manifestations of shock.

"Saline infusions and blood transfusions may be of value in maintaining the blood volume over a period of time to permit the true defensive functions to reverse the actions. They should not be neglected but should be used with an appreciation of their limitations.

"A more direct attack at the fundamental basis of shock would seem more rational. If this be accepted as adrenal deficiency, substitution therapy as a prophylactic and cure would be indicated."

Adrenalin only increases the constriction of the arterioles and sustains the vicious circle. Cortical extract has, however, been used with some valuable effect. "In our laboratories we believe that we have at least in part such evidence (factual), but it remains to be proved conclusively that it is so." The synthetic products do not seem as satisfactory as the cortical extract.

"Best and his colleagues have concentrated serum to a fourth or less of its volume, and this has been used in place of saline infusions or whole blood transfusions." The results are most encouraging though still on an experimental basis.

To recapitulate:
1. "Shock is best detected by frequent and accurate estimations of hemoglobin or by the hematocrit."
2. A fall in blood pressure is a rela-
Abstracts


"The present report, which concerns 24 cases of secondary shock that were selected for systematic study, provides data sufficient to justify tentative conclusions that can be tested by other observers with more material at their disposal. . . . Differentiation of primary from secondary shock is easy with extremes of injury, when the wounding is of such severity that immediate transfusion is obviously required and delay would be dangerous, or when there are no wounds and transfusion would be both unnecessary and foolish. But in intermediate cases differentiation is only possible by allowing a period of observation; the degree to which rest in the recumbent head-low position combined with warmth and morphia produces improvement will depend on how far the symptoms are due to primary shock. . . . All the 24 cases . . . had either wounds so severe as to brook no delay in instituting transfusion or a blood-pressure which failed to recover to 100 mm. Hg. with rest, warmth and morphia . . .

"In general we have found that the blood pressure is the most reliable measurable factor for assessing the severity of secondary shock and that other clinical manifestations are variable and not quantitative. The pulse rate has not always been as rapid as it is generally assumed to be in severe secondary shock nor does it regularly rise in proportion as the blood pressure falls. . . . The mental state is no index of the severity of shock. . . . As to pain, all of our 24 patients had had morphia; pain is recorded as absent in 4, slight or moderate in 8 and severe in 11, and was not recorded in 1. Colour changes are common but in no way quantitative; 22 out of 24 patients showed obvious pallor and most had also cyanosis of the lips and nails. Extreme pallor in association with a relatively high blood pressure suggests intense compensatory vasoconstriction. The general temperature of all severe cases is invariably subnormal and the extremities feel cold. . . . Sweating was present in 15 cases but the amount was by no means related to the degree of shock. . . .

"The obvious treatment is to restore blood volume. Information is required as to the best fluid for this purpose, when to transfuse, in what amount and at what rate. From the physiological aspect whole blood is needed by those whose reduction in blood volume is due mainly to whole blood loss, and this is usually the case with those who are neither burned nor crushed and who are seen soon after wounding before complex haematological changes have occurred. We have analyzed our cases in relation to fluid, time, amount and rate but have used more plasma than blood with a view to obtaining information as to its efficiency as a volume-