At the sleep point there always occurred a drop of about an equal amount in both systolic and diastolic pressure. Whether or not this drop is responsible for sleep is a question which can only be satisfied by circumstantional evidence.

THEORY OF ANALGESIA

With the present data on hand, viz., Exhibits "A," "B" and "C" let us study the various physical chemical changes of state of solution and vapor tension of ether as it passes through the blood stream with the end in view to find out how its synergists may act in order to allow analgesia.

The boiling point of absolute ether, as we all know, is about 31. 5C. When ether is administered, it passes through the walls of the alveoli to enter into solution in the blood circulation in the lung tissues. The temperature of this blood is between 36 and 37 C., i. e., 1.5-2.5C above the boiling point of ether. On this account, an ether gas tension will develop thereby limiting the amount of ether which can enter the blood stream.

The point of maximum heat production (in the circulation) is in the end capillaries of the peripheral tissues where combustion takes place. The temperature here suddenly rises to over 38 C. It will therefore be easily understood that when the ether from the lung tissue reaches this point through the arteries, the gas tension will be enormously increased, both on account of lowering of pressure and the increasing of the temperature.

Ether, like alcohol, acts centrally and otherwise on the nervous system to cause a general vaso-dilatation thereby slowing the stream in the capillaries and reducing the metabolism and temperature. This is exemplified by our diastolic pressure reduction taking place on administration of absolute ether.

Volatile anaesthetic substances enter the blood stream at the lung capillaries much more slowly than does ether. If they are present in the ether administered, the vaso-dilatation caused by the ether will be replaced by vaso-constriction as soon as they gather in the peripheral circulation in sufficient concentration. That is, when we administer an ether together with these anaesthetic gases, we will have a short period of vaso-dilatation followed by vaso-constriction, and increased capillary combustion. During this vaso-dilatation, the heart will beat more rapidly due to the relief of pressure, but as soon as vaso-constriction begins to occur, the pulse slows with the increasing resistance.

I have already mentioned the fact that when ether only is present in the blood stream, there is an ether gas tension increase in the peripheral tissues which is lessened according to the resulting vaso-dilatation, by the reduction in metabolism as well as the relative approximation of blood pressure between arteries and capillaries. It can thus be seen that when the ether gas tension in the capillaries becomes very great, as it would in the above case, and these capillaries are constricted, there will be an enormous escape, and retention of ether into the surrounding tissue fluids, and the ether becomes concentrated in the lymph and tissue cells. When this concentration is sufficient the sensory nerve endings lying in the tissue fluid will become insulated, and no longer able to function. The motor nerve endings are not affected as they enter directly into the cells that they govern.

In the central nervous system, where the matabolism is not nearly so great, the ether will not localize. In other words, if the capillaries throughout the body are kept normally constricted, ether will localize in tissues according to their metabolism rate state and this localization produces localized analgesia by sensory nerve ending insulation.

Our blood pressure curves show that ethylene and carbon-dioxide, as we have already assumed, tend to equalize the circulation by lowering of systolic as well as raising of diastolic