

ing the labor. Chloroform is often administered at this stage, but surgical anæsthesia must not be produced; our objective points being to blunt susceptibility and relax a rigid os uteri. This accomplished, then we must cease: if we do not, we will produce complete relaxation and invite post-partum hæmorrhage. In ordinary surgical cases, however, hæmorrhage is lessened by using chloroform as an anæsthetic, but the accoucheur cannot avail himself of its coagulating properties. The reason of this is in part anatomical: the maternal arteries and veins at the placental site are not connected by capillaries. The pains of the second stage of labor are generally due to forced dilatation of the soft parts, and it is at this time that we usually administer chloroform. The method should be intermittent, that is, we should remove the inhaler as soon as the pain has passed off. A sort of numbness of the parts is all we wish to obtain, and this point reached is quite sufficient.

After the preliminary increase of arterial tension produced by chloroform, the circulation becomes slower, the leucocytes oscillate and are arrested first in the capillaries, then in the arterioles, and finally in the larger vessels. The red globules agglomerate and form *magnæ*, which disappear when the pulsations become normal. The blood-vessels, owing to the absence of vaso-motor impulse, become constricted in calibre, and if anæsthesia be carried beyond surgical limits, vascular areas, which were well marked when the circulation was active, grow paler and are gradually effaced. This last explains why cessation of hemorrhage is a signal of danger. Those coagulating and constricting properties, however, recommend chloroform as *the* anæsthetic in all operations on the eye. The same, too, has been turned to advantage in connection with Esmarch's bandage in the treatment of external aneurisms.

The number of deaths which have occurred under chloroform, and I may add ether, under the first stroke of the surgeon's knife, leads us to inquire—Is it or is it not better to commence to operate before surgical anæsthesia has been obtained? The gravest accidents from anæsthetics are cardiac and respiratory syncope, but each form is brought about by an entirely different mechanism. Cardiac syncope is the result of a complete reflex act, and is by far the graver of the two. The mechanism of production is the following: Through

shock to a sensitive nerve-fibre, a centripetal impression is carried to the rachidian bulb and there transformed into a centrifugal current which, on passing down the vagus, inhibits the heart in diastole. What makes cardiac syncope so dangerous is that the heart is already too feeble to empty its ventricles at each systole, the excito-motors of the heart are not in a physiological state, and are so thoroughly overpowered by vagus inhibition that they fail to respond. To use an Americanism—The excito-motors of the heart come up groggy, and are "Sullivaned" by the first pass, which causes powerful vagus inhibition. Let me correct what was an error in practice by saying, that vagus inhibition may readily be produced by faradization of the phrenic nerve by an electrical shock to any portion of the periphery, the patient being under chloroform; consequently, in either form of syncope, we should never employ the faradic current. From the foregoing data we must conclude that we should never commence to operate until surgical anæsthesia has been reached. Surgical anæsthesia may be defined as loss of consciousness, complete abolition of sensibility, of voluntary movements, and of reflex action in the nerves of the "life of relation." To guard more carefully against cardiac syncope, it is our duty to give our patient, before placing him on the operating table, a hypodermic of atropine as a vagus paralyzer, and, if you wish, a glass of good liquor as a heart stimulant. With these precautions and with pure chloroform properly administered, there is no danger of cardiac syncope, or contra-indication from heart disease.

In respiratory syncope the centripetal excitation is carried to the rachidian bulb and there arrested. The reflex act is not completed. Centripetal excitation is not transformed into centrifugal incitation. It is usually due to anæsthesia being too profound, and as a rule can be overcome by resorting to artificial respiration. As patients under morphine require less chloroform to produce anæsthesia, a hypodermic of this alkaloid is indicated as a prophylactic against respiratory syncope. Our hypodermic, then, should contain—morphia  $\frac{1}{8}$  gr., atropine  $\frac{1}{60}$  gr.

I can best conclude this paper by giving as a *résumé* the following practical rules of procedure, which I take from the *Gazette des Hôpitaux*, of Paris:—

1. The compress is to be preferred to all other