mind, by poisoning with woorari, and by injuries or lesions of the peripheral extremities of the pneumogastric nerve in the lungs, liver, stomach, or intestines.

3. *Diminished destruction of sugar.* If the sugar into which the glycogen formed in the liver is converted did not disappear from the blood, it would necessarily form one of the normal constituents of the urine. Hence it cannot be denied that the pathological presence of sugar in the urine may possibly depend in some cases upon a failure of the conditions under which the normal transformation of the sugar takes place. We are, however, in a great measure ignorant as to what these conditions are. A number of experiments make it probable that the sugar in the blood, under the influence of a ferment, is converted into lactic acid and glycerine, which undergo combustion, and so maintain the animal heat; and it is believed that, when this ferment is absent, the sugar is not decomposed, but is excreted in the urine. Whether this be the true explanation or not, many facts seem to show that diabetes may result from a morbid state of the blood. Thus the introduction into the blood of ammonia, ether, chloroform, or phosphoric acid, is followed by glycosuria; while the introduction of carbonate of soda will prevent it. Prout long ago observed a saccharine condition of urine as not an uncommon result of gout; and glycosuria has been repeatedly observed in cases of pneumonia, whooping-cough, and phthisis, which lead to deficient oxygenation of the blood.

c. It is not improbable that other wasting diseases are in their origin connected with some functional derangement of the liver. When there is derangement of the disintegrative functions of the liver, the blood and fluid effused from it become loaded with effete matter, as the result of which, the nutrition of the tissues is often impaired, and the body wastes. In *phthisis*, again, long before tubercle is deposited in the lungs, there is evidence of deficient assimilation of nutriment and imperfect sanguification—functions, in which we know that the liver is deeply concerned. Again, the protracted purulent discharge which usually precedes *waxy disease* may entail a hasty and imperfect sanguification, resulting in anæmia and the formation of an albuminous material little capable of organisation.

II. *Abnormal Elimination.*

In discussing the functions of the liver, I stated that bile is, in part, excrementitious, although this is probably far from being its chief use in the economy of digestion. In accordance with the belief, still held by many members of the profession engaged in practice, that the chief function of the liver is to excrete bile, the retention of bile in the blood and tissues is believed to give rise to serious symptoms. For example, when such symptoms as delirium, stupor, muscular tremors, subsultus, carphology, paralysis of the sphincters, coma, convulsions, a dry brown tongue, and other symptoms of the "typhoid state" supervene in any case of jaundice, in acute atrophy of the liver, or in the advanced state of cirrhosis, it is customary to attribute them to poisoning of the blood with retained bile. Experiments, also, have been performed on animals, with the object of showing that bile is a deadly poison. But that dogs should die, after injection into the cellular tissue of the bile of other dogs, admits of another explanation than that of the essential elements of bile being a poison. The injection of decomposing mucus would probably produce a like result. Pure bile, from which all mucus has been removed, has been repeatedly injected into the large veins of dogs by Frerichs and other observers, without any cerebral symptoms or bad results ensuing, except that, in some instances, death was caused by the entrance of air into the veins.* The operation has even been repeatedly performed on the same animal, without any lasting injury. But it is scarcely necessary to have recourse to experimental researches on the lower animals for evidence on the matter, and in all these experiments there are sources of fallacy. Every medical practitioner must be familiar with the fact that the blood and tissues of man may be saturated with bile for months, without cerebral or any other symptoms of blood-poisoning resulting, so long as the glandular tissue of the liver is not destroyed, and the kidneys continue to perform their functions. Bearing such cases in mind, it is difficult to believe that bile is a deadly poison.

Reference has already been made to the views of an American physiologist, Dr. Austin Flint, jun., who has written a work to show that the cerebral symptoms, which occasionally occur in jaundice and in structural diseases of the liver, are due to the retention in the blood of cholesterin, or to what he has designated "*Cholestæremia*".† Dr. Flint regards cholesterin as an excrementitious product of nervous tissue, the elimination of which from the body he believes to be one of the functions of the liver. Having arrived in the bowel, the cholesterin, according to him, is converted into stercorin, and therefore it is not found in the fæces; but, when retained in the blood and tissues, he believes

it to be a virulent poison like urea. But if the non-excretion of all the elements of bile do not give rise to cerebral symptoms, it is difficult to understand how these symptoms can result from the retention of cholesterin alone. In cases, for instance, of permanent closure of the bile-duct, cholesterin is not discharged from the liver into the bowel; it does not accumulate in the biliary passages, nor, if it be present in the blood, does it necessarily give rise to cerebral symptoms.

From what has been stated, I think it must be clear that the cerebral symptoms, which occasionally supervene in certain morbid states of the liver, are independent of the non-excretion of bile, or of any of its component parts. The real cause of these cerebral symptoms will be referred to hereafter.

The symptoms usually associated with a deficient excretion of bile are an irregular, usually costive, state of the bowels, the stools being insufficiently coloured with bile, and of a pale yellow, drab, or whitish colour, loss of appetite, a white or yellowish furred tongue, a disagreeable, often bitter, taste in the mouth, especially in the morning, flatulence, a sallow or muddy tint of skin (indicating, unless there be concurrent hyperæmia of the liver, anæmia rather than jaundice), dingy conjunctivæ, languor and disinclination for exertion, frontal headache, dulness and heaviness, drowsiness after meals, great depression of spirits, and sometimes hypochondriasis, and frequent deposits of lithates in the urine on cooling. These symptoms are very apt to be induced, especially towards middle life, by sedentary or indolent habits, the habitual use of rich or indigestible food, neglect of the bowels, great or protracted anxiety of mind, or by a general want of vigour, consequent upon disease of the heart or some other organ; and the tendency to them is in many cases inherited. They are commonly, and perhaps correctly, ascribed to what is called "torpor of the liver"; but the non-excretion of bile may possibly be merely one of the symptoms, rather than the cause, of the morbid state, the real cause being the retention in the system, not of bile, but of those products of disintegration, which it is the purpose of the kidneys to eliminate. At the same time, it is very probable that engorgement of the liver with bile interferes with the normal processes of disintegration of albumen which take place in the gland.

REMARKS ON A CASE OF AMPUTATION.

By JAMES SEDGWICK, M.R.C.S., Boroughbridge.

I WAS hurriedly called on March 4th, 1874, to a farmer, aged 65, living four miles from me, who had just received such a severe injury to the wrist as to necessitate amputation. With the assistance of my friend Mr. Mitchell, when the patient was under chloroform, and after the application of Esmarch's plan of bandage, I amputated the forearm about three inches below the elbow-joint, and had the very great satisfaction of being able to make the skin-flaps, circularly divide the muscles, and saw through the bones, without the loss of a single drop of blood. But, after the arteries, six in number, were all secured, most troublesome venous and capillary oozing continued for between three and four hours, when it apparently ceased, and we were able to close the wound and apply steady and regular pressure.

The patient had influenza, with very frequent bronchial cough and expectoration. He was thin and very pale, and said that for many years no slight wound ever healed by first intention.

On March 6th, the oozing still continued to saturate the bandages; so much so, that in the evening I found it necessary, with the assistance of my partner, Mr. Leadman, to open and clear out the wound. We then applied styptic colloid freely, which at once stopped the bleeding without any further pressure.

Because a blush was visible along the course of the axillary vessels, tincture of iron was ordered every four hours, with frequent supplies of milk, beef-tea, eggs, etc., in small quantities.

As my object is to draw attention to the very troublesome oozing from the stump, after the adoption of Esmarch's plan for bloodless amputation, I will only add that now, March 21st, all risk of secondary hæmorrhage is past; for two days ago the last ligature separated, and the wound is closing rapidly, though for several early days it looked very unhealthy, and has made much slower progress than any other case that I have had in the country.

As Esmarch's plan is yet in its infancy, I thought it right to add my small quota of experience to the mass required to enable us to judge accurately of its perfection; and it certainly is perfect, as far as the comfort of the operator is concerned when he is using his knife and saw. I report my case especially because it was complicated, but trust that the extensive experience of many other surgeons may prove that the hæmorrhage was due to the condition of my patient, and not in any way to Esmarch's plan of bandage.

* *Diseases of Liver*, Sydenham Society's Translation, vol. i, p. 395.

† *Op. cit.*