chronic ones require chrysarobin. This would point to the presence of a particularly obstinate descendant of the bottle bacillus in these lesions, and to the predominance of the bottle bacillus itself and the special cocci in the more seborrhoeic lesions. In conditions due to the bottle bacillus, such as dandruff, nothing answers better than chrysarobin in weak strength, but where there is a combination of the bacilli and cocci, as in the scaly, greasy forms, chrysarobin shows only irritative properties and does harm, mercury and tar being much more efficient.

For use in the case described above a 0.2 per cent. colloidal solution of chrysarobin was made by Messrs. Crookes with a view to intravenous use; it is not stable in strong solutions at present. Quick progress in so chronic a case is not to be expected, but the results are far better than any hitherto obtained. A daily dose of 30 drops in water one hour after dinner is being given by the mouth, and the solution is well rubbed into the lesions night and morning with a pledget of cotton-wool. The scales are not previously removed; they seem to become partly absorbed where healing is occurring. This solution is the best I have yet tried for dandruff, and seems to promise a permanent cure after short use. It does not discolour the hair, but rather gives it a glint.

It might be concluded, then, on observed facts and the result of treatment—principally the differing actions of chrysarobin—that seborrhoea is the forerunner of psoriasis. Just as we find a gouty joint in the thin, anaemic descendant of a ruddy and gouty ancestor, so psoriasis may occur in the classically accepted form without concomitant sign of its seborrhoeic ancestor. What has been said about treatment was in the first place intended to clarify the etiology, but seems of some importance in itself. Thus the suggested intravenous injections in properly calculated doses might effect a permanent cure of even the most inveterate psoriasis.

It may be added that in the case of seborrhoea recorded acute gout developed at the age of 36, but the two diseases showed no other common factor except the general good health of the subject.

DIRECT OBSERVATION OF THE CIRCULATION IN THE LIVING LIVER.

BY

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Using one-quarter to half sized toads (pithed) I have been able, by laying a lobe of the liver on a sheet of glass on the microscope stage, to observe the circulation of the blood in the liver sinusoids and branches of the hepatic vein, under a low power. No cover-glass is required, and no solution of any sort is put in contact with the liver. The liver cells are clearly seen and the course of the blood flowing in the sinusoids (capillaries) can be accurately followed.

The liver circulation is now open to direct observation. Some years ago Leonard Hill greatly extended the microscopic study of the living tissue by his observation of the circulation in the glomerulus of the kidney, and the principle guiding me was the belief that organs apparently too thick for direct observation under the microscope in the adult might not prove so if the observation was made at an earlier stage of growth.

I have noted several times that a pulse may be apparent in the liver sinusoids and in branches of the hepatic veins. It appears to be a pulse of arrest of blood current, due to the contraction of the auricle. It will be easily understood that the blood flows from the liver through auricle into ventricle during diastole of the auricle and ventricle, and when the auricle contracts there is a retardation of the blood current in the liver sinusoids and hepatic veins.

Hence the description given by Sir James Mackenzie in his treatise on the heart is confirmed in great part. If the auricle is able to contract, the pulsation in the liver sinusoids and hepatic veins is auricular in time—that is, the *a* wave of the liver pulse is more or less synchronous with the *a* wave in the jugular pulse. If

the auricle fibrillates and no proper contraction occurs it acts as a passive receiving chamber, and the systole of the ventricle becomes the retarding agent on the circulation in the liver sinusoids and hepatic veins. Of course this retardation of blood current would be propagated to some extent to the portal vein and to the hepatic artery. Hence the pulse described in the portal vein must have, as one of its factors of origin, this retardation.

Mackenzie believed at one time that the auricle would have to be hypertrophied to produce an auricular form of liver pulse. He also believed an auricular form of liver pulse was a sign of tricuspid stenosis. Latterly he, as stated in his treatise, found cases of auricular liver pulse with no stenosis of the tricuspid valves. Drs. Turnbull and Wiel described such a case.

It will be seen from the foregoing observations that an auricular form of pulse must exist in the liver because the pulse is created in great part simply by retardation and arrest of the blood flow out of the liver. On systole of the auricle the liver swells as the blood flow is arrested because blood is continuously coming to it by the hepatic artery and the portal vein. The demonstration of that pulse is a matter of technique. It would be easier to demonstrate with swelling of the liver sinusoids in cardiac disease and enlargement of the organ. It would not require any hypertrophy of the auricle or tricuspid stenosis to be present. The auricle not only distends the liver—it dams the outflow. Of course, regurgitation of blood into the hepatic veins would add to some extent to the distension of the liver.

It is obvious that the shock of retardation of blood flow falls in great part on the liver cells. This shock is greatly increased when the auricle is practically non-contracting and the ventricle in its systole regurgitates blood through the auricle to the inferior vena cava. The blood pressure in the liver sinusoids must rise, and pulsation with increased capillary pressure is always damaging to cell life. It is apparent in bedsores, where the capillary pressure in the area of contact with the bed is raised by the compression of the venous outlets towards the value of the pressure in the entering arteries. Increased pressure in the capillaries with pulsating shock is disintegrative of cell life.

In the cardiac liver Sims Woodhead notes (*Practical Pathology*, p. 253):

"that the central or hepatic vein is considerably dilated and the lobules are distinguished more readily than in the normal liver. The capillaries leading to this central opening are also dilated. . . . The liver cells between the dilated capillaries are atrophied, etc. . . . Later the liver cells may have disappeared from the immediate neighbourhood of the central vein."

It will be noted from this description that the pathology follows physiological findings. The pulse of retardation (auricular form normally present) when exaggerated falls first on the hepatic veins and the liver sinusoids that feed them. Similarly with the ventricular form of pulse of retardation.

But the liver pulse differs from any other form of pulse in that it has three components:

A. The retardation of blood flow by the shuttering off, as it were, of the blood stream through contraction of the auricle.

B. Where the auricle is passive as a shuttering off agent, the ventricular systole acts similarly. In both A and B some blood may be driven back into the hepatic veins, but more in the case of B.

C. While A or B is in operation the liver swells through the entry of fresh blood by the portal vein and hepatic artery.

Consequently the fall in the liver pulse tracing is due to blood flowing unimpeded out of the liver.

This method of direct microscopical investigation of the liver will lead to further results. The pressure in the liver sinusoids will be measured and light thrown on the problem of the formation of liver lymph. The reactions, if any, of liver sinusoids (capillaries) to various stimuli, etc., are now open to investigation. The question of nerve stimulation and its effects on the liver will no doubt in time be submitted to direct visual examination; and perhaps in the future micro-chemical reactions in the living animal may reveal something of the working of the liver.

I am indebted to the Medical Research Council for the loan of apparatus.