over which there was dulness under percussion, coarse and clogged-wheel respiration, and increased vocal resonance, &c. Under the administration of cod-liver oil, chiretta and acid, citrate of iron and quinine, use of carbolie inhalations, chicken diet and extra milk, he improved so much as to be able to leave for the plains in November—the winter months in the hills being unsuitable for chest affections, and there being a chance of the symptoms remaining quiescent.

Sprains and accidents were rather more common in the hills, owing to the greater unevenness of the ground. In the individual who broke his fibula, the fracture was high up in the upper third—an unusual situation except in cases due to direct violence.

The other cases which came under treatment presented no features of unusual interest.

The foregoing sketch shows that young soldiers brought direct from England under the most favourable circumstances, as to the mode of travel and season, to the inner Kumaon hills, will not be exempt from attacks of the more commonly observed tropical diseases, but, as a rule, in a mild form and amenable to treatment—in other words, they become seasoned to climate and initiated into the ways and manners of Indian life with less risk to themselves than if left in the plains. As with all newly-arrived and young soldiers the percentage of admissions was high, but the mortality was exceedingly low, and the loss from invaliding nil. The two patients who died would probably have not contracted the disease which led to the fatal termination had they exercised ordinary prudence on the march and in quarters.

ART. V.—*Case of Pulsating Liver.*^a By WALTER G. SMITH, M.D., Dubl.; King's Professor of Materia Medica, School of Physic, T.C.D. ; Physician to Sir Patrick Dun's Hospital.

TRUE pulsation of the liver is sufficiently rare, and, I think, little known to justify my reporting the only case of it which I remember to have seen in hospital practice.

CASE.^b—J. M., aged thirty-five years, admitted into the Adelaide Hospital, August, 1878. He was a soldier, and had had ague, but with this exception was strong and healthy until 1875—i.e., three years prior

^a Read in the Medical Section of the Academy of Medicine in Ireland, Dec. 14, 1883. [For the discussion on this paper see page 87.]

^b From Notes taken by Dr. Wallace Beatty, resident pupil in the Adelaide Hospital, 1878.

to admission, when he caught cold, and was attacked with cough and breathlessness. Since that time he was never quite free from cough, and contracted several severe colds, the last one of which was followed by swelling of the legs. He was obliged to give up, and after a while was confined to bed. Without being a very hard drinker he was accustomed to take stimulants freely.

State upon admission.—Anasarca of the legs only; the swelling had never gone higher than the thighs. Loud bronchial râles over both lungs, with muco-crepitus, and attended with frothy expectoration. A faint systolic murmur heard over apex of the heart. The skin of the face and hands was dusky in colour, and there was visible pulsation in the jugular veins, which were of immense size—in short, there was extreme venous congestion of the upper half of the body.

The urine contained a large amount of albumen; no tube-casts were seen. He complained of a distressing feeling of load about the epigastric region, for the relief of which leeches were applied with benefit. The liver-dulness extended about two and a half inches below the ribs on the right side, and to within two inches of the umbilicus in the middle line, and its margin was readily felt. A distinct pulsation of the liver was visible in the epigastrium, extending over the right and left lobe. Dr. Beatty and I directed particular attention to this movement which arrested my notice, and we convinced ourselves that it was a true excentric throbbing of the liver, to be seen and felt, and not a mere shock, such as might be transmitted from the heart or inferior vena cava.

Subsequently the liver increased in size, and the lungs became engorged, while at times a triple cantering sound was heard over the apex of the heart, and the systolic murmur became more distinct. The area of cardiac dulness appeared to be enlarged, and the apex beat was visible in the sixth intercostal space. His condition fluctuated between better and worse, and he left hospital after a stay of nearly two months. I learned afterwards that he was soon re-admitted in a similar or worse plight, under one of my colleagues, and that he died rather suddenly. There is no record of a *post-mortem* examination having been made.

All hospital physicians have frequent opportunities of observing cases of heart disease, attended with enlargement of the liver, cervical venous pulse, and other evidences pointing to tricuspid regurgitation; yet, although on the watch for such an occurrence for some years past, I cannot call to mind a single other case in which I satisfied myself that there was regurgitant throbbing of the liver. Nor have some of my friends, of large experience, to whom I have addressed inquiries, been more fortunate, and, indeed, the phenomenon is sufficiently remarkable to be unlikely to be overlooked.

Dr. David Drummond, however, who has written an interesting paper on Pulsating Liver (*Dubl. Journ. Med. Sci.*, Oct. and Nov., 1881), believes that pulsating liver is of frequent occurrence, and is often overlooked by the physician. He gives details of nine cardiac cases in which this sign was present as a forcible and general movement of the liver.

Dr. Frederick Taylor has also written a paper deserving of perusal in *Guy's Hospital Reports*, 1875, based upon five cases of cardiac disease, in which hepatic pulsation was present. And in the *British Medical Journal*, March 18th, 1876, among the hospital reports, there is a brief reference to two cases under Dr. Ramskill's care in the London Hospital, in which diffused hepatic pulsation was observed. Both patients had cardiac disease and large nutmeg livers, as the *post-mortem* examination showed. In support of the proposition that the phenomenon under review is principally the result of a retrograde venous wave from the right ventricle (Friedreich, Taylor, Drummond), I may mention:—

1. The expansile sensation communicated to the fingers when placed upon the liver.

2. The duration of the pulsation. It continues after the ventricles have ceased to contract.

3. It has been observed that as the condition of the patient improved under treatment the hepatic pulsation became less and less (Drummond, Case II., *loc. cit.*). If the hepatic pulsation were due altogether to direct shock, the contrary would be the case, for steadier and more forcible action of the heart would materially increase the pulsation.

The question acquires practical importance, from its bearing upon the problem of diagnosis of tricuspid insufficiency, not always an easy matter for the physician to decide, and upon which authorities give out a somewhat uncertain sound.

Ought pulsation of the liver to be regarded as evidence of regurgitation through the tricuspid orifice? Taylor and Friedreich answer yes; and the latter goes further and states that, in point of time, hepatic pulsation precedes pulsation in the jugular veins.

Dr. Drummond maintains that pulsation of the liver is the most constant and most valuable sign of tricuspid regurgitation, less variable than systolic tricuspid murmur and jugular pulsation; but he does not contend that hepatic pulsation is pathognomonic of tricuspid regurgitation, and records himself a case *contra*. This matter appears to me to be deserving of more extended clinical

testing than it has yet received, and I shall be gratified if my paper have the effect of directing the attention of others to this point.

The questions still remain to be answered—How does regurgitation of blood into the hepatic veins in sufficient force to visibly agitate the liver sometimes occur? and why is it not more frequently observed in old-standing cases of mitral disease?

Neither Dr. Taylor nor Dr. Drummond make any reference to the peculiarities in the circulation of the liver in explanation of hepatic pulsation, nor is the topic alluded to in the standard textbooks of medicine.

Many years ago some ingenious and suggestive views were published upon the circulation of the blood through the liver, which were novel at the time, and which have since been corroborated by additional physiological and clinical evidence. Three authors, independently of each other, arrived at similar conclusions as to the conditions of the circulation through the liver—viz., M. Berard, Professor of Physiology in the Ecole de Médecine in Paris, in a paper published in the *Archives Générales de Médecine*, Juin, 1830; Dr. Carson, in his work, "Inquiry on the Circulation," Second Edition, 1833; and Mr. Alexander Shaw, Assistant Surgeon to the Middlesex Hospital, "On some Peculiarities in the Circulation of the Liver" (read before the British Association, June 24th, 1842, and published in the *London Medical Gazette*, July 15th, 1842).

A good abstract of Mr. Shaw's paper is given in a leader on "The Explanation of Pulsating Liver," *Med. Times and Gazette*, April 15th, 1876. Mr. Shaw set himself to show that the current of blood in the liver is subject to the influence of the respiratory movements of the thorax, and that the general effect of these actions is to assist its flow from the liver to the heart.

The vena portæ, a valveless and weak-walled vessel, has to perform the duties of an artery without possessing, like the arteries, an elasticity to aid it in propelling the blood, and without its fluid contents receiving much benefit in the way of direct impulse from the heart; consequently, when we consider the feebleness with which the blood must be sent through the gland by the portal vessels, and how its parenchyma must be exposed to the effects of congestion, it is natural to expect that some additional force should be brought into operation to accelerate the current. This auxiliary force is to be found in the actions of respiration, and the anatomical conditions which favour this influence are these:—

1. The trunks of the hepatic veins almost pierce the tendon of the diaphragm, to join the inferior vena cava.

2. The hepatic veins cannot collapse when subjected to atmospheric pressure—*i.e.*, to the aspiratory action of the mediastinum or suction power of the heart, because they are tunnelled out through the solid substance of the liver, and approximate in fact to rigid tubes. Hence the reason why, unlike the branches of the vena portæ, they are not surrounded by a bed of loose areolar tissue.

3. During respiration, by the contraction and descent of the diaphragm, the caval orifice of the diaphragm is stretched, the vena cava itself straightened, and the course and openings of the hepatic veins made more direct.

Conversely during expiration the venous trunks between the liver and the heart are shortened, their coats relaxed, and the opening in the diaphragm is diminished. Thus, at the very time when the thoracic cavity is lessening and the blood has a tendency to regurgitate into the hepatic veins, this tendency is counteracted by the provisions just described.

Accordingly, and because inspiration is a more powerful act than expiration, it follows that the circulation of the liver gains by the influence of the atmospheric pressure.

But if under any circumstances regurgitation of blood from the right heart takes place, it will be seen that this intimate relation of the hepatic veins to the liver-tissue, whereby they cease to be flaccid tubes and are converted into patulous sinuses, is a condition especially favourable for communicating to the liver as a whole any pulsation caused by reflux into the inferior cava and large hepatic veins. The degree to which the liver can yield is regulated by the elasticity of the enveloping capsule of the liver and of its peritoneal covering. Hence inflammatory thickening of the capsule is an obstacle to pulsation. Were it not for this elasticity of its envelopes the proper structure of the liver would, in cases of undue accumulation of blood within it, be injuriously compressed between the blood in the distended hepatic canals and the capsule of the liver.

Before hepatic pulsation can occur the impulse must be strong enough to overcome not only atmospheric pressure, but also the resistance of the parenchyma of the liver, the elasticity of its capsule, and all those conditions which have been already mentioned as tending to prevent regurgitation during ordinary expiration.

It is possible, I think, that there is commonly some degree of

retrograde pulsation in a nutmeg liver, but that in many cases it is very feeble and therefore inappreciable to ordinary methods of examination; and, independently of regurgitation, whenever, from any cause, there exists a condition of constant distension of the veins near to the right auricle, the fluid contained in these parts would be ready to receive and distribute an impulse by mere continuity with the contracting cavities.

CROTALINE AS A REMEDY FOR TETANUS.

THE marked antagonism displayed by the symptoms in cases of traumatic tetanus and snake-poisoning has led Dr. Annam, of New York State, to experiment with crotaline, which is the venom of the poisonous snake. In the one case we have some subtle influence at work in the nervous centres that causes extreme rigidity and spasm of nearly the entire voluntary muscular system—in the other an equally poisonous principle attacks the medulla oblongata and spinal cord, producing paralysis of both the voluntary and involuntary muscles. Dr. Annam narrates a case (in the *Medical News*, 29th September, 1883) of which the following is a précis:—Patient, male, twenty-five years of age; traumatic tetanus followed punctured wound of foot; first tetanic symptoms three days after receiving wound; twenty-four hours later, complete opisthotonos and other symptoms of the most aggravated form of tetanus. A few hours later patient was perceptibly worse, and the case presented no indications of a favourable termination. Chloral, bromide of potassium, and chloroform were freely used without any apparent beneficial effect. Woorara could not be obtained. Being in a locality where rattlesnakes are plenty, a snake-catcher was employed, and venom fresh from the fangs obtained. With this the point of a hypodermic syringe was moistened, and inserted beneath the cuticle in the dorsal region near the spine. Symptoms of snake-poisoning rapidly followed, with amelioration of tetanic spasms and rigidity, which entirely ceased at the end of ten hours, and patient slept for six hours. Thirty hours after insertion of poison, rigidity, with slight spasms, came on again. Snake-poison was again inserted, and the patient made a fairly rapid recovery; but extreme prostration followed the last introduction of venom, which necessitated active alcoholic stimulation, as in all cases of accidental snake-poisoning. There was no diffuse inflammation from the wounds, and Dr. Annam is of opinion that the second insertion of venom was too much, and might have been withheld. There were no results in the case which would deter him from adopting the same course again, and he is of opinion that crotaline may yet prove to be a valuable remedial agent in tetanus, and possibly in some other spasmodic diseases.

J. REX I.