BECKER, B., LANG, C. A. AND CHOW, B. F.: *Vitamin B₁₂ excretion and diabetic retinopathy*. J. Clin. Nutrition, 1, 6, Sept.-Oct., 1953, 417-423.

Following an intramuscular test dose of vitamin B₁₂, diabetic subjects with retinopathy were found to excrete significantly more of the vitamin than non-diabetics, while diabetics without retinopathy excrete considerably less than the non-diabetics. Administration of testosterone to diabetics with retinopathy decreased

the urinary excretion of a test dose of vitamin B₁₂. Cortisone increases the test dose excretion of Vitamin B₁₂ in rats. The urine of diabetics has been shown to contain a high concentration of oxysteroids related to but not identical with cortisone, and it is possible, though not proved, that some effect of the adrenal gland may account for the increased excretion of B₁₂ in diabetics with retinopathy. It seems unlikely that a subclinical renal defect is responsible.

ERRATUM

In the article "The Rapid Control of Uncomplicated Diarrhea with Resion" by Drs. Harry K. Gabroy and George J. V. Selsman, which appeared in the December 1953 issue, the third and fourth paragraphs under the RESULTS on page 396 should have read as follows:

"Of the 20 patients on kaolin plus pectin, 8 were

controlled between 24 and 36 hours. The rest were placed on Resion, 10 responding within the next 24 hours, 2 not responding.

Of the 20 patients on bismuth and paregoric, 10 responded within 24 to 36 hours; the other 10 were switched to Resion, 4 being controlled in 8 to 10 hours, and 2 within 24 hours. 4 did not respond."

EDITORIALS

ANGINA PECTORIS AND THE DIGESTIVE TRACT

It is well known that several abnormal conditions of the digestive tract may serve to precipitate an attack of angina in a heart capable of having angina. A common example, of course, is the diaphragmatic hernia, the earliest and chief symptom of which may be an attack of angina or a series of attacks, frequently in the presence of a normal cardiogram. There are many people alive and apparently well today who were diagnosed as having angina, but in whom the anginal attacks ceased, following the removal of a gallbladder full of stones. In such cases the distention of the gallbladder (or some of the bile ducts) is thought to have acted as a trigger mechanism, acting via the vagus, to set off the attacks of characteristic heart pains.

In all such cases, the question arises as to whether the patient actually had an attack of true angina. The answer depends partly on definitions. Is it thoroughly established, as yet, that every anginal attack is immediately caused by constriction of the coronary arteries? Possibly the majority of cardiologists would hesitate to be too dogmatic on this point. A breast pain which is relieved by nitroglycerine is not, for that reason alone, to be regarded necessarily as a pain caused by coronary artery constriction or spasm, because the general, body-wide vasodilatation brought about by the nitrites, lowers the blood pressure instantly and tends to relieve the cardiac load. Likewise, a spasm of the common bile duct may be quickly relieved by nitroglycerine, and so, presumably, would a so-called "reflex angina," depending on the biliary spasm, be relieved.

In a sense, this is splitting hairs, but yet it must be admitted that pure angina pectoris is not necessarily as simple a mechanism as supposed. Various facts sometimes make us wonder exactly *where* the pain of angina originates. The writer was once engaged in fluoroscoping a patient after a barium meal. The patient had had characteristic attacks of angina pectoris

as his chief complaint, but it appeared advisable to investigate the stomach radiographically. The stomach filled normally with the swallowed barium. Then the patient stated he was beginning to have an attack of angina. *Almost at once, the entire silhouette of the filled stomach disappeared from view.* Two minutes later the patient remarked that his pain was easing up. Almost at once the filled stomach again appeared on the fluoroscopic screen. Whether this spasm of the stomach had propelled the barium up into the esophagus, I never determined, but the experience left me with the question as to whether or not the patient's severe pain (which was completely characteristic of angina) had been due to pure gastrospasm. If so, then we have another possible initiator of angina.

We have seen patients with acute coronary occlusion complaining more of their stomachs than of their hearts. In several such patients, heroic doses of atropine brought remarkable and very prompt symptomatic relief, although, as it happened, all these patients eventually died of myocardial disease.

We are tempted to believe that, in some cases of coronary occlusion, the vagus plays a dominant etiological role. Banting, who produced coronary occlusion in dogs by chronic electrical stimulation of the distal ends of the cut vagi, once remarked that if he himself ever developed coronary occlusion, he would like to have plenty of atropine, and nothing else. He had found that dogs which were given atropine daily, failed to develop occlusion under the experimental conditions.

Unquestionably, in occlusions and in anginas as well, the vagus is involved either primarily or secondarily and such a thesis helps us to imagine how the distention of a common duct might cause a *referred pain* to the precordium or sternum, and how intrinsic myocardial disease could so easily cause pain in the stomach. For these reasons, when we are in doubt (and we usually are) we should not forget that in addition to nitroglycerine we possess a remarkable drug