was seen. The granules of the latter cell stained a very much lighter purple than they commonly do when fresh from the blood-stream. Here also the true character of the large cells was determined. There were one or two to a field ($\times 900$), 30 by 50 microns in size. The cells were mononucleated and multinucleated, the former predominating with rich basophilic protoplasm in which were held neutrophils, eosinophils, lymphocytes and occasionally bacteria in various stages of digestion. In some, the captive elements were practically intact. In others, the entire protoplasm had been digested and only the nuclear fragments remained. Surrounding such fragments a clear area was frequently seen in the protoplasm of the macrophagocyte, representing the zone of digestion. The illustration, drawn from cells observed, shows the beginning (A) and the advanced stage (B) of digestion. In the latter stage the protoplasm of the macrophagocyte took on a dark greenish tinge, often mottled, undoubtedly from the absorption of iron pigment.

In the peripheral circulation none of these cells was observed. The urine examinations showed nothing unusual.

The parasitic nature of the patient's trouble has not been determined. He was directed to Dr. George Dock, then of New Orleans, who confirmed our findings and considered the possibility of distomiasis. He was unable, however, to find any ova or parasites.

Thinking that these macrophagocytes may previously have been overlooked by observers, I have studied about thirty

A, macrophagocyte showing beginning stage of digestion; B, showing an advanced stage.

cases, showing more or less eosinophils in the blood and sputum, with negative results.

The patient wrote us in February, 1912, that his general health is fair and his weight normal, but that the bloody expectoration has continued and is more profuse than when under our observation and that the wheezing sounds in the chest are very annoying. He has been taking salvarsan and potassium iodid. Parasites have not been found in the sputum.

FATAL HEMORRHAGE FROM A SMALL BRANCH OF THE VENA SAPHENA PARVA

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Profuse hemorrhages from injuries of peripheral veins are not uncommon. Fatal hemorrhages from such veins are also on record, but are comparatively rare. Most of the cases of that kind have been hemorrhages from ruptured varicose veins, large distended branches of the long saphenous veins of the lower extremities.

As coroner, I was recently called on to attend a case of fatal bleeding from a very small branch of the small saphenous vein, or vena saphena parva, in a most unusual position, which I have never heard of before, nor have I been able to find a similar instance in the medical literature.

History.—Eliza C., colored, aged 50, was always a healthy woman. There was nothing remarkable in the family history.

There was no history of tuberculosis, malignant disease, nervous or mental degeneration or hemophilia.

Her past history was also not remarkable. She never had had typhoid, pneumonia or rheumatism. She gave birth to five healthy children and had had no miscarriages. There was no history of lues. She was not a hard drinker but indulged in alcohol.

On September 5, the patient retired at night as usual in good health. About 12:30 a. m. she had to get up from bed, and in doing so she struck the calf of her right leg against the edge of a trunk standing in her bedroom. The blow broke the skin over the calf of the right leg and ruptured a vein; she began to bleed very freely. She called to her son, who found her in a pool of blood with blood gushing from the wound as from "a spigot," he said. Not knowing what to do he sent one of the other children after a physician; but when the physician arrived about 1:15 a. m., or forty-five minutes after the injury, the patient had bled to death.

Examination of the body revealed a small hole at about the middle of the right calf, not more than an eighth of an inch in diameter, from which blood was still oozing. Had the body been found in the street, the first thought which the nature of the wound would have suggested would have been that the woman had been shot; the aperture was no bigger than that produced by a 22 caliber bullet. In this case the circumstances of the accident, and the victim's own account of it before she died excluded any such possibility. To clear up this most unusual case I ordered an autopsy.

Autopsy.—Performed by Dr. N. G. Keirle at the City Morgue. The wound on the right leg was found to be nearly round in shape and from about one-eighth to one-fourth inch in diameter. A probe passed into it slid for the distance of half an inch or so below and parallel to the surface. After making a semicircular incision and flapping the skin back, we found that the wound entered a small vein lying close to the surface, which continued for the distance of about half an inch and then dipped into the subcutaneous fat, which was rather abundant, and joined a larger vein. There was no sign of any stab or gunshot wound. The only injury found was the ruptured vein not more than an eighth of an inch in diameter. A most interesting feature about the vein was the marked rigidity of its walls. The opening in the lumen at the place of rupture stood gaping, owing to the calcification and sclerotic changes in the walls, and this probably accounted for the profuse and fatal hemorrhage.

The only other interesting features about the post-mortem examination were the marked general arteriosclerosis and atheromatous changes in the aorta, and the extreme pallor of the intestines and liver. The deceased was simply exsanguinated to death. The kidneys showed an adherent capsule and moderate interstitial nephritis. The heart and lungs were normal, and the brain showed nothing remarkable. There were no visible varicose veins on either leg or thigh.

I rendered a verdict of accidental death due to a most unusual fatal hemorrhage from a branch of the small saphenous vein, or vena saphena parva, caused by rupturing that vein through knocking the leg against the edge of a trunk.

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Therapeutics

DISTURBANCES OF THE HEART

MYOCARDIAL DISTURBANCES

(Continued from page 1719)

III. FATTY DEGENERATION

Fatty degeneration of the heart muscle may be caused by acute poisoning (as phosphorus, arsenic, etc.), by serious infections, or it may follow fibrosis of the heart or coronary artery disease. The symptoms are those of serious circulatory weakness, which does not seem to improve under any ordinary management. It is difficult, if the heart is enlarged, to determine whether there is more or less serious acute dilatation or whether the heart