SUMMARY

Abdominal examination was carried out in 75 women of varying parities one to eight days after normal delivery.

The liver was palpated in 23 of these women (31%) and the spleen in nine (12%).

The significance of these findings is discussed.

The author wishes to thank Dr. L. J. Harris and the obstetrical staff of the New Mount Sinai Hospital for permission to examine the postpartum women, and Professor K. J. R. Wightman for valuable suggestions.

REFERENCE

1. BARCROFT, J.: *Amer. J. Med. Sci.*, **179**: 1, 1930.

CASE REPORTS

Unilateral Arm Ischemia Due to Ergotamine Tartrate

C. H. TATOR, M.D., M.A., Ph.D.* and
R. O. HEIMBECKER, M.A., M.D., M.S., F.R.C.S.[C],† *Toronto*

ERGOTISM, or poisoning due to ergot, is no longer seen in the epidemic form, as when whole populations were exposed to foodstuffs infected with the fungus *Claviceps purpurea*. However, ergotism should not be forgotten as a clinical entity, because it is still seen in patients given ergot preparations for migraine or in the course of obstetrical or gynecological management. In the present report a man with ergot poisoning is described. This case serves as a reminder of a clinical entity that can be treated effectively if recognized. The case was unusual in that the only manifestation of ergotism was acute ischemia of one arm.

Mr. W.K., a 59-year-old construction company owner, was admitted to the Toronto General Hospital on January 17, 1966. For approximately 25 years he had had severe headaches which were usually right-sided, and were preceded by an aura of scotomata or flashing lights. Over the years the headaches increased in frequency and severity, and for the past year they were present almost continually. During the past few years his daily drug intake included meperidine 150-300 mg. subcutaneously, two caffein-ergotamine tartrate tablets (Cafergot, each containing ergotamine tartrate 1 mg.), and two caffein-ergotamine tartrate suppositories (Cafergot, each containing ergotamine tartrate 2 mg.). In addition, during the past few months because of particularly severe headaches, he took two tablets of methysergide daily and often increased his intake of caffein-ergotamine tartrate to three tablets and three suppositories daily.

From the Division of Cardiovascular Surgery, Toronto General Hospital, Toronto, Ontario.
*Assistant Resident, Cardiovascular Surgery, Toronto General Hospital.
†Cardiovascular Surgeon, Toronto General Hospital; Assistant Professor, Department of Surgery, University of Toronto, and Research Associate, Ontario Heart Foundation.
Supported by the Ontario Heart Foundation and by National Health Grant No. 605-7-253.
Reprint requests to: Dr. Ray Heimbecker, Cardiac Unit, Toronto General Hospital, 101 College Street, Toronto 2, Ontario.

On January 15, 1966, he noticed that his right hand was gradually becoming blue, numb and painful. The next morning the hand was even more cyanosed and painful, and was weak and cool. These symptoms slowly worsened over the next 24 hours. He consulted his doctor, who administered single doses of heparin and phenylindanedione and immediately referred him to this hospital.

He was a well-nourished, middle-aged man in moderate distress because of pain in the right hand and headache. The right hand and lower forearm were deeply cyanosed and very cold, and appeared to be almost gangrenous. There was marked muscle weakness for all movements of the right fingers and wrist, and hypoesthesia for all sensory modalities in the hand and lower forearm. At the junction of the middle and lower thirds of the forearm, there was a distinct line of separation between the cold, blue, ischemic limb below and the normal-appearing arm above. The right axillary pulse was normal, but there was an abrupt cessation of pulsation at a point judged to be the beginning of the right brachial artery. The right brachial pulse in the antecubital fossa and the radial and ulnar pulses at the right wrist were absent. The blood pressure was unobtainable over the right brachial artery in the antecubital fossa. The left arm and both legs were entirely normal with strong pulses and normal colour, temperature, sensation and muscle power. The blood pressure in the left arm was 140/80 mm. Hg. The apical rate was 84 per minute and regular. The remainder of the physical examination was normal.

At the time of admission an emergency right axillary arteriogram showed diffuse narrowing of the arteries below the axillary artery (Fig. 1). The lumen of the brachial artery was reduced to approximately 1 mm. throughout most of its length. The axillary artery appeared normal, without any evidence of atherosclerosis. Because the arteriogram showed arterial spasm, tolazoline (Priscoline) 25 mg. was injected through the arteriographic needle. Five minutes after injection the hand was less cyanosed and warmer, and at 10 minutes the brachial and radial pulses were palpable, although they were weak. A second arteriogram,

Canad. Med. Ass. J.
Dec. 17, 1966, vol. 95

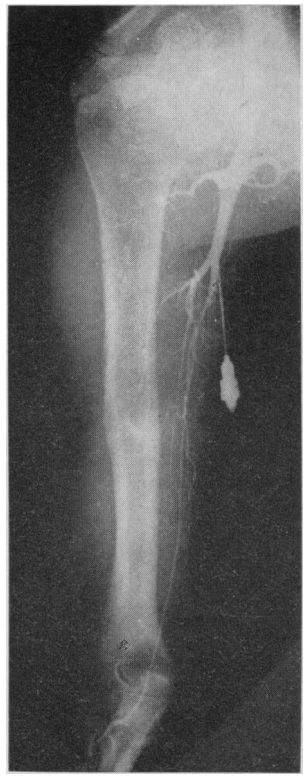

Fig. 1.—Right axillary arteriogram showing severe diffuse narrowing of all arteries below the axillary artery. The axillary artery itself appears normal up to the point where it becomes the brachial artery; here the vessel narrows markedly. Branches of the axillary are also in spasm.

performed about 30 minutes after the first arteriogram and 10 minutes after the injection of tolazoline, showed some dilatation of the brachial artery, especially at the elbow (Fig. 2).

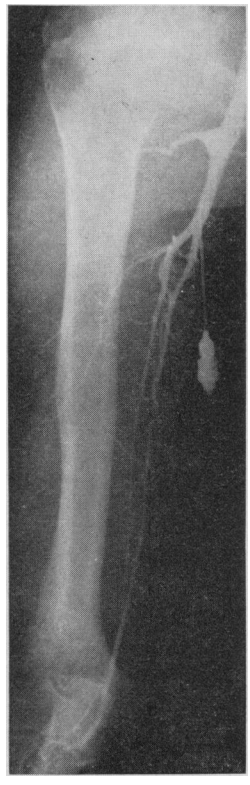

Fig. 2.—Right axillary arteriogram performed 30 minutes after the arteriogram in Fig. 1, and 10 minutes after the intra-arterial injection of tolazoline 25 mg. There is some dilatation of the brachial artery, especially at the elbow.

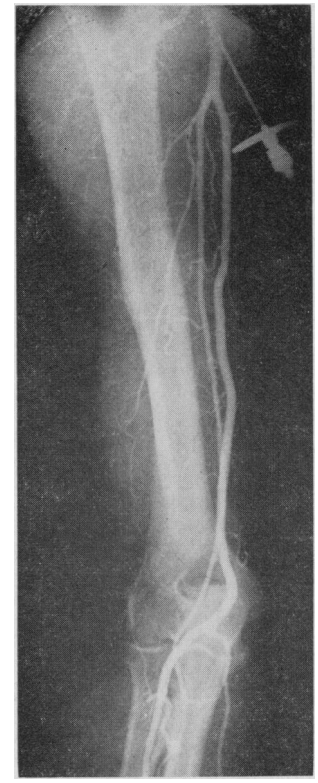

Fig. 3.—Right axillary arteriogram performed eight days after those in Figs. 1 and 2. The arteries now appear entirely normal. There is no evidence of atherosclerotic disease.

Shortly after the arteriographic studies on January 17, a sympathetic block was performed by injecting

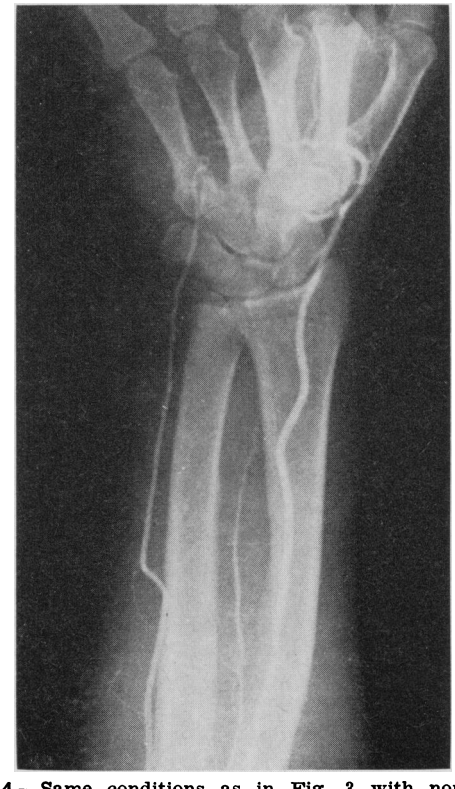

Fig. 4.—Same conditions as in Fig. 3 with normal-appearing arteries.

10 ml. of 1% mepivacaine through a plastic cannula inserted into the region of the right stellate ganglion. Two further injections of 10 ml. were given through the cannula at intervals of four hours during the night, and then it was removed during the morning of January 18 because of accidental contamination. Low-molecular-weight dextran in a 10% solution (Rheomacrodex) was also given after the arteriographic studies at a rate of 500 ml. intravenously every eight hours and was continued for 48 hours.

During the night of January 17 there was a gradual increase in the warmth of the arm and in the strength of the pulses, and by morning the pulses were almost normal. Twenty-four hours after admission to hospital, there was complete relief of pain in the arm, and it had normal colour and sensation. Strength returned much more slowly and was still not fully restored at the time of discharge on January 26.

On January 25, the right axillary arteriogram was repeated and the arteries appeared entirely normal (Figs. 3 and 4). When the patient was examined three weeks later the arm was normal.

DISCUSSION

The diagnosis of ergotism in this case was not made until the initial arteriograms were seen. Before arteriography, the working diagnosis was embolic or thrombotic occlusion of the brachial artery. However, when the arteriograms demonstrated the diffuse arterial narrowing characteristic of ergotism, we correlated these findings with the patient's history of excessive ergotamine tartrate intake. With cessation of ergot intake and active treatment, the pre-gangrenous limb became almost normal within a few hours.

As seen in this case, ergot alkaloids may cause intense arterial spasm. Their constrictor effect on the smooth muscle of blood vessels has been known for many years.[1-3] The amount of ergot that a person can consume before developing arterial spasm and ischemia varies greatly. In some cases, ergotism occurred after doses as small as 1 mg. of ergotamine tartrate daily for two weeks;[4] in others, like the present case, it occurred only after much larger doses over a period of years.[5] The reason for this variation in susceptibility is not definitely known, although pre-existing vascular disease or sepsis has been implicated.[6]

In addition to limb ischemia, ergot poisoning may produce headache, nausea and vomiting, diarrhea, convulsions and hemiplegia.[6, 7] In cases with limb ischemia, usually all four limbs are involved,[8, 9] although numerous examples have been recorded in which only the legs were involved.[10, 11] Less frequently, only the arms have been affected, and rarely this has been unilateral.[11]

The treatment of the ischemic effects of ergotism is directed towards relief of arterial spasm and prevention of vascular stasis and thrombosis. Treatment should be instituted as rapidly as possible, before necrosis and thrombosis produce irreversible changes. The two principal methods of relieving spasm are the administration of vasodilator drugs and sympathetic interruption. In the present case, intra-arterial tolazoline restored the pulses within a few minutes in a limb that was severely ischemic. Arteriographic proof was obtained that tolazoline was effective in relieving spasm. However, in other reported cases tolazoline was ineffective.[7, 9] Vasodilators given intravenously have not been very helpful.[7] Sympathetic block, sympathectomy, periarterial stripping of vessels and continuous spinal anesthesia have been beneficial in some cases.[5, 7] Our patient also had a cervical sympathetic block and was given intravenous low-molecular-weight dextran. However, it is not clear whether or not these measures potentiated the improvement already initiated by intra-arterial tolazoline and withdrawal of ergot.

In managing limb ischemia due to ergot, several measures may relieve spasm and prevent stasis and thrombosis. A reasonable treatment regimen would be to give heparin and low-molecular-weight dextran as soon as possible and to follow this with intra-arterial tolazoline and sympathetic interruption.

SUMMARY

A case of acute, severe limb ischemia due to ergot poisoning has been presented. The diagnosis was made from the arteriographic findings and a history of intake of ergotamine tartrate. Treatment, including heparin, low-molecular-weight dextran, intra-arterial tolazoline and sympathetic block, was successful. Limb ischemia may occur even after small doses of ergot and should be watched for in all patients using the ergot alkaloids. Arteriography is of great value in confirming the diagnosis because the arteriographic picture of diffuse arterial narrowing is highly characteristic. Treatment, which should be started as soon as possible, is directed towards the relief of arterial spasm and the prevention of stasis and thrombosis.

REFERENCES

1. DALE, H. H.: *J. Physiol. (London)*, **34**: 163, 1906.
2. BLUNTSCHLI, H. J. AND GOETZ, R. H.: *Amer. Heart J.*, **35**: 873, 1948.
3. PICHLER, E. *et al.*: *A.M.A. Arch. Neurol. Psychiat.*, **76**: 571, 1956.
4. CLEVELAND, F. E. AND KING, R. L.: *Bull. Mason Clin.*, **2**: 19, 1948.
5. JOHNSSON, K. A.: *Acta Radiol. (Stockh.)*, **57**: 280, 1962.
6. VON STORCH, T. J. C.: *J. A. M. A.*, **111**: 293, 1938.
7. YOUNG, J. R. AND HUMPHRIES, A. W.: *J. A. M. A.*, **175**: 1141, 1961.
8. CRANLEY, J. J. *et al.*: *New Eng. J. Med.*, **269**: 727, 1963.
9. CAMERON, E. A. AND FRENCH, E. B.: *Brit. Med. J.*, **2**: 28, 1960.
10. YATER, W. M. AND CAHILL, J. A.: *J. A. M. A.*, **106**: 1625, 1936.
11. SAENGER, H.: *Zbl. Gynaek.*, **53**: 586, 1929.