The Effect of Intra-arterial Injections of Hydergine and Dihydroergocornine on the Peripheral Circulation in Man

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If given by the intra-arterial route, the immediate effect of the hydrogenated alkaloids of ergot is unpredictable. Vasodilation is obtained in some patients only. However, it was found that in those cases who do not react, the vessels respond more easily to the release of sympathetic tone, proving that, nevertheless, the drug has had an (potential) effect, probably on the neurovascular apparatus. This effect could be demonstrated for many hours and is, therefore, of considerable therapeutic interest. Even with the higher local concentrations possible by the intra-arterial route, the hydrogenated alkaloids of ergot still did not exhibit any adrenolytic action on the peripheral blood vessels in man.

EXTRACT of ergot, widely used for its oxytocic action and notorious for its vasoconstrictor effect, had long been suspected of possessing, in addition, an inhibitory action on the sympathetic nervous system. This sympatholytic action of ergot has proved of great interest to physiologists and clinicians ever since Rothlin1 demonstrated its presence in ergotamine. However, such conditions which would have benefited from the sympatholytic action, namely, hypertension and peripheral vascular diseases, remained inaccessible, due to the direct vasoconstrictor action still present in ergotamine. The sympatholytic principle became accessible to the clinician only when Stoll and Hoffman2 in 1943 succeeded in producing derivatives, which in animal experiments proved void of all vasoconstrictor effects.4,5

Our investigations on the circulatory effect of one of these alkaloids, namely, dihydroergocornine3,6 and Hydergine* in man7 demonstrated that, if given by the parenteral route, they produce bradycardia, a fall in blood pressure and dilatation of the peripheral blood vessels, which suggested some usefulness in the treatment of hypertension. These effects were shown to be partly central in origin and partly dependent on the integrity of the sympathetic nervous system.2,6 These findings have subsequently been fully confirmed by numerous investigators.8-13

Besides these effects, Goetz and Katz14 showed that dihydroergocornine may suppress or reverse the blood pressor response to adrenalin, although it was unable to do the same to the vasoconstrictor action of adrenalin on the digital vessels, at least in doses which were sufficient to produce a reversal on the blood pressure.

Whereas hypotension and bradycardia are very constant and can be obtained with the greatest regularity, the vasodilator effect on the peripheral vessels is a capricious effect not invariably produced by intravenous Hydergine. As the blood vessels of the human extremity do not contain vasodilator fibers, ablation of the vasomotor tone alone does not always seem sufficient to produce vasodilation. As a matter of fact, the failure of sympatholytic drugs to produce vasodilatation has been quoted in support of the assumption that the blood vessels of the human skin are not supplied by active vasodilators.22

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* Hydergine is a mixture of equal quantities of the three hydrogenated ergot alkaloids: dihydroergocornine, dihydroergocristine and dihydroergokryptine and has, in principle, the same action as dihydroergocornine.

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reference to the sympathetic nervous system, the question arose as to whether in man, too, a more constant and reliable peripheral dilator action could be obtained by the intra-arterial route of administration. This question has become of particular interest since, within recent years, intra-arterial (as opposed to intravenous) therapy in cases of peripheral vascular diseases has found many protagonists.23-25

Bircher and Cerletti26 were able to demonstrate that in the anesthetized cat dihydroergocornine readily produced reversal of the adrenalin effect on the peripheral blood vessels, whereas, as already mentioned, our own investigations14 failed to elicit this effect in man. Our failure, which referred to intravenous injections, challenged an inquiry into the possibility of producing adrenalin reversal by administering the ergot alkaloids by the intra-arterial route, when the drug could reach the peripheral neurovascular apparatus in higher concentrations.

METHOD AND MATERIAL

Continuous records of the peripheral circulation were obtained by means of the optical digital plethysmograph, as described in detail elsewhere.29 For the present investigation, a portable model, fitted with two pipettes for the simultaneous recording of the blood flow in two extremities, was used.31 This method is sensitive enough not only to allow the correct registration of the height of the pulse volume, but also the calculation of the arterial inflow at any one moment by means of the so-called venous congestion test. Simultaneously with the blood flow, the respiration and the skin temperature of one or more digits could be recorded with a built-in mirror galvanometer and thermocouples. The heart rate was, of course, available from the plethysmogram. The blood pressure was registered by clinical methods.

All examinations were carried out in a noiseless room with the patient reclining quietly on a couch. The tests were usually carried out after 30 to 60 minutes rest. By that time the volume record showed a minimum of spontaneous fluctuations in the peripheral blood flow.

Reflex dilatation was obtained by immersion of one extremity into a specially constructed tank containing water of 44 to 45°C. An immersion heater connected in series with a thermostat and a stirring propeller were fitted in the tank to keep the water temperature constant at that level.30, 32 The patient was covered with blankets in order to prevent the dissipation of heat.
In some cases, the drugs were administered slowly by means of an intravenous drip. The rate of the drip was continuously recorded simultaneously with the plethysmogram by means of the photo-electric drop recorder. From the number of drops recorded, the exact amount of drug given could be calculated for any one moment.

For the present investigations 19 patients were subjected to the following procedures:

1. Intravenous injection of dihydroergocornine or Hydergine.
2. Intra-arterial injection of dihydroergocornine or Hydergine.
3. Intra-arterial injection of dihydroergocornine or Hydergine, followed by intra-arterial injection of adrenalin.
4. Intravenous injection of dihydroergocornine or Hydergine, followed by adrenalin.
5. Intra-arterial injection of a mixture containing dihydroergocornine and adrenalin.

It will be readily appreciated that by simultaneous recording of the vascular reactions in both limbs, following the intra-arterial injection into one, the effect of the drug on the other limb may be similar to that of an intravenous injection, provided the drug is not destroyed or escapes from the vascular tree during its course through the capillary network.

Ten of the patients were normal from the cardiovascular aspect, varying between the ages of 18 and 49. Two patients suffered from essential hypertension, being 55 and 64 years of age, respectively. One patient, aged 42, had had a stellatectomy. Four patients suffered from acrocyanosis and two from Raynaud's phenomenon. They were selected for these investigations because they regularly exhibited a high vasomotor tone.

In the beginning, 0.3 mg. dihydroergocornine was given, usually into the right femoral artery. Later we used Hydergine, 0.5 to 1.5 cc., throughout.

**RESULTS**

1. **The Immediate Effect of Intra-arterial Injection of Dihydroergocornine or Hydergine on the Blood Flow:** The local effect of an intra-arterial injection of Hydergine or dihydroergocornine in doses large enough to produce a general systemic effect, such as bradycardia and hypotension, is unreliable and unpredictable, both in the normal subject and in the patient exhibiting a high vasomotor tone.

The general tendency is to produce either an immediate increase in blood flow up to or above the minimum vasodilation level, or no reaction in the blood vessels at all. With the doses employed, a moderate improvement in the

![Fig. 2. Injection of 0.5 mg. Hydergine into the right femoral artery in two cases, demonstrating the two extremes in the response obtained. (A) Complete lack of response in a case with a high vasomotor tone. (B) Immediate dilatation of the vessels, as judged by the increase in pulse volume and rate of arterial inflow, in a case suffering from essential hypertension.](http://circ.ahajournals.org/)

arterial blood flow of the limb was only seen twice.

Two extreme reactions are shown in figure 2A and B. Figure 2A was obtained in a young female patient suffering from typical acrocyanosis. As can be seen at a glance, the intra-arterial injection of 0.5 mg. Hydergine into the right femoral artery did not induce any response in the arterial tree of the limb whatsoever. The vessels, being under a high vasomotor tone at the time of the injection, remained constricted, and within 20 minutes there was no change in the height of the pulse volume, digital volume or the rate of arterial inflow. The lack of response is well appreciated from the cuttings of the plethysmogram (fig. 3A-A').

Figure 2B was obtained from a patient, 55 years of age, who had suffered from essential hypertension for many years. In his case the intra-arterial injection of 0.5 mg. Hydergine into the right femoral artery produced an immediate increase in the arterial circulation, as judged by the rise in pulse volume, digital volume and, particularly, the rate of arterial inflow. The pulse volume, which recorded 0.008 cc. in both toes before the injection, increased in the right within a few seconds and became
produce any direct peripheral response, eventually brings about the same systemic effect on the circulation as an intravenous injection of the same magnitude. Therefore, provided it is large enough, it will produce a fall in the systemic blood pressure and bradycardia.

Although the intra-arterial injection of Hydergine may produce a powerful vasodilator reaction in the injected limb, it is only occasionally that this is followed by a similar reaction in the remaining limbs.

Similarly, such side-effects as may occur with intravenous injections may also be noted after the same amount given by the arterial route.

(2) The Effect of Reflex Vasodilatation on the Peripheral Blood Flow following Intra-arterial Injection of Hydergine: Rothlin drew attention to the fact that the principle actions of the ergot alkaloids may be actually visible, such as the direct stimulation of smooth muscular organs, or they may be latent or potential. Into the latter category, he grouped all such effects which are only elicited when the respective organ is activated either by sympathetic stimulation or by circulating adrenalin.

Thinking along these lines, the question arose as to whether the intra-arterial injection of dihydroergocornine, although it may not produce any immediate and “visible” response on the peripheral vessels, did, in fact, produce changes, either on the muscle itself or the neurovascular apparatus, which were “latent or potential” in the sense used by Rothlin and, therefore, could only be demonstrated under certain physiologic conditions involving reflex mechanisms.

In order to answer this question, eight subjects, who did not show any response to the intra-arterial injection of dihydroergocornine or Hydergine, were subsequently submitted to reflex body heating as routinely used for diagnostic purposes in cases of peripheral vascular disease. One arm was immersed in water of 45 C. to a point 6 inches above the elbow for 30 minutes. The dissipation of heat was prevented by means of blankets. In the normal individual this causes reflex dilatation in the remaining extremities. Normally, the response is elicited after a delay of eight to 10 minutes, which is explained by the fact that dilatation

Fig. 3. The effect of an injection of 0.5 mg. Hydergine into the right femoral artery. Cuttings of the plethysmograms obtained in the same two cases as demonstrated in figure 2. (A-A’), case 1: Complete absence of response in pulse volume (C-B’), case 2: Dilatation obtained in the right leg in the absence of any reaction in the left leg.

established at about 0.015 cc. (fig. 3B-B’). The rate of arterial inflow increased from 12 to 42 cc. per minute for 100 cc. of tissue during the same period. The blood flow through the left leg, which was simultaneously recorded, showed no change in this case, although in some patients a temporary decrease could be registered, as judged by the height of the pulse volume and the rate of arterial inflow, provided the intra-arterial injection produced dilatation of the injected limb.

As already indicated above, the intra-arterial injection of Hydergine, although it may not
is produced by the increase in the blood temperature resulting from the return of heated blood from the immersed extremity. This causes ablation of the central sympathetic tone in the hypothalamus. In cases exhibiting a high vasomotor tone, and therefore having an initially diminished blood flow through the limb, such as cases of acrocyanosis and Raynaud's phenomenon, it regularly takes considerably longer to initiate dilatation.

As vasodilatation induced by body heating is produced by central ablation of vasoconstrictor tone, it occurs simultaneously in either both lower or both upper extremities, although it does not necessarily occur simultaneously in both the upper and the lower extremities, as the vasomotor tone is generally known to be higher in the latter.

Figure 4 demonstrates the response to reflex body heating following an intra-arterial injection of Hydergine in a patient in whom it did not produce any immediate visible peripheral hemodynamic effect. It refers to a young woman suffering from acrocyanosis, in whom the vessels are normally under a high vasomotor tone and peripheral blood flow is diminished. The injection of 0.5 mg. Hydergine into the right femoral artery did not have any effect whatsoever on the peripheral blood flow in that leg. Twenty minutes after the intra-arterial injection, the digital blood flow was still negligible in both limbs. The pulse volume was hardly registrable in either limb, and the rate of arterial inflow registered only 2 to 3 cc. for 100 cc. of tissue per minute. At this stage, body heating was started by immersing the right arm into water at 45 C. Considering the response in the left leg first, which may serve as a control, we see that, as is typical of cases with vasospastic disorders, 20 minutes pass before the blood vessels begin to dilate. However, once initiated, vasodilatation proceeds and the pulse volume, which before was hardly registrable, rapidly increases to well above 0.020 cc. By virtue of the vasodilatation an increase of 1.20 cc. in the digital volume is recorded, and the rate of arterial inflow increases from 3 cc. to just about 100 cc. per minute for 100 cc. of tissue. After 35 minutes of body heating, the vessels are fully dilated.

It can be seen in figure 4 that the response of the blood flow in the right leg differs greatly from that in the normal left leg, both in regard to onset and to extent. Within five minutes, the vasomotor tone begins to relax and, after 10 minutes, dilatation is well on its way. Within 15 minutes, the pulse volume has risen to 0.020 cc. and after 20 minutes, body heating reaches values round about 0.030 cc., that is, at a time when in the left leg the vessels are still fully constricted. The digital volume is the first to rise, and after 20 minutes has increased by fully 1.8 cc. At the same time, the rate of arterial inflow reaches 180 cc. per minute for 100 cc. of tissue.

As is very well illustrated in figure 4, re-
The changes in height of the pulse volume are readily appreciated by consulting figure 5, which shows cuttings of the original plethysmogram. The upper tracing refers to the right, that is, the “pretreated” leg, and the lower to the “normal” left limb. The difference in the reaction is particularly well demonstrated 15

flex dilatation not only occurs more readily but proceeds to a much higher level following the intra-arterial injection of Hydergine. By the time the vessels of the left limb start to dilate, those in the right have already dilated to a level far exceeding that eventually reached in the left leg.

Fig. 5. Cuttings of the plethysmogram obtained in the same case as depicted in the chart of figure 4. (A) Pulse volume before injection; (B) No effect from the intra-arterial injection; (C) After 15 minutes body heating: marked dilatation obtained in the right leg only; (D) twenty-five minutes after body heating: full dilatation of the vessels in the right leg but still hardly any response in the left and (E) thirty-seven minutes after body heating: vessels of the left leg dilated, indicating that the lack of response was not due to an organic involvement of the peripheral vessels.
FIG. 7. Reflex dilatation of the vessels of the lower extremities in a patient who had an intra-arterial injection of Hydergine into the right femoral artery two days previously. There is still a difference in the response between the two legs.

and 25 minutes after the onset of bodily heating, when the difference in vasomotor tone is at its maximum, as judged by the difference in the height of the pulse volume. It is also well appreciated on recording the rate of arterial inflow by means of the venous congestion test (fig. 6).

How long following the injection can this difference in the response to reflex body heating following an intra-arterial injection of Hydergine be demonstrated? To answer this question, four patients were subjected to reflex body heating at various time intervals following the injection of Hydergine. In every one, the effect was still demonstrable after eight hours and in one fully 48 hours after the intra-arterial injection (fig. 7).

(3) The Effect of an Intra-arterial Injection of Adrenalin following the Intra-arterial Injection of Hydergine: As Goetz and Katz demonstrated, the vasoconstrictor effect on the digital circulation of an intravenous or subcutaneous injection of adrenalin is not affected by a preceding intravenous injection of dihydroergocornine in man, whereas it is so in the cat. It might be argued that the discrepancy between our results in man and those obtained in the animal were due to the higher concentration possible in the latter. Obviously, if such an effect could be demonstrated in man, it would be of some practical importance. The question was, therefore, reinvestigated following intra-arterial injection of Hydergine, when higher concentrations reach the peripheral vessels.

Five subjects were chosen in whom the intra-arterial injection of Hydergine produced an immediate peripheral vasodilatation. When this had reached its maximum and was well established, adrenalin was injected intra-
arterially. A typical response is illustrated in figure 8, which was obtained in a patient showing no signs of any cardiovascular disease or disorder. As can be seen, the injection of 0.5 mg. Hydergine into the right femoral artery produced an immediate increase in pulse volume, digital volume and the rate of arterial blood flow. Seventeen minutes after the intra-arterial injection, 0.10 mg. of adrenalin was injected intra-arterially into the same limb, which resulted in an abrupt and powerful constriction of the arterial tree. The pulse volume, which before varied around 0.01 cc., disappeared completely, the digital volume fell to well below the level recorded before the injection of dihydroergocornine and the arterial inflow on venous congestion could not be recorded. This constriction of the arterial tree was sustained for more than five minutes. When the blood vessels relaxed, the blood flow returned to the

Fig. 9. Failure of intra-arterial Hydergine to modify the vasoconstrictor effect of a subsequent injection of adrenalin. Note, immediate dilatation following Hydergine (B) and vasoconstriction following injection of adrenaline resulting in the disappearance of the pulse volume (A'). Same case as figure 8. B and C: pulse volume after 0.5 mg. Hydergine into right femoral artery. A', B' and C': pulse volume 5,8 and 13 minutes after 0.1 mg. adrenalin into right femoral artery given 17 minutes after Hydergine.
highest value recorded after the injection of Hydergine, and the degree of reactive hyperemia was far in excess of that normally seen. In the opposite limb, the effect of the intra-arterial adrenalin was only slight and less than it would have been, had the injection been by the intravenous route.

Both the degree of vasodilatation, obtained from the intra-arterial injection of Hydergine, and the degree of vasoconstriction, recorded during the subsequent injection of adrenalin, are very well appreciated by consulting the cuttings from the plethysmogram (fig. 9).

Similarly, vasoconstriction produced by subcutaneous or intravenous injection of adrenalin is not affected by preceding intra-arterial injection of Hydergine.

(4) The Effect of an Intra-arterial Injection of a Mixture of Hydergine and Adrenalin: We know from previous investigations that dihydroergocornine does not affect the potency of adrenalin when mixed in vitro and given intravenously. This holds good for the intra-arterial route as well. In figure 10, 0.5 mg. dihydroergocornine plus 0.1 mg. of adrenalin were injected into the right femoral artery and the effect compared with an intra-arterial injection of 0.1 mg. of adrenalin, simultaneously administered in the left limb. Although the immediate reaction is the same in both limbs, that is, a complete arrest of the peripheral circulation for about five minutes, there is a difference when the circulation resumes. In the "normal" left leg, a moderate degree of reactive hyperemia is observed, the pulse volume and the rate of arterial inflow only temporarily assume a higher level and return to the original values within 30 minutes. In the right leg, however, in which Hydergine had been added, recovery is not only more rapid but the reactive hyperemia reaches a much higher level and the vessels go on to full dilatation and stay at that level, while under observation for the next 60 minutes. At the end of that time, the pulse volume in the right leg recorded 0.020 cc. as against 0.006 cc. in the left leg.

**DISCUSSION**

The results of the treatment of peripheral vascular diseases by the hydrogenated alkaloids of ergot have not been entirely satisfactory. Although these drugs are sympathicolytic in action, their effect on the peripheral vessels, when administered by the intravenous route, is unpredictable. In contrast, Bircher and Cerletti reported that, when given arterially, these drugs invariably produced peripheral vasodilatation in the cat, which seemed to fit in with the observations of Edwards and associates. They state that the results from intra-arterial therapy are better than with any other type of treatment—excluding sympathectomy.

In our experience, the effect of the hydrogenated alkaloids of ergot is the same whether given by the arterial or by the venous route. Only in about half the patients did we record any peripheral vasodilatation following intra-arterial injection of Hydergine or dihydroergocornine, as judged by a rise in skin temperature, pulse volume and rate of arterial inflow, but failed in the remaining limbs to produce any visible effects. This failure was particularly striking when it occurred in patients with a high vasomotor tone, who, theoretically,
should be most responsive to the administration of sympathicotomolytic drugs. In this, our observations did not agree with the results of Bircher and Cerletti, obtained in the anesthetized cat. However, this is not very surprising as it is well known that the results obtained in the animal cannot necessarily be transferred to man, particularly, when the sympathetic nervous system is involved.

Although it may be concluded that Hydergine had no effect in those cases where it failed to produce vasodilatation, this would be misleading. The intra-arterial injection of Hydergine did produce changes, most likely on the neurovascular apparatus, which are only revealed when we start dilating the vessels, either by body heating or by producing reactive hyperemia. We then discover that the vessels of the injected extremity respond sooner and to a greater degree to these procedures.

Rothlin has already drawn attention to the fact that the various effects of the ergot alkaloids can be divided into two groups; those which are immediately visible and those which are latent and will only be apparent when the respective organ is stimulated. No doubt we are dealing here with the same type of reaction; the intra-arterial injection of Hydergine produces both, the immediately visible and latent or potential effects. Although in the past only the former effects have been considered, the latter are no less important on account of their therapeutic potentialities. It would appear that therapy with the hydrogenated alkaloids of ergot could be made more effective by combining the intra-arterial injection with other vasodilator procedures, notably body heating, but not necessarily applied to such a degree as to produce general vasodilatation throughout the body.

Vasodilator therapy suffers from one major disadvantage. If given by the oral, subcutaneous or intravenous route, dilatation, if effective, affects all vessels throughout the body. If all vessels are normal, as in many vascular "disorders," there is no harm in this. But if the resistance in one area, due to arterial "disease" or other reasons, should differ from the remainder, vasodilatation may affect the delicately balanced distribution of the blood considerably and may lead to a marked movement of blood from the area with the higher into that with the lower resistance. Consequently, vasodilator therapy in cases of arterial disease may achieve the exact contrary to what it sets out to do.

Intra-arterial instead of intravenous therapy obviously offers a way out of this dilemma. It is able to overcome some of the difficulties inherent in intravenous therapy with vasodilator and sympathicotomolytic drugs. Its advantages are the possibility of producing more powerful dilatation by virtue of the selective effect and the higher local concentration, achieved from smaller doses than are necessary with intravenous therapy. Thus, the effect of intra-arterial therapy is purely local, without dilatation in the rest of the body with its accompanying side-effects. In this, intra-arterial therapy can be compared with treatment by para-vertebral block. However, it is obvious that unless such drugs which are to be given intra-arterially produce vasodilatation constantly, repeated arterial puncture will not be justified, and intra-arterial therapy has come in for some criticism in this respect.

In a previous communication, we reported that dihydroergocornine suppressed or reversed the rise in blood pressure, following the injection of adrenalin. Although we could not demonstrate a similar effect with respect to the effect of adrenalin on the peripheral vessels, Bircher and Cerletti subsequently found that in the cat, Hydergine, when given intra-arterially, abolishes and reverses peripheral vasoconstriction normally produced by adrenalin. We have now confirmed our previous results and can state that Hydergine, when given intravenously or by the intra-arterial route, exhibits no adrenolytic action on the peripheral vessels in man.

Summary

The effect of intra-arterial Hydergine or dihydroergocornine on the peripheral blood vessels has been recorded and compared with the blood flow in the contralateral limb. The immediate result is unpredictable, vasodilatation occurring in about half of the cases. In none of our cases was there ever any direct
vasoconstrictor action recorded. In principle, the effect was, therefore, the same as after an intravenous injection.

Although the intra-arterial injection of the hydrogenated alkaloids of ergot may not produce any visible effects on the peripheral blood vessels, the injection has not been without effect, as it does change their responsiveness to the release of the central vasoconstrictor tone. It was found that after an intra-arterial injection of Hydergine, reflex vasodilatation, as produced by body heating, occurs considerably earlier and proceeds to higher levels than in the contralateral normal limb.

The intra-arterial injection of the hydrogenated alkaloids of ergot produces, therefore, both visible and potential changes in the peripheral blood vessels. The potential changes can be demonstrated for many hours following the original injection and are of considerable therapeutic interest.

The vasoconstrictor effect of adrenalin on the peripheral blood vessels is not affected by preceding intra-arterial injections of Hydergine or dihydroergocornine. It is, therefore, confirmed that in man the hydrogenated alkaloids of ergot have no adrenergic properties in respect to the peripheral circulation.

The reactive hyperemia, observed following the temporary arrest of the peripheral circulation as produced by adrenalin, is greater and more lasting if this was preceded by an intra-arterial injection of Hydergine or dihydroergocornine.

In some respects, the results are contrary to observations made in the animal. This stresses the caution which has to be exercised, in trying to apply to man results obtained in animal experiments.

The special merits of intra-arterial, as opposed to intravenous therapy, are discussed with special reference to the treatment of cases of peripheral vascular diseases.

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SUMMARIO IN INTERLINGUA

Le effecto immediate del hydrogenate alkaloides de ergota, si administrate intra-arterialmente, es inpredicibile. Vasodilatation es obtenite solmente in certe patientes. Tamen, il esseva constata que in le casos que non reage per vasodilatation le vasos responde plus facilmente al relaxation del tono sympathetic, lo que prova que le droga habeva nonobstante un effecto (potential) probablemente super le apparato neurovascular. Iste effecto esseva demonstrabile durante multe horas e es consequentemente de alte interesse therapeutic.

Mesmo in le plus alte concentrationes local que es possibile per le administration intra-arterial, le hydrogenate alkaloides de ergota non exhibiva ulle action adrenolytic super le peripheric vasos sanguine of humanos.

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HYDERGINE AND DIHYDROERGOCORINE AND CIRCULATION


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