

nism. Such a possibility, however, appears to be remote in this case as: a) A combination of DNP and IAA is not synergistic to rooting (Table). b) DNP is not believed to exercise its effect through IAA metabolism, as DNP

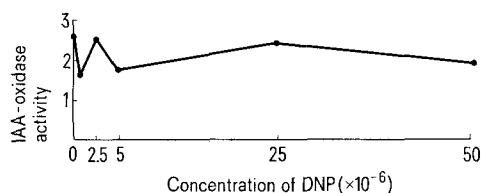


Fig. 1. In vitro effect of different concentrations of DNP on the activity of IAA-oxidase of the dark-grown *Phaseolus mungo* hypocotyl cuttings with excized apex and cotyledons. IAA-oxidase activity was assayed as described earlier and expressed in terms of μg IAA oxidized under specified conditions.

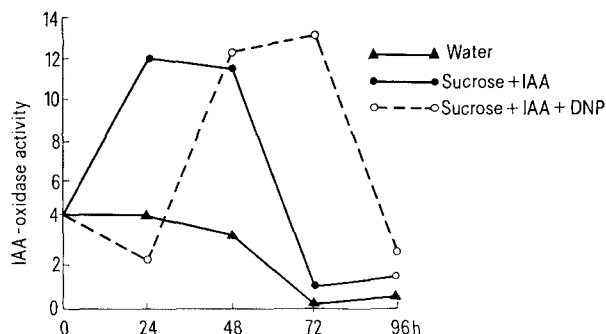


Fig. 2. Effect of DNP in combination with IAA+sucrose on the in vivo IAA-oxidase activity of the dark-grown *Phaseolus mungo* hypocotyl cuttings with excized apex and cotyledons. IAA-oxidase activity was assayed as described earlier and expressed in terms of μg IAA oxidized under specified conditions.

exerted no influence on in vitro IAA-oxidase activity (Figure 1); nor did the treatment with DNP have any considerable effect on the in vivo IAA-oxidase activity of the cuttings, except for a 'delaying effect' on the enzyme activity peak (Figure 2). Protection of IAA from oxidation¹³ or enhanced IAA synthesis¹⁴ by DNP has not been observed.

The enhancement of respiration by DNP by removing the obligatory link to phosphorylation is well known^{2,3,15,16}. Promotion of rooting by DNP might, therefore, involve the uncoupling of oxidative phosphorylation and increased respiration; 2×10^{-6} M Cadmium (Cd^{++}), a known inhibitor of respiration¹⁷, checked the DNP-caused promotion of rooting (Table), thus lending support to such a possibility. That the uncoupling of oxidative phosphorylation and not the interaction with auxin metabolism is likely to be involved in the DNP-caused promotion of rooting, is further supported by the fact that arsenate (10^{-6}M), a non-phenolic uncoupler of phosphorylation¹⁸, also enhanced rooting (Table), contrary to the earlier observations¹⁹.

Thus, increased root production by DNP, a *para* substituted phenol, appears to be related to uncoupling of oxidative phosphorylation and is considerably different from that by other phenolic compounds which act through IAA metabolism¹⁰⁻¹².

¹³ P. L. GOLDACER, A. W. GALSTON and R. L. WEINTAUB, *Arch. Biochem. Biophys.* **43**, 358 (1953).

¹⁴ S. A. GORDON and L. G. PALEG, *Plant Physiol.* **36**, 838 (1961).

¹⁵ H. A. LARDY, P. WITOUSKY and D. JOHNSON, *Biochemistry* **4**, 552 (1965).

¹⁶ H. A. LARDY, J. L. CONNELLY and D. JOHNSON, *Biochemistry* **3**, 1961 (1964).

¹⁷ H. R. MAHLER and E. H. CORDES, *Biological Chemistry* (Harper and Row Publishers, New York 1971), p. 686.

¹⁸ H. F. TERWELLE and E. C. SLATER, *Biochim. biophys. Acta* **89**, 385 (1964).

¹⁹ W. R. KRUL, *Plant Physiol.* **43**, 439 (1968).

Relation of the Character of Capacitance Vessel Responses in Spleen and Small Intestine Elicited by Electrical Stimulation of the Sympathetic Fibres to the Level of Oxygen Exchange in the Organs

B. I. TKACHENKO, P. K. POZDNIJAKOV and N. JA. MEDVEDEVA

Institute of Experimental Medicine, Laboratory for Circulation, Leningrad P-22 (USSR), 10 October 1975.

Summary. There is a correlation between character and magnitude of capacitance vessel responses and venous blood oxygen saturation under electrical stimulation of sympathetic fibres in the spleen and small intestine.

It was shown¹ that the electrical stimulation of sympathetic fibres caused both constrictory and dilatory responses of the spleen and small intestine capacitance vessels, resistance vessel response being always constrictory. The lack of uniformity of the capacitance vessel responses was not due to the resistance vessel responses, capillary filtration and changes in arteriovenous anastomotic flow. In spite of the fact that the dilatory responses of the capacitance vessels were observed in small number of experiments (21% for spleen and 16% for intestine), it was necessary to study mechanisms of these reactions. This study was intended to compare the dynamics of changes of venous blood oxygen saturation and the character of the capacitance vessel responses.

Method. The experiments were performed on cats (30) anaesthetized with urethane (1 g/kg) under artificial respiration. A spleen and a section of small intestine were humorally isolated and autoperfused with a constant blood volume pumps. The resistance and capacitance vessel reactions were studied by the method described previously²⁻⁴. The arterial pressure, the perfusion pressure and the venous outflow were recorded with an electro-manometer on the optical oscillograph. Simultaneously with the resistance and capacitance vessel responses, the dynamics of the venous blood oxygen saturation was recorded with an oxymeter (model 057) using a probe attached to the special glass cuvette. The stimulation of the splanchnic or spleen sympathetic fibres was accom-

plished with a right-angle impulse generator (0.25–30 imp/sec, 6v, 5 msec). Experimental data were processed in a digital computer 'Minsk-32', the factor and regression analysis being used.

Results. Our experiments showed that the electrical stimulation of intestinal vasomotor fibres produced the constriction of resistance vessels in the intestine; the intestine vascular capacity decreased in 60%, increased in 16% and was not changed in 24% of the experiments. The oxygen saturation of the intestine venous blood decreased in 82%, increased in 12% and was not changed in 6% of the experiments. The electrical stimulation of spleen vasomotor fibres caused in all experiments a constriction of resistance vessels. Capacity of the spleen vascular bed decreased in 67%, increased in 21% and was unchanged in 12%. Oxygen saturation of spleen venous blood decreased in 57%, increased in 30% and was unchanged in 15% of the experiments.

Since the qualitative analysis did not reveal any distinct relation between vasomotor reactions and venous blood oxygen saturation in investigated organs, we performed quantitative analysis of these data using mathematical methods.

There was no correlation between both the value and character of capacitance and resistance vessel responses, elicited by electrical stimulation of sympathetic fibres: correlation coefficient was near the zero (for spleen $r = -0.029$; $p > 0.05$, for intestine $r = 0.138$, $p > 0.05$). At the same time we revealed the correlation between the value of resistance vessel response and the character of venous blood oxygen saturation changes in the organ. When the increase of perfusion pressure in intestinal vessels under electrical stimulation of splanchnic nerve was not more than 30–35 mm Hg, the venous blood oxygen saturation might be either decreased or increased. In these experiments, where the value of perfusion pressure increase was higher than 30–35 mm Hg, the venous blood oxygen saturation was always increased. The latter correlation

was not observed in experiments on spleen because perfusion pressure decrease under electrical stimulation of spleen nerve was not higher than 30 mm Hg.

Analysis of correlation between character and magnitude of capacitance vessel responses and absolute values of venous blood oxygen saturation changes under electrical stimulation of sympathetic fibres showed reversed line correlation between these parameters in both organs (Figure 1). The decrease of venous blood oxygen saturation during the sympathetic stimulation was accompanied as a rule by the constriction of capacitance vessels (venous outflow increase) and the greater the decrease of the venous blood oxygen saturation the greater was the constrictory response. When the increase of the venous blood oxygen saturation was comparatively small, the venous outflow might be both increased and decreased, but the greater the increase of the first parameter, the greater and more often was the decrease of the venous outflow, i.e. dilatation of capacitance vessels.

Rather close correlation was revealed as well between the peak values of venous blood oxygen saturation and both the magnitude and character of capacitance vessel response elicited by electrical stimulation of sympathetic nerves in either organ investigated (Figure 2a). When the

- ¹ B. I. TKACHENKO, N. JA. MEDVEDEVA and P. K. POZDNIJAKOV, *Experientia* 30, 1413 (1974).
- ² B. I. TKACHENKO, N. G. KRASILNIKOV, S. A. POLENOV and G. V. CHERNJAVSKAJA, *Experientia* 25, 38 (1969).
- ³ B. I. TKACHENKO, D. P. DWOREZKY, N. I. OWSJANNIKOV, A. W. SAMOJLENKO and W. G. KRASILNIKOV, *Regional and Systemic Vasomotor Reactions* (Medicine, Leningrad 1971).
- ⁴ B. I. TKACHENKO and G. W. CHERNJAVSKAJA, *Experientia* 27, 782 (1971).

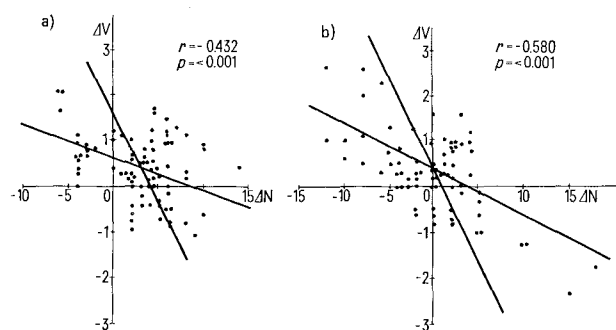


Fig. 1. The character of relation between venous blood oxygen changes and volume of the venous outflow in the intestine (a) and spleen (b) under electrical stimulation of the sympathetic fibres. Abszissa; venous blood oxygen saturation change (% Hb O₂); ordinate, outflow changes (ml).

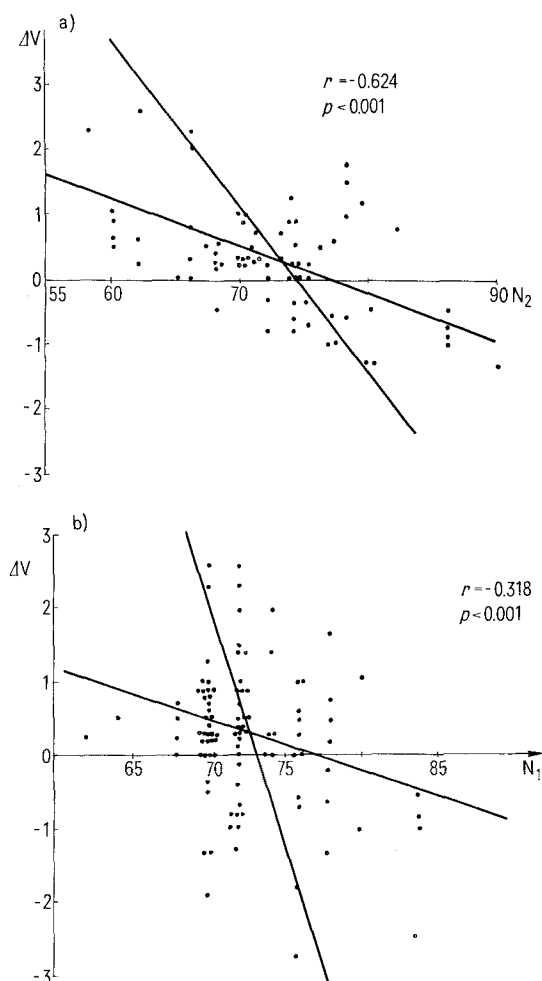


Fig. 2. The character of relation between the peak (a) and initial (b) values of the venous blood oxygen saturation and venous outflow changes in spleen under electrical stimulation of spleen nerve. Abszissa, peak (a) and initial (b) means of venous blood oxygen saturation. Ordinate, venous outflow changes.

peak value of venous blood oxygen saturation during reaction was not more than 68% HbO_2 , venous outflow constantly increased. When the peak value reached 70 to 85% HbO_2 , the venous outflow either decreased or in-

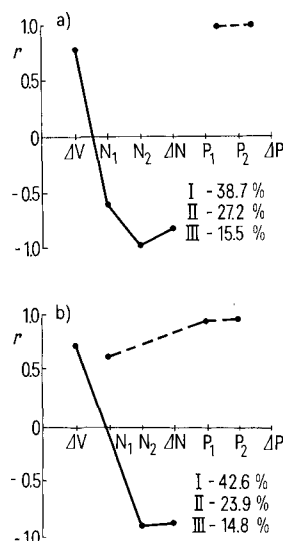


Fig. 3. Results of the factor analysis of vasomotor reactions and dynamics of venous blood oxygen saturation changes in the spleen (a) and intestine (b) under electrical stimulation of sympathetic fibres. Abszissa, analyzed prizmaks; ordinate, means of coefficient of correlation. Line, I factor; interrupt line, II factor; point, III factor. Weights of factors: a) I, 38.7%; II, 27.2%; III, 15.5%. b) I, 42.6%; II, 23.9%; III, 14.8%. V, venous outflow (ml); N_1 , initial level of the venous blood oxygen saturation (% Hb O_2); N_2 , peak means of the venous blood oxygen saturation (% Hb O_2); ΔN , venous blood oxygen saturation changes; P_1 , initial level of the perfusion pressure; P_2 , peak means of the perfusion pressure; ΔP , the perfusion pressure changes.

creased. When venous blood oxygen saturation reached, under stimulation of sympathetic nerves, the value of 85% HbO_2 or more, the venous outflow decreased. We revealed as well reversed line correlation between initial level of venous blood oxygen saturation and the character of venous outflow from an organ under electrical stimulation of sympathetic fibres (Figure 2b). It turned out that if the initial level venous blood oxygen saturation was small, i.e. the oxygen consumption by the tissue of an organ was high, venous outflow as a rule was increased. On the contrary, the more the venous blood oxygen saturation before stimulation, the more evident was the tendency to the decrease of the venous outflow under electrical stimulation of sympathetic fibres.

To elucidate the relation between both the value and character of capacitance vessel responses of spleen and intestine and all the parameters recorded, we used one of the methods of the factor analysis – method of chief components. It was shown (Figure 3) that the most essential factor was the close negative correlation between magnitude and character of capacitance vessel responses from one side and dynamics of venous blood oxygen saturation from another.

Thus the results of the analysis permit us to suggest that the more intensive the oxygen exchange in spleen and intestine before and during the sympathetic stimulation, i.e. the more is venous blood desaturated with oxygen, the more frequent the constrictory response of capacitance vessels. On the contrary, the lower the oxygen exchange in spleen and small intestine, i.e. the more the venous blood saturated with oxygen, the more frequent is dilatory response of capacitance vessels.

It is necessary to emphasize, however, that this correlation was obtained in experiments with sympathetic fibres cut, i.e. when the organs investigated were practically deprived of neurogenic control. Whether this correlation exists in the normally functioning spleen and small intestine will be shown in further investigations.

Coordinated Activation of Muscle Fibres by Different Conduction Velocities in Branches of a Crustacean Motor Axon

C. K. GOVIND¹ and F. LANG²

Scarborough College, University of Toronto, West Hill, Ontario, (Canada M1C 1A4); and Boston University Marine Program, Marine Biological Laboratory, Woods Hole (Massachusetts, USA), 23 February 1976.

Summary. Higher conduction velocities in branches of the fast excitator axon to distal muscle fibres ensure that these fibres are activated almost simultaneously with proximal fibres in the claw closer muscle of lobsters, producing a contraction of maximal force.

In muscle contraction, maximal force is exerted when all fibres within a muscle (or motor unit) are excited simultaneously. The resulting coordination problem is accentuated in crustaceans, in which entire muscles are innervated by few (1–4) excitatory motor axons, so each neuron serves a large number of muscle fibres. In addition, the muscle fibres are arranged in pinnate fashion resulting in a wide separation between proximal and distal fibres (Figure A). What is the mechanism for exciting the fibres within such muscles so that they contract almost simultaneously? In the lobster claw closer muscle, different conduction velocities in the branches of a single excitatory axon allow widely separated muscle fibres to be activated within a few milliseconds of one another.

The claw closer muscle in lobsters is the largest limb muscle and is innervated by 2 excitatory (fast and slow) axons and an inhibitory axon³. The fast axon is the major motor fibre to the dorsal surface of the muscle, although all regions of the muscle are innervated by both axons to some extent⁴. Furthermore, it is possible to selectively stimulate the fast axon, since it invariably has the lowest threshold of all 3 axons⁴. We therefore examined the temporal differences in excitation of widely separated dorsal fibres when the fast excitator was stimulated.

In a typical experiment, the dorsal surface of the closer muscle was exposed by removing the opener muscle. A proximal and a distal fibre were penetrated simultane-