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The action of piracetam in ischaemic flaps

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Summary—The effects of piracetam on skin flap viability, capillary blood flow and temperature were studied in abdominal cutaneous flaps in rats.

The drug significantly increased the viability of the distal (random) portion of the flap and this response was dose-related. Compared with controls, the area of skin necrosis was 12.4% less in the piracetam-treated animals and extended necrosis was not observed.

Piracetam appeared to act by increasing the capillary blood flow, mainly in the distal portion of the flap.

The improved perfusion was reflected as a smaller drop in the recorded temperature compared with controls.

Blood levels of this drug in animals receiving the maximum effective dose corresponded to the active drug concentration in human rheology.

Piracetam (Nootropil®, UCB), is a pyschotropic drug which directly improves the efficiency of higher telencephalic functions of the brain (Mindus *et al.*, 1976). It has been used over the past decade in the management of psychosenescent syndromes.

Since 1979, several authors have reported the beneficial effect of this agent on microcirculation: piracetam has been shown to enhance deformability of erythrocytes (Henry *et al.*, 1981) and to decrease their adherence to vascular endothelium (Nalbandian *et al.*, 1983). This agent also suppresses platelet membrane activity, contact activation and the release of platelet-factor 4 and beta-thromboglobuline (Bick, 1979; Bick *et al.*, 1981). Last but not least, it relieves induced vascular spasm (Reuse-Blom and Polderman, 1980).

On the basis of these newly described effects, this study has been designed to assess the beneficial effect of piracetam on ischaemic flaps in rats.

Three experiments were performed to investigate the *in vivo* effect of piracetam on skin flaps designed to be partially ischaemic:

(i) The first experiment assessed the effect of different doses on skin viability and determined the dose-response effect.
(ii) The second measured the capillary blood flow with and without the administration of the drug.
(iii) The last experiment evaluated the drug effect when given after surgery. It was also designed to evaluate the difference of skin flap temperature with and without the drug.

Materials and methods

Experimental animal model

Wistar rats weighing 289 ± 2 g were housed in individual cages in a controlled room with a temperature of 23°C and light from 08.00 hours to 18.00 hours. They were offered food and water *ad libitum* but food was withdrawn 6 hours before surgery or measurement. They were anaesthetised with intramuscular neuroleptanaesthetic (fluanison 1 mg/100 g + Fentanyl 0.02 mg/100 g).

In the three experiments we studied an abdominal flap 9×3 cm based on the thoracic artery (Fig. 1). Modified from the model described by Norton *et al.* in 1984, it has an axial pattern with a random pattern extension.

The flap was designed after clipping the hair. The medial border was the midline and the distal limit was a point 1 cm proximal to the pubis.

After careful incision of the distal border, the epigastric artery was ligated with 8/O nylon to prevent neovascularisation. The flap was raised from its distal end toward the pedicle under the panniculus carnosus as shown in Figure 2. It was directly returned to its bed and closed with 4/0

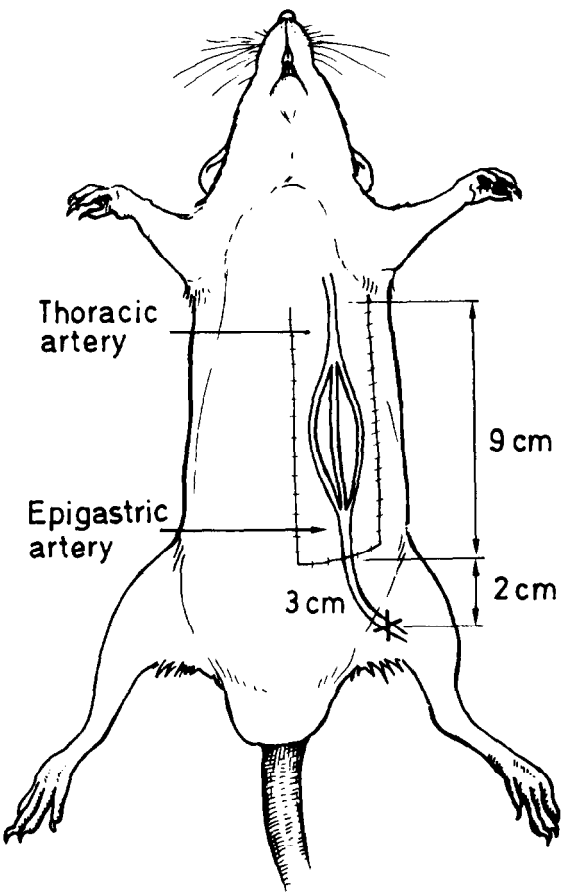

Fig. 1

Figure 1—Experimental model. Schema of the 9 × 3 cm abdominal flap with thoracic pedicle outlined on each animal.

prolene interrupted sutures and with additional vertical mattress sutures at its angles.

The thoracic blood supply was not sufficient to maintain a full vascularisation of the flap; the distal portion became ischaemic and variably necrotic (Fig. 3).

Experiment 1

In this experiment, the necrotic area of the flap was measured each day for 8 days with a transparent grid divided into squares of 0.25 cm^2 as described by Griffiths and Humphries (1981). Since there was no contraction of the flap, the results were expressed as a percentage of the original flap area.

Five experimental groups were studied, each containing 30 animals. Control group 1A received the placebo (distilled water) whereas groups 1B, 1C, 1D, 1E received respectively the piracetam at a dose of 20, 40, 80 and 100 mg/100 g/day.

Each animal received a first intramuscular dose of piracetam or placebo along with the anaesthetic. Then, for 8 days after surgery, they daily received the same oral dose of piracetam whereas control animals received the placebo.

Piracetam blood concentration was determined by gas chromatography in the rats receiving 40 mg/100 g/day.

Experiment 2

Sixty rats divided into five groups were studied. The blood flow in four zones of the skin flap, the renal blood flow and the cardiac output were measured as follows. Group 2A (n = 10) was not operated on. The measurements were made immediately. Group 2B (n = 20) was operated on and received the placebo exactly as in group 1A. Their measurements were made on the second postoperative day. Group 2C (n = 20) was operated on and received 80 mg/100 g/day piracetam exactly like group 1D. The measurements were made on the second postoperative day as in group 2B. Group 2D (n = 5) was operated on and received the placebo as in group 1A. Measurements were assessed on the fifth postoperative day. Group 2E (n = 5) was operated on and received 80 mg/100 g/day piracetam exactly like group 1D. Measurements were made on the fifth postoperative day.

The cardiac output and the capillary blood flow were measured with radioactive microspheres (Malik *et al*, 1976; McDevitt and Nies, 1976). In the present experiment, 57 Cobalt labelled microspheres (15 ± 0.02 μm diameter) (New England Nuclear, Boston, Mass.) with a specific activity of 10 mCi/g were used.

In each rat to be injected with microspheres, the right carotid and right femoral arteries were catheterised with polyethylene-50 tubing. Using pressure monitoring (Beckman), a carotid cannula was introduced into the left ventricle (LV). Microspheres were diluted in dextran 10% solution to reach a concentration of about 200,000 microspheres/ml. An homogeneous suspension was obtained by careful mixing with a vortex shaker. 0.4 ml of Co^{57} solution was drawn into a glass injection chamber and the radioactivity determined by a gamma counter (Berthold BF Gamma S ZINT 5300) before and after microsphere injection, the

Fig. 2

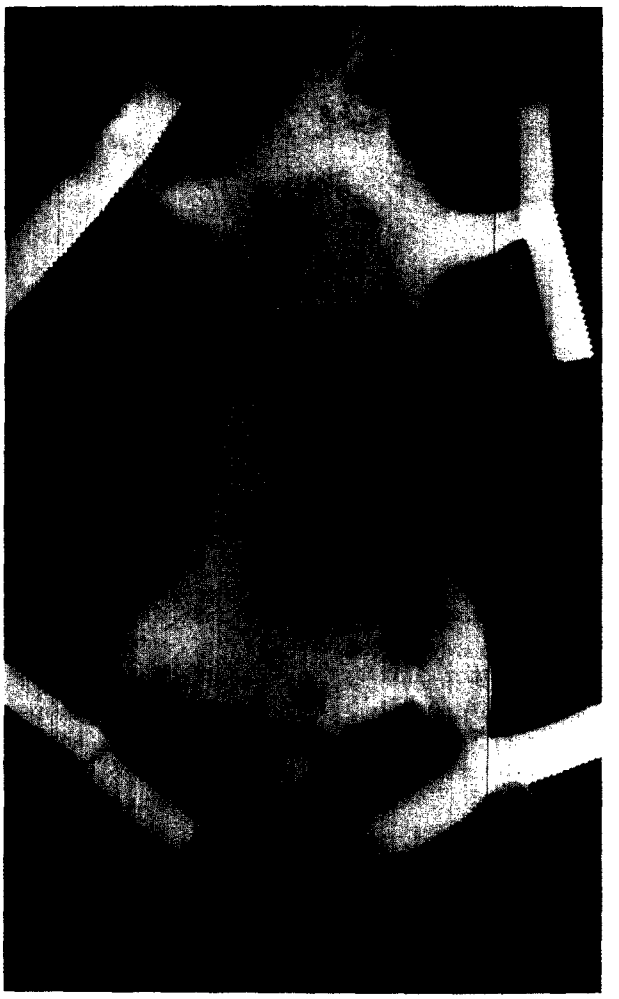

Fig. 3

Figure 2—The elevated flap. When flaps were constructed the inferior epigastric pedicle was cut, the sides were incised and the flap was undermined so that only the superior epigastric neurovascular bundle remained intact. Figure 3—Necrosis of the tip of the flap.

difference being the amount of radioactivity injected. The solution was injected into the LV catheter over 20 seconds. Simultaneously, arterial blood from the femoral artery was withdrawn at 1 ml/min for 60 seconds with a Harvard syringe withdrawal pump. This reference blood sample was used to calculate the cardiac output by the formula:

$$\text{cardiac output} = \frac{\text{counts injected} \times \text{reference sample withdrawal rate}}{\text{reference sample counts}}$$

After obtaining the reference sample, the animals were sacrificed with an overdose of pentobarbitone (60 mg/100 g).

The flaps were then divided into 4 segments of 2.25 × 3 cm. Each segment was weighed and radioactivity measured.

The capillary blood flow per gram of tissue was calculated in each portion of the flap and in the two kidneys according to the method described by Malik *et al.* (1976):

$$\frac{\text{organ flow}}{\text{activity in the organ}} = \frac{\text{reference sample flow}}{\text{activity in reference sample}}$$

In order to control the homogeneity of the

microspheres, the blood flow per gram of tissue of the two kidneys was compared. Animals were discarded if a 10% difference or more was noted.

Experiment 3

Thirty rats divided into 2 groups were studied. Control group 3A (n = 15) received the placebo whereas group 3B (n = 15) received the piracetam at a dose of 80 mg/100 g.

Each rat received a first dose of isotonic saline or piracetam intravenously 30 minutes after the elevation and suture of the flap. Then, for 8 days after surgery, the same oral dose of placebo or piracetam was administered. A different control solution was used for the IV injection to avoid the haemolysis caused by an IV injection of distilled water.

The area of necrosis was measured in group 3B (piracetam-treated) as in the first experiment and compared with the data of group 1D in order to assess whether preoperative administration of piracetam was important.

The difference of temperature between the skin flap and normal skin was recorded in the two groups 3A and 3B according to the technique of Jones *et al.* (1983). The device used was a Vasotract Thermocouple that measured simultaneously six different temperatures.

The first measurement was performed in the middle of the four segments described in the second experiment (1.13 cm, 3.38 cm, 5.63 cm, 7.88 cm from the base of the flap) and at the corresponding points on the contralateral side every 24 hours after the elevation of the flap.

To abolish the variations of local conditions and central physiological changes, the four measurements of flap temperature were calculated with reference to a symmetrical contralateral electrode, as described by Jones *et al.* (1983).

Statistics

Statistical analysis was performed with an IBM XT-3 computer using the NWA Statpack programs.

All values were expressed in mean value plus or minus the standard error of the mean (SEM) Analysis of variance (F-test) and Student's *t*-test were performed to compare the mean values.

Linear regression and correlation coefficient were used to study the relationship of temperature and distance from the base of the pedicle.

Significance of the difference was considered at the $p < 0.05$ level.

Results

Experiment 1

Expressed as a proportion of the total original flap area, the extent of flap necrosis measured at the 7th postoperative day is illustrated diagrammatically (Fig. 4).

Control series 1A necrosed an area of 13.8 ± 2.6%, with an extensive range (0–75%) as seen in the histogram of Figure 5.

As soon as 20 mg/100 g piracetam were admin-

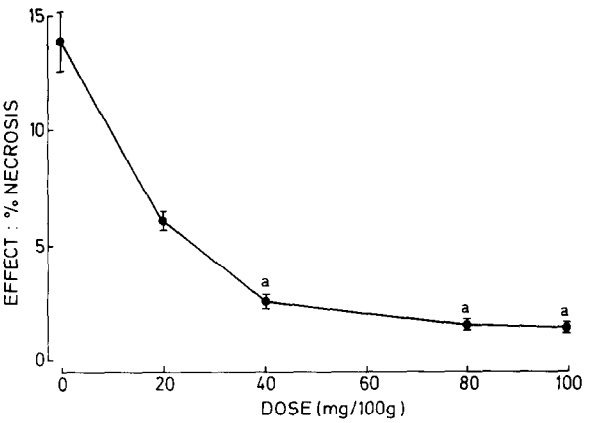

Fig. 4

Figure 4—Dose-effect curve: percentage of skin flap area that has necrosed according to different doses of piracetam (mean ± SEM; N = 30). Means without a common letter are significantly ($p < 0.05$) different.

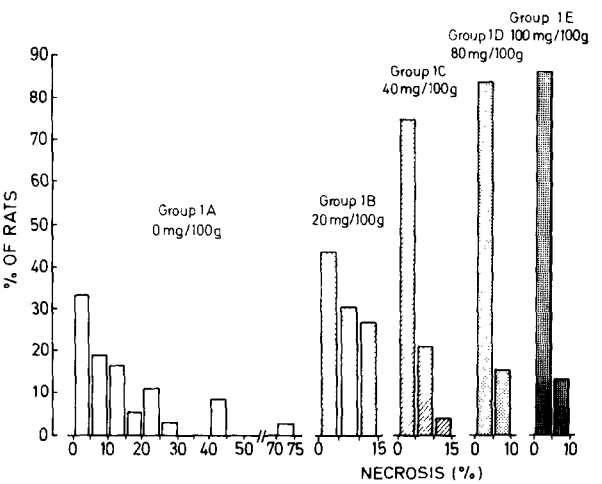

Fig. 5

Figure 5—Histogram showing the percentage of rats corresponding to the area of necrosis observed in the control animals and in the piracetam-treated groups (N = 30).

istered (group 1B), we observed a significant decrease of the necrotic surface ($p < 0.0037$) when compared with the placebo-treated animals.

Moreover, extended necrosis was never evidenced in the piracetam-treated animals, in marked contrast with the control group.

Following a dose of 40 mg/100 g, skin necrosis was either non-existent or very limited. A maximum skin necrosis of 5% was observed in the following percentages of animals: 75% (Group 1C); 84.3% (Group 1D); 86.2% (Group 1E).

The skin necrosis decreased with increasing dosage of piracetam (Figs 4 and 5). From a dose of 40 mg/100 g BW, the dose/response curve reached a plateau. There were no significant differences between doses from 40 to 100 mg/100 g with respect to the necrotic area limitation. However, the dose of 20 mg/100 g was less effective in reducing the necrotic area as there was a marked difference when comparing group 1B with group 1C ($p < 0.0006$), and group 1D ($p < 0.00001$).

Piracetam blood level measured in group 1C revealed levels of 0.45 ± 0.06 mM), which corresponded to the active concentration in human rheology (>0.45 mM) (Costa *et al.*, 1979; Sonnet *et al.*, 1985).

Experiment 2

The values for cardiac output calculated with the microspheres technique were not statistically different between groups 2A, 2B and 2C.

The overall mean value was 25.8 ± 2.5 ml/min/100 g.

The mean abdominal skin blood flow measured in group 2A (unoperated) averaged 0.065 ± 0.0032 ml/min/g. No significant differences occurred within the region of the proposed flap.

The mean blood flow measured in control group 2B (operation + placebo) increased significantly ($p < 0.0002$) to 0.094 ± 0.005 ml/min/g.

The distribution of the flow within the flap had changed compared to that in the unoperated abdominal wall—there was a decrease of the flow from the base to the tip of the flap. Comparison of the blood flow in different portions of the flap demonstrated a significantly reduced flow between 1 and 2 ($p < 0.02$), 2 and 3 ($p < 0.01$) and 3 and 4 ($p < 0.01$).

With piracetam (group 2C), the blood flow significantly increased in the four segments of the flap when compared with control group 2B: $+67.7 \pm 14.7\%$ ($p < 0.0001$) in the first segment; $+67.8 \pm 17.8\%$ ($p < 0.0003$) in the second segment; $+98.4 \pm 20.9\%$ ($p < 0.0000$) in the third segment and $+116.8 \pm 34.3\%$ ($p < 0.0012$) in the fourth distal segment (Fig. 6).

The largest increase of blood flow was found in the most distal segment. In this segment, the blood flow of treated rats (piracetam) was more than twice that of control flaps (placebo).

Similarly, 5 days after the elevation of the flap the blood flow was significantly increased ($p < 0.0009$) with piracetam (group 2E) when compared to control group 2D.

Experiment 3

The mean area of necrosis of the piracetam-treated group 3B was $1.4 \pm 0.5\%$. There were no significant differences between this group and group 1D of the first experiment treated before the elevation of the flap.

Temperature was inversely correlated with the distance from the flap base ($r = -0.99$; $p < 0.005$ in the control series; $r = -0.98$; $p < 0.01$ in the piracetam series). With piracetam, the flap temper-

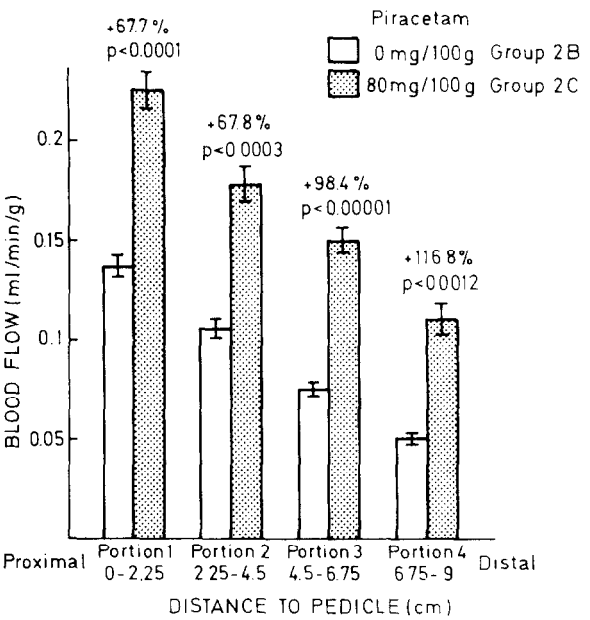

Fig. 6

Figure 6—Blood flow in the four portions of the flap in control animals (group 2B) and 80 mg/100 g piracetam-treated rats (group 2C). The largest increase was observed in the most distal segment.

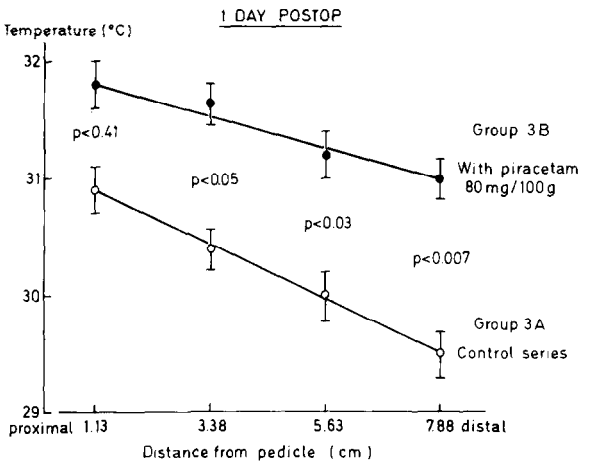

Fig. 7

Figure 7—Linear regression line of skin temperature in control and piracetam-treated rats related to distance from pedicle (mean ± SEM; N = 15).

ature decrease was reduced (slope of 0.28 ± 0.04 against 0.44 ± 0.01) (Fig. 7).

At the most proximal point of measurement, the temperature difference between control group 3A and piracetam-treated group 3B was not significant. The difference in temperature gradually increased until reaching a maximum level at the tip of the flap. At that point, the difference was statistically the most significant ($p < 0.007$).

Discussion

Many factors which might induce necrosis in ischaemic flaps have been described (Myers and Cherry, 1968; Hoopes and Im, 1978; Marks *et al.*, 1984):

(i) The decrease of the capillary blood flow.
(ii) The opening of A/V shunts which deviates the blood flow towards thick wall vessels, thus preventing nutritional exchange.
(iii) The release by neighbouring tissues of substances which are toxic for healthy tissues.

Many drugs have been proposed to reduce or prevent flap necrosis. Some of them induced arteriovenous shunt closure (6 hydroxydopamine; Reinisch, 1974), others caused vasodilatation (Finseth and Adelberg, 1978, 1979) but their effects were not really convincing.

So far, no drug that directly acts on the capillary microcirculation has been tested.

Piracetam may improve microcirculation (Bick, 1979; Reuse-Blom and Polderman, 1980; Henry *et al.*, 1981; Nalbandian *et al.*, 1983). The aim of this study was to check if this drug can increase flap viability.

The experimental model we used originated from Norton's model. In our control series, the mean area of necrosis (13.8%; range of 0% to 75%) was smaller than that reported by Norton. Actually our flap was shorter (9 cm instead of 11 cm) and had a wider base (3 cm instead of 2 cm). Moreover, Norton employed an island flap.

A wide range of necrosis was recorded, as in other flaps already described in the rat (Donovan, 1975; Sasaki and Harii, 1980; Griffiths *et al.*, 1981). Flaps might indeed survive as skin grafts, the bed providing the vascular supply.

In order to get a more constant area of necrosis, some authors have ingeniously interposed a polyethylene sheet between the flap and bed (Dibbell *et al.*, 1979; Griffiths *et al.*, 1981).

Piracetam markedly improved the skin viability of thoracic flaps in rats. This beneficial effect was already observed with a dose of 20 mg/100 g but an even more significant response was recorded with the 40 to 100 mg/100 g doses. With a dose of 80 mg/100 g, the mean surface of necrosis decreased by 12.1%.

In addition, in the piracetam-treated rats there was almost no variation in the area of necrosis. With a dose of 80 mg/100 g, the standard error of the mean was very low (0.42%) and 83.3% of the rats were found in the 0% to 5% range of necrosis.

Therefore piracetam seemed to act mainly on ischaemic tissues, reducing necrosis.

We have tried to explain the improvement in flap viability, using radioactive microspheres as an indicator of blood flow distribution.

There was no significant difference in the cardiac output in the diverse experimental groups and all values were within the ranges reported by others for rats of the same weight (Sasaki and Wagner, 1971; (Malik *et al.*, 1976; McDevitt and Nies, 1976; Kennedy *et al.*, 1979; Ishishe *et al.*, 1980).

Many investigators have accurately measured the physiological flow in various organs in rats, but few have measured the cutaneous capillary blood flow (Kennedy *et al.*, 1979).

The mean blood flow distribution to the abdominal skin in our group 2A (no flap elevation) was similar to the flow measured by others in rats' dorsal skin (Kennedy *et al.*, 1979).

Two days after flap elevation, the control series

blood flow (group 2B) had decreased significantly from the flap base to its distal point. It fell below critically low values only at the level of zone 4 where we found mainly necrosis. Therefore, in agreement with Kennedy *et al.* (1979), the capillary blood flow seems to be a determinant factor in flap survival.

When compared with group 2B, the total flap blood flow increased significantly ($p < 0.00001$) with piracetam (group 2C). While the blood flow was raised at the base (+67.7%), the main increase was noted in the distal random portion of the flap (+116.8%). Therefore piracetam mainly increased the capillary blood flow of the distal ischaemic tissues doomed to necrosis and raised significantly the perfusion distance.

Because the microsphere method does not allow the record of the blood flow variations in the same animal, we used, in experiment three, a non-invasive method with a thermocouple.

Flap temperature is an indirect measurement of the global blood flow: capillary + arteriovenous shunts (Reinisch, 1974; Jones *et al.*, 1983).

As flap perfusion decreased from the base towards the periphery, there was a temperature decrease proportionate to the distance. The inverse correlation observed was excellent. With piracetam, the improved perfusion of the flap found expression in a smaller decrease of temperature. We did not observe the peak of temperature in the distal zone 4 described by Reinish (1974) that would correspond to arteriovenous shunt opening.

In clinical use, it has been demonstrated that a vasodilator should be given prophylactically in situations of threatened necrosis (Finseth and Adelberg, 1978, 1979). In experiment three, we have shown that piracetam can be effective even if administered after the operation: indeed, the area of necrosis was not significantly different when piracetam was administered before (group 1D) or 30 minutes after the operation (group 3B).

Finally, piracetam has been known as a non-toxic agent for 19 years (Kunneke and Malan, 1979; Wilsher *et al.*, 1979; Mark *et al.*, 1985).

A 40 mg/100 g oral dosage of piracetam in rats resulted in a plasma level equivalent to that required in man to act on the microcirculation. This level is reached in man by the administration of 160 mg/kg/day (Sonnet *et al.*, 1985).

From evidence available in this study, the use of piracetam is suggested to protect and improve microcirculation in surgically created flaps which are known by experience to be risky.

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References

Bick, R. L. (1979). In-vivo platelet inhibition by Piracetam. *Lancet*, **2**, 752.

Bick, R. L., Skondia, V. and Fritch, D. (1981). Hypercoagulability in diabetes and its correction by an antiplatelet drug: Piracetam. *Clinical Research*, **29**, 400A.

Costa, F. F., Zago, M. A. and Bottura, C. (1979). Effects of Piracetam on iodoacetamide on erythrocyte sickling. *Lancet*, **2**, 1302.

Dibbell, D. G., Hedberg, J. R., McCraw, J. B., Rankin, J. H. G. and Souther, S. G. (1979). A quantitive examination of the use of fluorescein in predicting viability of skin flaps. *Annals of Plastic Surgery*, **3**, 101.

Donovan, W. E. (1975). Experimental models in skin flap research. In *Skin Flaps*, edited by Grabb, W. C. and Myers, M. B. Boston: Little-Brown.

Finseth, F. and Adelberg, M. G. (1978). Prevention of skin flap necrosis by a course of treatment with vasodilator drugs. *Plastic and Reconstructive Surgery*, **61**, 738.

Finseth, F. and Adelberg, M. G. (1979). Experimental work with isoxsuprine for prevention of skin flap necrosis and for treatment of the failing flap. *Plastic and Reconstructive Surgery*, **63**, 94.

Griffiths, R. W. and Humphries, N. L. (1981). Isoxsuprine and the rat abdominal pedicle flap: a controlled study. *British Journal of Plastic Surgery*, **34**, 446.

Griffiths, R. W., Hobby, J. A. E., Humphries, N. L. and Trengove-Jones, G. (1981). The influence of postoperative pharmacological vasodilator agents on the pattern of necrosis in a standardised rat skin flap. *British Journal of Plastic Surgery*, **34**, 441.

Henry, R. L., Nalbandian, R. M. and Dzandu, J. K. (1981). Effect on membrane-bound protein phosphorylation of intact normal and diabetic human erythrocytes: enhanced membrane deformability. *Diabetes*, **30**, 83A.

Hoopes, J. E. and Im, J. M. (1978). Skin flap necrosis in guinea pigs. *Plastic and Reconstructive Surgery*, **61**, 748.

Ishise, S., Pegram, B. L., Yamamoto, J., Kitamura, Y. and Frohlich, E. D. (1980). Reference sample microsphere method: cardiac output and blood flows in conscious rat. *American Journal of Physiology*, **239**, H443.

Jones, B. M., Dunscombe, P. B. and Greenhalgh, R. M. (1983). Differential thermometry as a monitor of blood flow in skin flaps. *British Journal of Plastic Surgery*, **36**, 83.

Kennedy, T. J., Pistone, G. and Miller, S. H. (1979). The effects of Reserpine on microcirculatory flow in rat flaps. *Plastic and Reconstructive Surgery*, **63**, 101.

Kunneke, P. S. and Malan, G. M. (1979). A controlled clinical trial on the effect of Piracetam in epileptic children. *British Journal of Clinical Practice*, **33**, 266.

Malik, A. B., Kaplan, J. E. and Saba, T. M. (1976). Reference sample method for cardiac output and regional blood flow determination in the rat. *Journal of Applied Physiology*, **40**, 472.

Mark, D. I., Wilsher, C. R., Blank, M. S. and Conners, C. K. (1985). The effects of Piracetam in children with dyslexia. *Journal of Clinical Psychopharmacology*, **5**, 272.

Marks, N. J., Trachy, R. E. and Cummings, C. W. (1984). Dynamic variations in blood flow as measured by laser Doppler velocimetry: a study in rat skin flaps. *Plastic and Reconstructive Surgery*, **73**, 804.

McDevitt, D. G. and Nies, A. S. (1976). Simultaneous measurement of cardiac output and its distribution with microspheres in the rat. *Cardiovascular Research*, **10**, 494.

Mindus, P., Cronholm, B., Levander, S. E. and Schalling, D. (1976). Piracetam-induced improvement of mental performance: a controlled study on normally aging individuals. *Acta psychiatrica scandinavica*, **54**, 150.

Myers, M. B. and Cherry, G. (1968). Causes of necrosis in pedicle flaps. *Plastic and Reconstructive Surgery*, **42**, 43.

Nalbandian, R. M., Henry, R. L., Burek, C. L., Diglio, C. A., Goldman, A. I., Taylor, G. W. and Hoffman, W. H. (1983). Diminished adherence of sickle erythrocytes to cultured vascular endothelium by Piracetam. *American Journal of Hematology*, **15**, 147.

Norton, K. J., Silverman, D. G. and Brousseau, D. A. (1984). Assessment of delay in bilateral thoraco-abdominal island flaps in the rat. *Archives of Otolaryngology*, **110**, 660.

Reinisch, J. F. (1974). The pathophysiology of skin flap circulation. *Plastic and Reconstructive Surgery*, **54**, 585.

Reuse-Blom, S. and Polderman, J. (1980) Influence of piracetam upon the pial micro-circulation. In *Gefass Patient Therapie*, edited by Loose, K. E. and Loose, D. A. Baden-Baden: Verlag Gerhard Witzstrock.

Sasaki, A. and Harii, K. (1980). Lack of effect of isoxsuprine on experimental random flaps in the rat. *Plastic and Reconstructive Surgery*, **66**, 105.

Sasaki, A. and Wagner, H. W. (1971). Measurement of the distribution of cardiac output in unanesthetized rats. *Journal of Applied Physiology*, **30**, 879.

Sonnet, J., Gini, E. K. and Cornu, G. (1985). Essai de prévention des crises vaso-occlusives de la drepanocytose homozygote par le iracetam. *Annales de la société Belge de Médecine Tropicale*, **65**, 77.

Wilsher, C., Atkins, G. and Manfield, P. (1979). Piracetam as an aid to learning in dyslexia. *Psychopharmacology*, **65**, 107.

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