visualization of platelets, flowing in mesenteric arterioles, was achieved by labeling the platelets in vivo with the fluorochrome acridine red. Using the peak intensity of their flashed fluorescence microscopic images as displayed on video the platelets could be localized objectively within a thin optical section. The median plane of a cylindric blood vessel represents its cross-sectional area because of rotational symmetry. Thus platelet concentration distribution, orientation and velocity profile were studied using only platelets located within a shallow section around this plane. Platelet concentration distribution was found to be non-uniform: the concentration decreased from the wall towards the vessel center. Branch points had a pronounced influence on the concentration distribution. In vessel segments without nearby branch points the average concentration near the wall was about twice that in the center. Platelet orientation is not random: they tend to align themselves with their equatorial plane parallel to the wall. The tendency to alignment increased from the vessel center towards the wall. In addition, evidence was found for tumbling of the platelets. In straight vessel segments platelet velocity profiles had the shape of a flattened parabola, both in systole and diastole, with maximum velocities ranging from 1 to 14 mm/sec.

Response of vertebral and carotid blood flows to isocapnic hypoxia

J.H.G.M. van Beek, A. Berkenbosch, J. de Goede and C.N. Olievier
Department of Physiology, University of Leiden, Leiden, The Netherlands

One of the mechanisms proposed to explain the depression of ventilation by central hypoxia is a change in cerebral blood flow. We therefore measured blood flows in one carotid and one vertebral artery of cats, anethetized with chloralose-urethane, using perivascular electromagnetic flow sensors. During hyperoxia ($PETO_2$ 55 kPa) the mean carotid and vertebral blood flows were 20 and 2 $ml \cdot min^{-1}$, respectively. The percentage increase in flows upon lowering of the $PETO_2$ are summarized in the table. The time course of the blood flows following step-like changes in $PETO_2$ was analyzed with a first order model. For the carotid blood flow the mean time constant for steps into hypoxia was 25 sec and for steps out of hypoxia 18 sec. The corresponding values for the vertebral blood flow were 38 sec and 15 sec. The observed changes in blood flow can explain a major part of the ventilatory depression by central hypoxia.

$PETO_2$	14 kPa	9 kPa	6.5 kPa	4.1 kPa
Carotid flow	4%*	17%*	28%*	74%*
Vertebral flow	3%	12%*	39%*	76%*

* Significantly different from hyperoxic value ($p < 0.05$).

Pulmonary extraction of dopamine in the conscious dog

F.W. van Schaik, G.M. van Heeswijk*, J.M. den Hertog** and G.H. Huisman***
** Medical Physiology Department, Utrecht; and ** Veterinary Physiology Department, Utrecht, The Netherlands*

The pulmonary handling of dopamine was compared to that of adrenaline and noradrenaline in the conscious dog in order to study the intrapulmonary significance of the metabolism for the perfusion. 15 nmol of each catecholamine was injected into the inferior caval vein together with cardiogreen as intravascular indicator. Dilution curves of each substance were derived from plasma samples withdrawn from the pulmonary artery and the aorta. Adrenaline was extracted for 3% which is comparable to the accuracy of the assay. The extraction of noradrenaline and dopamine amounted resp. 13% and 34%. The extraction of dopamine was remarkable, the more so as dopamine is assumed in literature to be unaffected in the pulmonary circulation just as adrenaline. The extraction has to be ascribed to selective uptake of dopamine and noradrenaline into the endothelial cells. Increased permeability of the capillaries can be excluded as the mechanism of removal from the pulmonary circulation, since each catecholamine appears in the aorta at the same time as cardiogreen. Inactivation within the vascular lumen can also be excluded. Circulating enzymes would metabolize adrenaline similarly as dopamine. A transient change in perfusion of the lung by shunting can be provoked by injection of adrenaline and noradrenaline, which is accompanied by a momentaneous decrease of p_{aO_2}. Probably a similar effect of dopamine can be attenuated locally by pulmonary metabolism (F.W. van Schaik, G.M. van Heeswijk, J.M. den Hertog and G.H. Huisman, Pulmonary extraction of dopamine in the conscious dog, Arch. int. Physiol. Biochim. *91* (1983) 215–222).

Skeletal muscle flow during endotoxin shock

A.A. van Lambalgen, W. Bronsveld, G.C. van den Bos and L.G. Thijs
Department of Physiology, Free University, Amsterdam, The Netherlands

Endotoxin causes redistribution of the cardiac output. Since a large part of body mass is skeletal muscle ($\pm$ 40%) changes in its perfusion could have marked effects on perfusion of other organs. We have studied the effects of endotoxin (*E. coli* endotoxin, 1.5 $mg \cdot kg^{-1}$) on muscle blood flow in forelimb, thorax, diaphragm and hindlimb (5 different muscles) and on blood flow in skin. We have used radioactive microspheres in 6 control and 6 endotoxin treated dogs before saline or endotoxin (at $t = 0$) and at $t = 90$ and 120 min. Flows in femoral artery and vein were also measured (electromagnetic flowtransducer). Immediately after endotoxin cardiac output, mean arterial pressure and flow in femoral artery and vein fell markedly followed by partial recovery at $t = 30$. Subsequently hemodynamic variables gradually deteriorated but flow in femoral artery and vein did not. The ratio of flow in femoral artery and vein decreased (by 63% at $t = 120$; $p < 0.05$). Apart from the diaphragm, flow to skeletal muscle did not change or increased after endotoxin but decreased in the control group. Percentage of the cardiac output to brachial, intercostal and hindlimb muscle and skin increased after endotoxin (by 163, 167, 111 and 120% at $t = 120$, respectively; $p < 0.05$). The 5 muscles of the hindlimb did not respond differently to endotoxin. In spite of diminished arterial flow (from 60 to 40 $ml \cdot min^{-1}$, at $t = 120$), skeletal muscle flow was thus maintained in the hindlimb probably due to closing of shunts and redistribution of blood away from bone.

Generation and propagation of epileptiform activity in the hippocampal slice preparation

J. Holsheimer, F.H. Lopes da Silva and A.J. Verloop
Twente University of Technology, Enschede, and University of Amsterdam, Amsterdam, The Netherlands

For the investigation of epileptiform events in the hippocampal CA1 field, in-vitro slices of the guinea-pig were used. After adding 0.1 mmol 4-aminopyridine to the bathing medium, field potentials were recorded with an electrode array, consisting of 8 semi-microelectrodes at spacings of 0.1 mm. A comparison was made between the spontaneously occurring field potentials (SFP) in CA1 and those evoked by different inputs to the CA1 pyramidal cells, namely alveus, str. oriens and Schaffer collaterals. For this purpose the electrode array was placed in CA1, parallel to the axes for the pyramidal cells. The regularly occurring SEP's presented a similar distribution as the potentials evoked by stimulation of str. oriens or alveus of CA1, but differed from those evoked by stimulation of the Schaffer colaterals. This indicates that in CA1 SFP's are generated in a similar way as field potentials evoked by alveus or str. oriens stimulation. It was also found that SFP's are propagated from CA3 and CA1 at a velocity of 0.16–0.30 m/sec. Therefore pathways in alveus and str. oriens, connecting CA3 and CA1, may be important in propagating epileptiform activity. This was supported by experiments in which different pathways were sectioned.