COWIE, A. T. (1966) in *The Pituitary Gland* (HARRIS, G. W. & DONOVAN, B. T. eds), vol. 2, pp. 412-443. Butterworths, London.
COWIE, A. T. (1969) in *Lactogenesis* (REYNOLDS, M. & FOLLEY, S. J., eds) pp. 157-169. University of Pennsylvania Press, Philadelphia.
DENAMUR, R. (1971) *J. Dairy Res.* **38**, 237-264.
FORSYTH, I. A. & HART, I. C. (1976) *Proc. 3rd Int. Symp. Growth Hormone and Related Peptides.* Excerpta Medica, Amsterdam.
GUNTHER, M. & STANIER, J. E. (1951) in *Spec. Rep. Serv. Med. Res. Coun., London*, No. 275, pp. 379-400.
HART, I. C. (1975) *J. Endocrinol.* **64**, 305-312 & 313-322.
HART, I. C., BINES, J. A., BALCH, C. C. & COWIE, A. T. (1975) *Life Sci.* **16**, 1285-1292.
HART, I. C. & FLUX, D. S. (1973) *J. Endocrinol.* **57**, 177-178.
HART, I. C. & LINZELL, J. L. (1976) *J. Endocrinol.* (Submitted).
HUTTON, J. B. (1957) *J. Endocrinol.* **16**, 115-125.
HYTTEN, F. E. & THOMSON, A. M. (1961) in *Milk : The Mammary Gland and its Secretion* (KON, S. K. & COWIE, A. T., eds) vol. 2, chap. 13. Academic Press, New York & London.
MACHLIN, L. J. (1973) *J. Dairy Sci.* **56**, 575-580.
MARTAL, J. (1955) *C. R. Hebd. Séances Acad. Sci. Sér. D Sci. natur. (Paris)* **280**, 197-200.
REYNAERT, R., DE PAEPE, M., MARCUS, S. & PEETERS, G. (1975) *J. Endocrinol.* **66**, 213-224.
REYNAERT, R., DE PAEPE, M. & PEETERS, G. (1972) *Ann. Endocrinol.* **33**, 541-543.
REYNAERT, R. & PEETERS, G. (1972) *Arch. internat. Pharmacodyn. Thér.* **197**, 207-208.
SELYE, H. (1934) *Amer. J. Physiol.* **107**, 535-538.
TUCKER, H. A. (1971) *J. Anim. Sci.* **32**, 137-141.

B. — COMMUNICATIONS

G. DHONDT and G. PEETERS *(Department of Physiology, Veterinary Faculty, University of Ghent, Casinoplein, 24, 9000 Ghent).*

Influence of acute experimental mastitis on mammary blood flow in lactating ruminants.

It is well known that endotoxin produces the clinical signs of acute mastitis when introduced into a mammary quarter of ruminants (SCHALM *et al.*, 1972). We have studied the effect of this treatment on blood flow through the udder of goats and cows.

Methods.

Nineteen experiments were performed on 8 goats yielding 0.4 to 1.4 litres of milk per day. Six experiments were carried out on 2 cows yielding 10 and 16 litres of milk daily. An electromagnetic

flow-probe and a pneumatic arterial occlusion cuff were chronically implanted around the mammary artery (external pudic artery) of one udderhalf (HOUVENAGHEL & PEETERS, 1972). For flow measurement, the flowprobe was connected to a square wave flowmeter. The phasic flow and its integrated value were recorded on a Mingograph 800. All registrations were performed on conscious animals which were well accustomed to the experimental procedures. Bloodflow was expressed in absolute integrated values of the flow signal/5 sec. Each experiment lasted 5 days. Acute mastitis was induced by administration of 1 mg *Escherichia coli* endotoxin (lipopolysaccharide 0.26 : B6 from Difco Lab., Detroit, U. S. A.) by way of the teat canal into one udderhalf (goats) or two quarters (cows) homolaterally to the flow probe. Mammary blood flow was measured 6 times per day during 2 days before and 2 days after treatment. Endotoxin was administered in the morning of the third day and afterwards bloodflow was measured at 30- or 90-min intervals over a 14-h period.

Results.

Goats. — After the administration of endotoxin, bloodflow started to increase quickly and reached a peak at the 2nd hour. At that time, the average bloodflow was 100 % above control values. Around the 4th or 5th hour the flow returned to normal. Then it rose again and reached a second peak around the 9th or 10th hour. The flow returned to control levels around the 13th or 14th hour.

Cows. — Administration of endotoxin induced a strong and biphasic increase in mammary bloodflow. At peak 1, which occurred during the 3rd hour, bloodflow was 4 times higher than the control level. During the 6th hour, blood flow had returned to normal. Then it started to increase again, reaching peak 2 between the 10th and 11th hours. After the administration of endotoxin, the flow returned to the control level between the 13th and 14th hours.

In both species, body temperature rose to a peak value by the time the bloodflow had temporarily returned to normal between peak 1 and peak 2. During the 1st peak, there was extreme swelling of the gland. The chloride concentration in milk reached peak values during peak 2 or a little earlier.

This study was supported by a grant from the Belgian *I.R.S.I.A.* Foundation.

REFERENCES

HOUVENAGHEL, A. & PEETERS, G. (1972) *Arch. internat. Pharmacodyn. Ther.* **200**, 320-329.
SCHALM, O. W., CARROLL, E. J. & JAIN, N. C. (1971) in *Bovine Mastitis*. Lea & Febiger, Philadelphia.

J. J. LEGROS (1), G. PEETERS, S. MARCUS, W. DE GROOT and R. REYNAERT ((1) *Radioimmunoassay Laboratory, University of Liège* and *Department of Physiology, Veterinary Faculty, University of Ghent).*

Independent release of neurophysin I and II during and following parturition in cows.

Neurophysins are the carriers of active neurohypophysial hormones, vasopressin and oxytocin; many authors have been able to demonstrate a concomitant release of neurophysins and hormones in various experimental conditions. Previously we have demonstrated a release of BN_pI without a release of BN_pII during milking or suckling in the cow (LEGROS *et al.*, 1974) and a specific release of BN_pII during venous or arterial haemorrhage (LEGROS *et al.*, 1975). In the present work we investigated BN_pI and BN_pII serum levels before, during and after parturition in cows.

Methods.

Jugular blood samples were collected through a previously inserted catheter during different stages of labour in 17 cows. Prepartum, first stage, second stage and postpartum bloodsamples were collected from 9, 13, 17 and 17 animals respectively. Radioimmunoassays for separate neurophysins were made on each sample as described previously (LEGROS & FRANCHIMONT, 1974). The total number of samples per animal varied from 7 to 46.