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Original article

Electromyographic activity and noradrenaline content of the rabbit oviduct under different hormonal states

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Summary — Spontaneous electromyographic (EMG) activity of the oviduct recorded *in vivo* in untreated, estrogen-treated, and progesterone-treated castrated rabbits was found to exhibit two main patterns : short spike bursts lasting 1—10 s and long trains of action potentials lasting several minutes, which constituted the major component of EMG activity.

After estrogen treatment, both wet weight and noradrenaline (NA) content of the castrated rabbit oviduct were enhanced mainly at the ampullary—isthmic junction; long trains of discharges were significantly shorter (2.0—2.7 min *vs* 3.6—4.6 min) and appeared at more frequent intervals (9.8—12.2 min *vs* 14.2—22.6 min). After progesterone treatment, spontaneous EMG activity was not significantly different from that in untreated castrated rabbits (as was the NA content) except at the ampullary—isthmic junction.

NA injection elicited a stimulatory response of the oviduct lasting 1—7 min in the three hormonal states. Phentolamine strongly depressed spontaneous EMG activity but the inhibition was more transient in castrated rabbits than in estrogen-treated and progesterone-treated animals. Propranolol had no effect on spontaneous EMG activity.

These data and the high NA concentrations found in all parts of the isthmus support the hypothesis that adrenergic innervation plays a role in the organization of oviductal motility in the rabbit.

EMG activity — sexual steroids — noradrenaline — oviduct — rabbit

Résumé — Activité électromyographique et teneur en noradrénaline de l'oviducte de lapine sous différentes imprégnations hormonales. *L'activité EMG spontanée de l'oviducte a été enregistrée* in vivo *chez des lapines castrées non traitées, traitées par les estrogènes et traitées par la progestérone. Elle se présente sous la forme de courtes salves de 1 à 10 sec de durée et de longues décharges de potentiels qui durent plusieurs minutes et constituent l'aspect le plus caractéristique de l'activité EMG. Après traitement par les estrogènes, le poids de l'oviducte et sa teneur en noradrénaline augmentent, surtout à la jonction isthme—ampoule; les salves de longue durée deviennent significativement plus courtes (2—2,7 min* vs *3,6—4,6 min) et apparaissent à des*

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intervalles plus rapprochés (9,8—12,2 min vs 14,2—22,6 min). Après traitement par la progestérone, l'activité EMG spontanée n'apparaît pas différente de celle enregistrée chez l'animal castré non traité, de même que la teneur de l'oviducte en noradrénaline, sauf pour la jonction isthme—ampoule. Une injection de noradrénaline provoque l'apparition d'une salve de potentiel de 1 à 7 min de durée dans les trois conditions hormonales. La phentolamine déprime fortement l'activité EMG spontanée, mais l'inhibition est plus transitoire chez les lapines castrées non traitées que chez les animaux traités par les estrogènes et par la progestérone. Le propranolol est sans effet sur l'activité EMG spontanée. Ces résultats, ainsi que les teneurs élevées en noradrénaline mesurées dans toutes les parties de l'isthme, montrent qu'il est possible que l'innervation adrénergique de l'oviducte joue un rôle dans l'organisation de la motricité spontanée de l'oviducte chez la lapine.

activité électromyographique — stéroïdes sexuels — noradrénaline — oviducte — lapine

Introduction

The oviduct of the rabbit has a rich adrenergic innervation; the nerve terminals are distributed primarily in the smooth muscle of the isthmus, with the isthmic portion having the greatest number of fluorescent nerve terminals that just below the ampullary—isthmic junction (Brundin, 1965; Owman and Sjöberg, 1966). These nerves are mainly composed of "short" adrenergic neurons, which differ morphologically and functionally from the ordinary "long" adrenergic neurons (Owman and Sjöberg, 1966; Sjöberg, 1967, 1968). One such difference is that noradrenaline (NA) metabolism is influenced by the ovarian steroids, *i.e.,* estrogen and progesterone (reviewed in Marshall, 1981).

The importance of adrenergic innervation, and the responsiveness of the oviductal smooth muscle to adrenergic drugs and nerve stimulation, have led to the hypothesis that nerves might play a role in the regulation of oviductal motility and perhaps in ovum transport (Brundin, 1965; Pauerstein *et al.,* 1974). Nevertheless, surgical or pharmacological denervation fails to alter significantly ovum transport and fertility (Paton *et al.,* 1978). These observations are not entirely conclusive, however, because denervation is always incomplete and its effects might be partially compensated for by an increase in postjunctional sensitivity to NA and by morphological changes within the muscle (Marshall, 1981; Westfall, 1981).

The present study was performed to investigate a possible correlation between the noradrenergic activity and the spontaneous electromyographic (EMG) activity of the rabbit oviduct under different ovarian steroid treatments. With this objective, the effects of adrenergic agonists and antagonists on EMG activity were studied and the noradrenaline content in the different parts of the oviduct was estimated.

Material and Methods

Experiment 1

Animals

Fauve de Bourgogne rabbits weighing 2.5—4.0 kg were used. They were bilaterally ovariectomized on d1 under general anaesthesia (40 mg sodium pentobarbital/kg IV). They received daily injections of estradiol benzoate (5 µg/kg IM) from d14 to d26 and daily injections of progesterone (1.5 mg/kg IM) from d27 to d40.

Experimental conditions

Electromyographic (EMG) activity was recorded *in vivo* in 16 rabbits. Recordings were made every morning for about 3 h from d6 to d15 in

untreated ovariectomized rabbits, from d21 to d27 in estrogen-treated rabbits, and from d34 to d40 in progesterone-treated animals. Effects of catecholamines and of their antagonists were studied separately in the three hormonal states. Drugs were injected in the marginal ear vein at the following doses : adrenaline (adrenaline, Meram), 5 µg/kg; noradrenaline (Levophed, Winthrop), 5 µg/kg; phentolamine (Regitine, Ciba—Geigy), 0.3 mg/kg; propranolol (Avlocardyl, ICI Pharma), 1.5 mg/kg.

EMG recordings

Bipolar electrodes were implanted during laparotomy performed for bilateral ovariectomy. They were made from insulated nickel—chrome wires with a diameter of 80 µm (Stabilhom 133; Johnson Matthey Ltd., London) coated together with medical silicone (Rhodorsil-Silicones), with the insulation of the tip being burnt before use. They were inserted in pairs 2—3 mm apart, through the muscular layer of the oviduct, with a hypodermic needle used as a trocar. The electrodes were externalized through the abdominal wall and tied to the skin. Five bipolar electrodes were inserted: on the ampulla between the ampullary—isthmic junction (AIJ) and the fimbriae; on the AIJ; on the isthmus between the AIJ and the uterotubal junction (UTJ); on the UTJ; and on the uterus at 2 cm from the UTJ. Electrical activity was recorded with a 6-channel polygraph (Reega VIII, Alvar, Paris) with a time constant of 0.1 sec.

Analysis of records

EMG activity was described by the frequency and duration of spike bursts. In addition, the amount of electrical activity recorded during a session was calculated from the myoelectrical index (MI) of Krishnamurti *et al.* (1982) : MI = $d_1/T \times 100$, d_1 being the sum of the durations of the bursts recorded during a session of T seconds.

MI, frequency, and duration of spike bursts were calculated throughout each record. For statistical analysis, each animal was used as its own control, differences between electrodes and between hormonal states were tested with paired data by means of Wilcoxon's matched-pairs signed-rank test.

Experiment 2

Animals

Fifteen ovariectomized rabbits weighing 2—3 kg were used in the experiment. Animals were caged in pairs under controlled light (12:12, L—D). They received water and food *ad libitum*.

Experimental conditions

Three groups of 5 animals were used:

Group 1 : animals were ovariectomized, then kept at rest and sacrificed 1 wk later;

Group 2 : after ovariectomy, animals were kept without treatment for 1 wk, then injected with a daily dose of 5 µg/kg IM of estradiol benzoate (E_2) during a 2nd wk, and sacrificed 15 d post-ovariectomy;

Group 3 : 5 rabbits treated like the group E_2 animals were injected with a daily dose of 1.5 mg/kg IM of progesterone (P) during a 3rd wk and sacrificed 21 d post-ovariectomy.

Preparation of oviductal portions

Each oviduct was excised and placed in an ice-cooled dish and rapidly dissected free of adipose and connective tissue.

The oviduct was divided into two parts. The first part near the ovary was the infundibulum—ampullar region (IA). The second part was divided into three portions of equal length : the ampullary—isthmic junction (AIJ); the median isthmus (Ith); and the uterotubal junction (UTJ) near the uterus.

The four parts were frozen in isopentane (–160 °C). After rapid freezing (15 sec), the parts were then stored in a refrigerator (–20 °C) until biochemical assay.

Biochemical analysis

The content of noradrenaline (NA) was estimated in each portion of the oviduct using high—performance liquid chromatography (HPLC) with electrochemical detection, as previously described (Bernet *et al.*, 1987). The HPLC apparatus was a Beckman model equipped with a model 112 pump and a 5 µ ultrasphere ODS analytical column. The mobile phase was the solvent system used by Mefford (1981) for separation of catecholamines. It contained 0.1 M sodium acetate, 0.02 M citric acid, 100 mg/l sodium octylsulfate, 50 mg/l ethylene diaminetetraacetic acid (EDTA), and 12% methanol (vol/vol). The NA was oxidized at + 0.60 V relative to an Ag/AgCl reference electrode.

Group means were compared by using Student's *t*-test.

Results

In vivo *recording of EMG activity*

Untreated ovariectomized rabbits

The electrical activity of the rabbit oviduct was found to exhibit two main patterns: short spike bursts lasting 1—10 sec and long spike bursts occurring for periods of several minutes (Fig. 1). The amplitude of spikes was low in the two groups: 50—100 μV, with maxima of 300 μV. Histograms of the durations (Fig. 2) show that short spike bursts were the most numerous. They represented 74.1% to 78.7% of all the electrical events, but their specific MI was only 2—3%, due to their short mean duration (3.0—3.5 sec). Their mean frequency ranged from 20.7 to 29.5/h in the four parts of the oviduct (Table I).

In contrast, the MI of long spike bursts ranged from 14.2% at the AIJ to 30.2% in

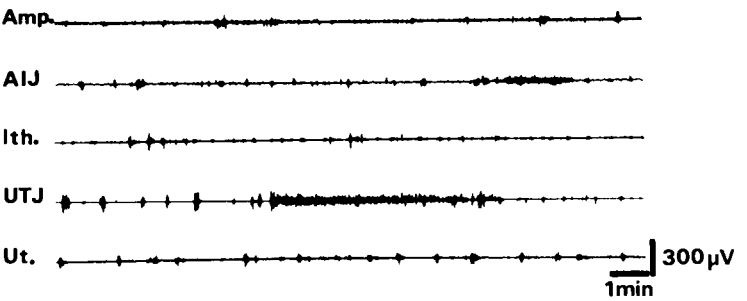

Fig. 1. Electromyographic activity of oviduct and uterus in untreated ovariectomized rabbits recorded with electrodes located in ampulla **(Amp)**, ampullary—isthmic junction **(AIJ)**, median isthmus **(Ith)**, uterotubal junction **(UTJ)**, and uterine horn, 2 cm from uterotubal junction **(Ut)**.

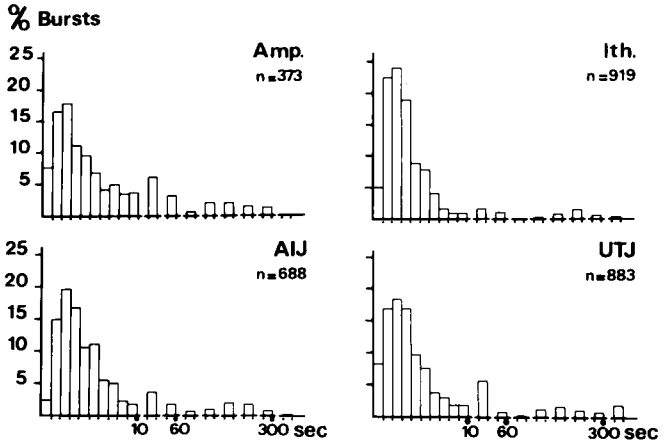

Fig. 2. Histograms of spike burst duration in untreated ovariectomized rabbits. Abbreviations: see Fig. 1.

Table I. Duration and frequency of short and long electric discharges at various portions of the oviduct in untreated ovariectomized rabbits, 8 days after ovariectomy.

		Ampulla	*AIJ*	*Isthmus*	*UTJ*
Short spike bursts	Myoelectrical index (%)	2.2 ± 0.6	2.0 ± 0.5	2.7 ± 0.7	2.2 ± 0.5
	Duration (sec)	3.5 ± 0.3	3.0 ± 0.2	3.3 ± 0.2	3.3 ± 0.2
	Frequency (per hour)	20.9 ± 8.8	20.7 ± 8.9	29.5 ± 6.6	24.9 ± 6.0
Long spike bursts	Myoelectrical index (%)	16.3 ± 3.6	14.2 ± 3.5	30.2 ± 4.6	25.9 ± 4.3
	Duration (min)	3.6 ± 0.5	3.9 ± 0.3	4.6 ± 0.7	3.8 ± 0.5
	Frequency (per hour)	3.7 ± 0.2	2.7 ± 0.3	3.7 ± 0.5	4.2 ± 0.4

Mean values ± SEM for 16 animals.
AIJ : ampullary—isthmic junction; UTJ : uterotubal junction.

the isthmus, compared to the respective values of overall MI (16.8% and 35.2%). It was clear that spike bursts of long duration constituted the major component of EMG activity. Long bursts generally consisted of trains of potential firing at a frequency of 2/sec, more rarely of spike bursts lasting 10—12 sec and recurring at short intervals during several minutes. They lasted 3.6—4.6 min and occurred 2.7—4.2 times every hour, depending on the different regions (Table I).

Effects of estrogen and progesterone

Overall, the MI was not modified by estrogen or progesterone treatment: it ranged from 19.7% to 36.0% according to the tubal region and the hormonal state. After estrogen treatment, the duration and frequency of short spike bursts remained unaffected, but characteristics of long spike bursts were clearly modified. As shown in Fig. 3 and Table II, long trains of discharges were significantly shorter after

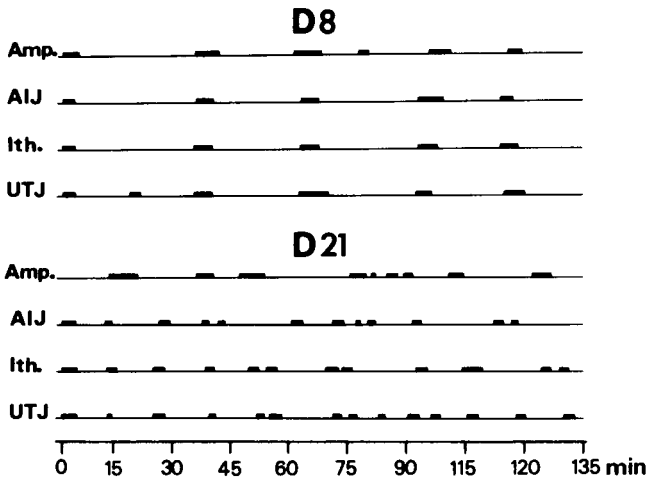

Fig. 3. Pattern of long duration spike bursts in the same ovariectomized rabbit before (d8) and after (d21) estrogen treatment. Spike bursts are more frequent but shorter under estrogen domination. *Abbreviations*: see Fig. 1.

Table II. Duration of long spike bursts and intervals between them (mean ± SEM).

		Ampulla	*AIJ*	*Isthmus*	*UTJ*
Spike burst duration (min)	C $N = 16$	3.6 ± 0.5	3.9 ± 0.3 (a)	4.6 ± 0.7 (a)	3.8 ± 0.5 (a)
	E_2 $N = 11$	2.7 ± 0.6	2.0 ± 0.2 (a) (b)	2.8 ± 0.4 (a) (b)	2.7 ± 0.5 (a) (b)
	P $N = 7$	3.5 ± 0.8	3.4 ± 0.6 (b)	4.1 ± 0.8 (b)	4.1 ± 0.6 (b)
Intervals between spike bursts (min)	C $N = 16$	16.2 ± 1.1 (a)	22.6 ± 2.5 (a)	16.2 ± 2.2 (a)	14.2 ± 1.4 (a)
	E_2 $N = 11$	12.2 ± 1.7 (a) (b)	12.0 ± 1.5 (a) (b)	10.0 ± 0.8 (a) (b)	9.8 ± 1.4 (a) (b)
	P $N = 7$	14.3 ± 4.1 (b)	19.6 ± 2.5 (b)	17.4 ± 3.1 (b)	19.3 ± 1.8 (b)

C = untreated ovariectomized rabbits (day 8 after ovariectomy).
E_2 = estrogen-treated ovariectomized rabbits (day 21 after ovariectomy).
P = progesterone-treated ovariectomized rabbits (day 34 after ovariectomy).
AIJ = ampullary—isthmic junction; UTJ : uterotubal junction.
N = number of animals.
(a) Significant difference between estrogen-treated and untreated ovariectomized rabbits ($P \leq 0.05$).
(b) Significant difference between estrogen-treated and progesterone treated rabbits ($P \leq 0.05$).

estrogen treatment (2.0—2.7 min on day 21 *vs* 3.6—4.6 min on day 8; $P < 0.05$) and they were separated by intervals lasting 9.8—12.2 min instead of 14.2—22.6 min in untreated ovariectomized animals ($P < 0.05$).

After progesterone treatment, parameters of short and long spike bursts measured on d34 were not significantly different from those observed on d8 in untreated ovariectomized does.

Effects of ovarian steroids on the propagation of EMG activity

The criterion of propagation of EMG activity was the successive appearance of bursts in two or more neighbouring electrodes; latencies between sequential bursts were generally > 1.5 sec, implying an apparent conduction velocity of 4—13 mm/sec. As shown in Table III, only 36% of short spike bursts were propagated in untreated castrated rabbits and the distance of spread was short. Estrogen induced an increase in the number of propagated spike bursts (54%) and in the distance of spread, but progesterone had no effect. By contrast, the percent propagated bursts of long duration was high (≈ 80%) and did not depend on the hormonal state. Whatever the type of burst, about 50% originated in the median isthmus, 25% at the UTJ, and 25% at the AIJ or (more rarely) in the ampulla. In untreated and progesterone-treated castrated animals, 70% of the short spike bursts originating from the AIJ propagated

Table III. Percentage of short and long spike bursts in the oviduct.

	Short spike bursts						*Long spike bursts*					
	Number of spike bursts	*Percentage of short spike bursts*					*Number of spike bursts*	*Percentage of long spike bursts*				
		Unpropagated	*Propagated*					*Unpropagated*	*Propagated*			
			2el.	*under 3el.*	*4el.*	*Total amount*			*2el.*	*under 3el.*	*4el.*	*Total amount*
C *N* = 6	715	64,0	21.6	9.0	5.4	36.0	16	19.0	49.4	25.1	6.5	81.0
E_2 *N* = 7	545	46,0	24.3	20.0	9.7	54.0	57	21.0	14.2	30.0	34,8	79.0
P *N* = 5	462	64,0	23.4	6.1	6.5	36.0	21	19.0	28.3	28.4	24.3	81.0

In untreated ovariectomized rabbits : C; estrogen-treated ovariectomized rabbits : E_2; progesterone-treated ovariectomized rabbits : P.
N = number of animals.

towards the UTJ. Estrogen modified this proportion: only 38% of the spike bursts appearing first at the AIJ were descending, 35% were ascending, and 27% propagated in the two directions (Fig. 4).

In the three hormonal states, long spike bursts propagated towards the uterine end of the oviduct as well as in the opposite direction. However, their distance of spread was enhanced by steroid hormones: whereas 6.5% of the recorded bursts propagated through the entire oviduct in castrated animals, the proportion reached 24.3% after progesterone treatment and 34.8% after estrogen treatment.

Effect of catecholamines and adrenergic blocking agents

Adrenaline and noradrenaline elicited a stimulatory response of the oviduct in the three hormonal states. Trains of potentials 50—250 μV in amplitude appeared simultaneously in the four parts of the oviduct within 5 sec after injection of 5 μg/kg and lasted for 1—7 min (Fig. 5). The large variability observed in EMG burst duration did not allow us to detect any significant difference between the three hormonal states. However, EMG activity recorded at the AIJ and in the isthmus presented a higher mean amplitude after estrogen treatment (200 μV *vs* 100 μV; $P < 0.05$).

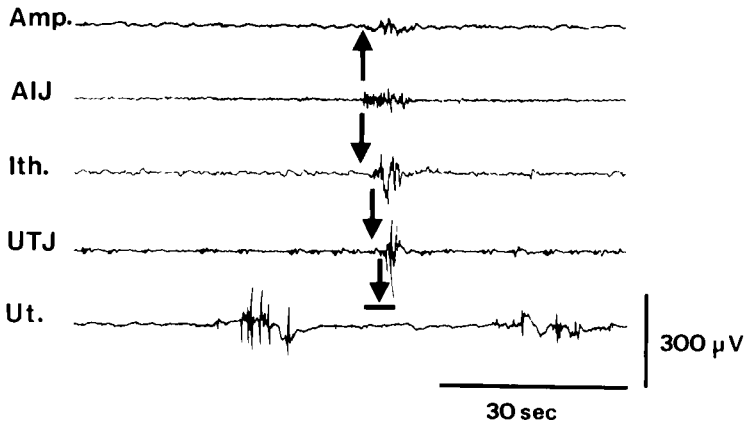

Fig. 4. Propagation of EMG activity in estogren-treated ovariectomized rabbit. As shown by arrows, a short spike burst originates at the ampullary—isthmic junction **(AIJ)**, propagates towards the ampulla **(Amp)** and towards the median isthmus **(Ith)**, and stops at the uterotubal junction **(UTJ)**. The EMG activity of the uterine horn **(Ut)** is not correlated with the tubal activity.

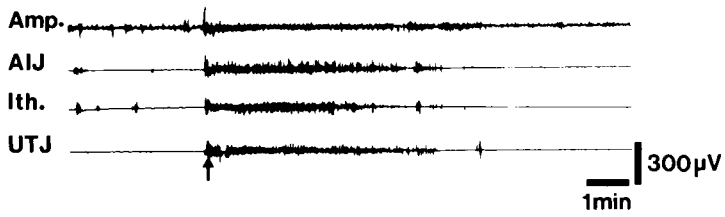

Fig. 5. Activation of oviductal EMG activity after a single IV injection of 5 μg/kg of noradrenaline (arrow). *Abbreviations* : see Fig. 1.

To test the effects of adrenergic blocking agents, the chosen parameter was the MI calculated for each period of 10 min preceding and following the injection. Phentolamine strongly depressed spontaneous EMG activity in all the regions of the oviduct in the three hormonal states (Fig. 6). Both types of spike bursts were equally inhibited. The inhibition was more transient in castrated rabbits than in estrogen-treated and progesterone-treated animals in which the electrical activity remained significantly lowered ($P < 0.05$); 90 and 70 min, respectively, instead of 20 min in untreated castrates.

Unlike phentolamine, propranolol had no effect on spontaneous EMG activity.

Noradrenaline levels in the oviduct

Weight variations of the oviducts

Estrogen treatment significantly increased the weight of the oviducts: 271 ± 45 mg (mean ± SD) in the estrogen-treated rabbits *versus* 125 ± 12 mg in the

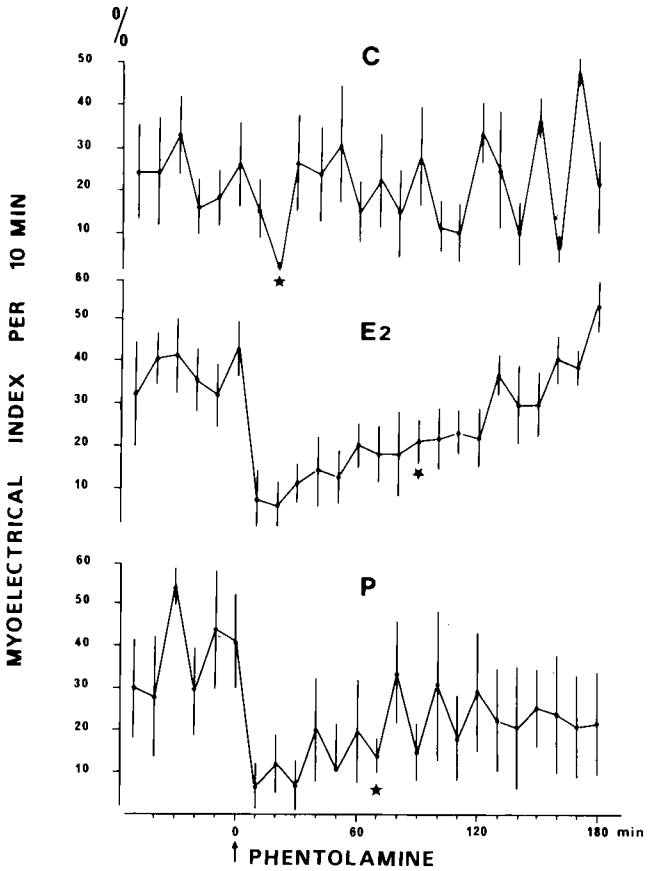

Fig. 6. Effects of phentolamine on MI (for definition see Materials and Methods) of the median isthmus in untreated **(C)**, estrogen-treated **(E_2)**, and progesterone-treated **(P)** castrated rabbits. Injection of phentolamine is followed by a depression of electrical activity which remains significantly lowered ($P < 0.05$) for 20 min in untreated, 90 min in estrogen-treated, and 70 min in progesterone-treated castrates (stars). The mean values are indicated with the SEM.

ovariectomized rabbits ($t = 3.13$, $P < 0.01$) and *versus* 149 ± 9 mg in the progesterone-treated rabbits ($t = 2.67$, $P < 0.02$). No significant difference was observed between the weights of the oviducts in the untreated ovariectomized rabbits and the progesterone-treated rabbits.

The weights of each oviductal portion in the three experimental conditions are shown in Fig. 7. The portions of the oviduct in the E_2 rabbits were heavier than the corresponding portions in the untreated ovariectomized rabbits and than the AIJ and MI portions in the progesterone-treated rabbits.

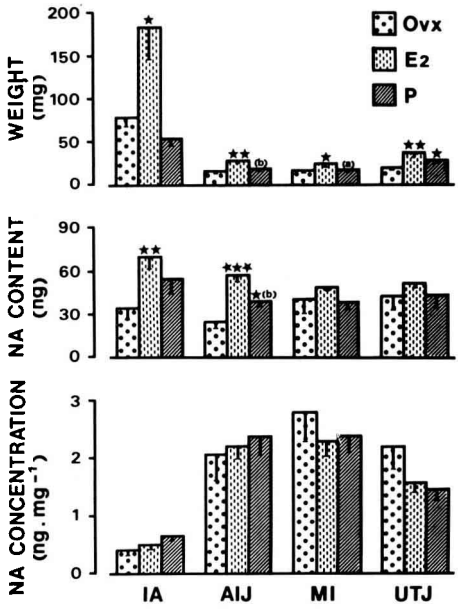

Fig. 7. Weight of the oviductal portions, noradrenaline (NA) content, and NA concentration in the different parts of the oviduct in the untreated ovariectomized rabbits **(Ovx)**, in the estrogen-treated rabbits **(E_2)**, and in the progesterone-treated rabbits **(P)**. The mean values are indicated with the SEM. Compared with Ovx: *, $P < 0.05$; **, $P < 0.02$, ***, $P < 0.01$. Compared with E_2: (a) $P < 0.05$; (b) $P < 0.02$.

NA content

The oviducts of estrogen-treated rabbits contained significantly more NA than the oviducts of the other two groups: 227 ± 15 ng (mean ± SD) *versus* 142 ± 29 ng in the untreated castrated rabbits ($t = 2.62$, $P < 0.02$), and *versus* 175 ± 15 ng in the progesterone-treated rabbits ($t = 2.44$, $P < 0.05$). A significant difference was also observed between the NA content of the oviducts in the untreated rabbits and in the progesterone-treated rabbits.

The content of NA in nanograms per portion of oviduct (Fig. 5) was significantly higher in estrogen-treated rabbits than that of the IA and AIJ portions in untreated and progesterone-treated rabbits. The NA content of AIJ in the progesterone-treated rabbits was higher than that of the same portion of oviduct in the ovariectomized rabbits.

NA concentration

The fact of bringing the NA content down to nanograms per milligram wet tissue abolished the differences between the rabbits of the three experimental groups.

Discussion

Electrical activity of the rabbit oviduct clearly shows two components: short spike bursts and long trains of discharges lasting several minutes. *In vivo* studies of oviductal pressure changes have shown a similar pattern of small contractions interspersed with outbursts of increased activity in rabbits, monkeys, and humans (De Mattos and Coutinho, 1971; Coutinho *et al.*, 1975; Fredericks *et al.*, 1982). It is generally believed that short bursts originate in the circular muscle layer, and long trains of spikes in the longitudinal

muscles which cover the oviduct dorsally and ventrally and are continuous with the muscles of the mesotubarium superius and mesosalpinx (Talo and Brundin, 1973; Gonzalez de Vargas *et al.*, 1976). No direct connection between the oviduct circular muscle and the longitudinal peritoneal muscle was observed, with the two layers of muscle always being separated by a distinct layer of connective tissue. Nevertheless, cells with morphological characteristics of ganglion cells were seen in the connective tissue layer (Black *et al.*, 1980). This may provide a neural mechanism for muscular coordination between the circular layer and the longitudinal peritoneal muscle. It is likely that the long trains of discharges originated in the longitudinal muscle or in the peritoneal membranes (Talo and Brundin, 1973; Meiri *et al.*, 1978), but the possibility that excitation spread between the two muscle layers cannot be precluded.

In accordance with other reports, we have found that both adrenaline and NA have an excitatory effect on the oviductal EMG activity. This response is mediated by α-excitatory adrenoceptors (Levy and Lindner, 1972; Doteuchi and Takeda, 1978). Inhibitory β-adrenoceptors were also found, but they usually seem to be masked by the α-adrenoceptor under normal conditions, at least in the rabbit oviduct (Johns and Paton, 1975).

In vitro, the spontaneous EMG activity of the oviduct is unaffected by phentolamine or by tetrodotoxin and is therefore presumably myogenic (Meiri *et al.*, 1978). However, myogenic activity might be modulated *in vivo* by changes in neuronal activity. This hypothesis was investigated by *in vivo* recording of oviductal activity in rabbits injected with phentolamine and propranolol. Propranolol, a β-adrenoceptor antagonist, had no effect on EMG activity, which is consistent with the findings of Johns and Paton (1975) showing that the rabbit oviduct is always α-dominant.

Phentolamine, an α-adrenoceptor antagonist, produced a rapid and marked fall in electrical activity. Such inhibition of spontaneous activity after phentolamine has been reported in the uterus of rhesus monkeys (Harbert and Spisso, 1981) and rats (Legrand and Maltier, 1986). Overstreet and Tom (1982) have shown that the rapid phase of sperm transport from vagina to ovary occurring immediately after mating is effectively blocked with the α-adrenergic antagonist phenoxybenzamine in the rabbit. These data and the high NA concentration found in all parts of the isthmus support the hypothesis that adrenergic innervation may play a role in the organization of tubal motricity.

It is generally accepted that estrogen increases tubal motility and that progesterone or ovariectomy has an inhibitory effect (Spilman and Harper, 1974; Borda *et al.*, 1975; Ruckebusch and Bayard, 1975; Gimeno *et al.*, 1976). Comparisons of the MI in our experiments show that the amount of time spent in contractile activity did not change in the three hormonal conditions. Although the MI of longlasting bursts was not affected, their duration and the intervals between them were shortened. The major effect of estrogen was an increase in the rate of propagated spike bursts of short duration and of their distance of spread. As the percentage of propagated long spike bursts was already high ($\approx$80%) in untreated ovariectomized rabbits, estrogen treatment did not modify this proportion and enhanced only the distance of spread.

The oviductal NA concentration remained unchanged in the three hormonal states despite large variations

of tissue weight. These results show that estradiol enhances both wet weight and NA content. Actually, Meiri *et al.* (1978) have shown that changes in muscle size can be dissociated from changes in transmitter content in the adrenergic nerves of the rabbit oviduct; it is possible to prevent muscle atrophy after ovariectomy without preventing the decrease in NA content, indicating that the adrenergic nerves constitute a target system for the ovarian steroids separated from the oviductal muscle cells (Marshall, 1981). It is noticeable that the increase in NA content was most pronounced at the AIJ: 134% *versus* 101% in the ampulla. The AIJ is known for the high density of its adrenergic innervation (Brundin, 1965). The unchanged NA concentration does not exclude mediation by noradrenergic systems in the effects of estradiol on tubal motricity, because tissue levels of NA are not always a reliable index of neuronal activity. The turnover of NA might be modified without change of tissue levels, and the number of α-adrenergic receptors or their affinity for NA might be increased by estradiol in the oviductal smooth muscle, as in the uterine myometrium (Hoffman *et al.*, 1981). However, these authors revealed that only α_2-adrenergic receptor number was increased by estradiol treatment without a change in either the number of α_1-adrenergic receptors or the sensitivity of the myometrium to agonist-induced contraction, and that this could not lead to modifications of muscular activity.

It should also be noted that the NA estimation was performed in the circular layer of the oviduct. This could explain why there is no correlation between the modifications of NA concentration and the major effects of estrogen which mainly affect the long spike bursts if this kind of activity really originates from longitudinal smooth muscles.

Our results show that the main characteristic of tubal EMG activity recorded in vivo was the appearance of spike bursts of long duration, representing 85—88% of the overall MI, irrespective of the oviductal region or the hormonal state. This activity was stimulated by catecholamines and inhibited by phentolamine, but did not seem markedly affected by the ovarian steroid impregnation, as the oviductal NA concentration.

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