

Introduction

The mechanism by which the alveoli physically express milk from the lumen during milk removal is known as milk ejection. Stimulation of the mammary gland, particularly the teats or nipples, results in the secretion of the hormone oxytocin from the posterior pituitary. Oxytocin travels via the blood to the mammary gland and causes contraction of the myoepithelial cells surrounding the alveoli. This results in the expulsion of luminal milk from the alveolus into the ducts and out of the gland, resulting in the physical removal of milk from the alveoli. The role of milk removal complicates the interpretation of hormonal requirements for milk synthesis and secretion. Without frequent emptying of the mammary gland (milk removal), milk synthesis will not persist despite an adequate hormonal status. Conversely, maintenance of intense suckling or milking stimuli will not maintain lactation indefinitely. Nevertheless, suckling or actual removal of milk from the glands is required to maintain lactation.

Milk ejection is important during milking or suckling to obtain the alveolar milk fraction, which can represent more than 80% of the milk stored in the udders of dairy cows. In response to tactile teat stimulation, either manually or by the milking machine, milk ejection is induced by the release of oxytocin and resultant myoepithelial contraction. The time from the start of tactile stimulation until the occurrence of milk ejection spans between 40 s to >2 min and increases with a decreasing degree of udder filling. Therefore, cows need a longer pre-stimulation in the late stages of lactation or

if milking is performed shortly after the previous milking, whereas in full udders, pre-stimulation is less important. Milk ejection is disturbed under several conditions, such as during milking in unfamiliar surroundings or for several weeks immediately after parturition in primiparous cows. Disturbed milk ejection is due to a reduction or absence of oxytocin release from the pituitary gland. Milk ejection can occur under various conditions. Milk ejection can occur underwater, as in the case of whales, porpoises, sea cows, sea otters, and hippopotamuses. Milk ejection can also occur during flight, as in some bat species.

Milk Ejection Reflex

The reflex has an afferent (neural) and efferent (hormonal and blood-borne) pathway. **Afferent Pathway:** The greatest amount of innervations in the mammary gland is in the teats, where pressure-sensitive receptors are present in the dermis. Mechanical stimulation of the teats activates pressure-sensitive receptors in the dermis, where the pressure is transformed into nerve impulses that travel via the spinothalamic nerve tract to the brain. These nerves synapse in the paraventricular and supraoptic nuclei of the hypothalamus. When the cell bodies of the oxytocin-containing neurons are stimulated by these impulses originating in the teat, an action potential moves down the oxytocin-containing neurons from the cell body in the hypothalamus down the axon to the neuron ending in the posterior pituitary. This causes the release of oxytocin and neurophysin into the blood. The efferent pathway begins at this point.

Efferent Pathway: The efferent pathway begins with the release of oxytocin into the

bloodstream. Oxytocin then travels to the mammary gland via the blood, binds to oxytocin receptors on the myoepithelial cells, causing the myoepithelial cells to contract, resulting in increased intraluminal (intramammary) pressure and ejection of milk from the alveolar lumen. Oxytocin receptors are associated with myoepithelial cells, not the smooth muscle of the mammary gland. In mice, these receptors increase throughout gestation but are fairly constant during lactation.

Other Mechanisms of Milk Ejection

1. Myoepithelial cells also contract in response to vasopressin (ADH or antidiuretic hormone). Vasopressin has approximately 20% oxytocic activity.
2. Visual or auditory stimuli can induce milk ejection. Milk ejection is a conditioned response.
3. Stimulation of the genital tract, such as vaginal distention, causes the release of large amounts of oxytocin.
4. The mechanical tap stimulus does not involve the use of oxytocin. It occurs under anesthesia or denervation of the udder. It is not inhibited by epinephrine. Kneading or butting of the udder by the young may elicit this response in the mother. This may involve distortion of the alveolar or myoepithelial cell structures, resulting in milk ejection.

Timing is very important for milk ejection: The timing of oxytocin release relative to milk removal is an important factor affecting milk ejection. Oxytocin has a short half-life in the blood (0.55–3.6 min). This means that the removal of milk by machine or nursing must be closely timed with teat stimulation. The oxytocin concentration in the blood normally increases within 1-2 minutes after udder stimulation, but the amount released declines during milking. It is estimated that the bovine pituitary gland has about

800 mg of oxytocin. This is approximately 40 times the blood level in the resting condition. Only ~1/3 of pituitary oxytocin is released during milking. The sensitivity of the neuroendocrine reflex appears to decline as lactation progresses. Peak oxytocin occurs at 1 minute at 1-2 weeks of lactation and 2 min at 5-6 weeks. The maximum oxytocin concentration during milking also declines as lactation progresses. The minimum amount of oxytocin required to cause milk ejection is approximately 0.02 IU. However, an injection of 10 IU of exogenous oxytocin is used to induce milk letdown.

Stress in Milk Ejection

Inhibition of milk ejection: Various stressful stimuli that inhibit milk ejection are associated with increased activity of the sympathetic nervous system. Oxytocin action can be blocked by catecholamines (epinephrine and norepinephrine). These hormones are usually released in response to stressful situations and increase the tone of the smooth muscles of the mammary ducts and blood vessels. This results in a reduction in the amount of oxytocin reaching the myoepithelial cells and partial occlusion of the mammary ducts. Moreover, epinephrine directly blocks oxytocin from binding to myoepithelial cells. This phenomenon is termed peripheral inhibition of milk ejection. Thus, exogenous oxytocin does not cause milk ejection in animals exhibiting peripheral inhibition. A common cause of failure to milk ejection is associated with stress of milking in the early postpartum period, especially for primiparous cows. Stress inhibits the release of oxytocin from the posterior pituitary gland (central inhibition of milk ejection). Exogenous oxytocin is usually administered in these cases, causing milk ejection. Based on the above discussion on the peripheral and central inhibition of milk ejection, it can be stated that milk ejection occurs as a result of oxytocin release, which is normally coupled with the inhibition of central and peripheral inhibitory controls.

Role of the Autonomic Nervous System:

The autonomic nervous system is part of the central nervous system. It mainly controls visceral function. The autonomic nervous system is composed of two types of nerves: parasympathetic and sympathetic. There is no direct innervation of alveoli or myoepithelial cells

Parasympathetic nerves:

The neurotransmitter of parasympathetic nerves is acetylcholine. The mammary glands lack parasympathetic innervation.

Sympathetic nerves:

The neuroendocrine components of sympathetic nerves are epinephrine and norepinephrine (NE). Epinephrine (adrenaline) is primarily produced in the adrenal medulla. Norepinephrine is a neurotransmitter produced by peripheral nerves and nerves in the brain. Norepinephrine can also be produced by the adrenal medulla. It is well known that fright and stress interfere with the milk ejection reflex and may inhibit milk ejection. Fear and stress activate the neuroadrenal system and cause the release of epinephrine. The inhibitory effect of epinephrine on milk ejection occurs both centrally in the brain and in mammary glands. Norepinephrine and epinephrine inhibit oxytocin-induced contraction of myoepithelial cells. Stressful stimuli inhibit milk ejection. This occurs via epinephrine or norepinephrine derived from the adrenal gland or the sympathetic nerves by the following mechanisms:

1. Norepinephrine reduces myoepithelial cell contractile response to oxytocin; this is a direct inhibition at the myoepithelial cell level. Norepinephrine decreases mammary blood flow (amount of oxytocin to the gland), which inhibits mammary tissue.
2. Norepinephrine reduces oxytocin release from the pituitary gland; this is an indirect effect mediated by the inhibition of oxytocin release at the hypothalamic level. In bovine species,

norepinephrine is the primary catecholamine. Injections of norepinephrine into cows, which increase blood levels to 2 to 5X above normal, will cause a decrease in milk yield by 10%. Oxytocin levels were not altered. Emotional disturbances can inhibit the CNS part of the milk ejection reflex. This may occur, especially after calving in first-calf heifers. Injection of oxytocin may be needed to remove milk because failure to remove the milk will result in reduced yield through lactation

References

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