A NEUROPHYSIOLOGICAL THEORY OF A REPRODUCTIVE PROCESS

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Some of the known neuroanatomy and neurophysiology relevant to male sexual responses is briefly reviewed. A theory is presented which attempts to explain many of their properties. In particular, increases in chemical concentrations of neuroexcitatory chemicals due to stimulation of genital receptors leads by wave-like spread to ejaculation with orgasm as a concomitant. Threshold phenomena and refractoriness are natural consequences of the theory.

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The neurophysiological correlates of mammalian sexual responses have not been fully worked out. Although the broad features of the neuroanatomical pathways involved have been known for a long time (Vick, 1976) the details of such pathways and the activity of the cells within them are unknown. Consequently there is no satisfactory description of the neurophysiology of orgasm, as recently pointed out by Changeux (1985) and Rosen and Beck (1986). The present article contains an attempt to formulate a theory which might explain some of the observed phenomena. The author is not aware of any relevant studies of concomitant unit activity, so that for these there is a definite need. Summaries of what is known about the anatomy and physiology can be found in Kalat (1984) and Katchadourian (1985). A more detailed discussion is contained in Bancroft (1983).

We briefly review some of the material which pertains to the theory to be described, focusing attention on the male for which certain aspects are more clearly delineated. There are three components of the central nervous system which play significant primary roles in mediating sexual responses. Two of these are in the spinal cord and the other is in the limbic system, with emphasis on the septum. The neocortex does not play a major primary role in orgasm according to electroencephalographic studies (Heath, 1972; Bancroft, 1983; but see also Cohen. Rosen-Goldstein, 1976) although it is obviously important when there are associated visual stimuli and motor activity.

One spinal region that has a primary role is in the sacral segments S2, S3 and S4 and the other is in the lower thoracic and upper lumbar regions T12, L1 and L2. The latter is the source of sympathetic efferents which form part of the hypogastric plexus and cause smooth muscle contraction in the vas deferens, seminal vesicles and prostrate gland to deliver fluid to the urethra. The sacral component contains efferent and afferent neurons of parasympathetic and somatic types. The parasympathetic cells have cell bodies in the lateral horns of segments S2, S3 and S4. Their axons extend via branches of the pelvic splanchnic nerve (nervus erigentes) to the genitalia where synapses are made within ganglia. The postganglionic cells are involved with the supply of blood through vasodilation in the corpora cavernosa. It has also been suggested that sympathetic outflow is perhaps also involved in this process. The parasympathetic activity is influenced by efferents from the frontal lobe of the cerebral
cortex to the anterior periventricular nucleus of the hypothalamus which send fibers via the brainstem and spinal cord.

In addition to the autonomic fibers there are both motor and sensory fibers of importance in the sacral roots. These form the pudendal nerve. The sensory afferents, which enter the cord mainly at S2 and S3, originate in part from Meissner’s corpuscles in the external genitalia. The large numbers of such afferents are associated with an unusually high proportion of gray matter in the cord relative to higher segments. Within the cord, the central processes of such receptors may ascend without synapsing until they reach a nucleus in the thalamus, whereupon secondary neurons send fibers to the sensory cortex (postcentral gyrus). However, the processes of some receptors may ascend one or more spinal segments before synapsing with secondary neurons. The axons of these secondary neurons may give off collaterals as they ascend to the thalamic nuclei. Not without relevance is the fact that the central processes from hairless skin, including that concerned with sexual sensations, extend into the posterior horn and adjacent regions which are quite removed from the cell bodies of neurons which form the sympathetic (thoracolumbar) outflow. The latter are found in the lateral horns. This is also the location of the majority of the cell bodies and dendrites of motoneurons whose axons run in the pudendal nerve (mainly from S4) to target junctions in such skeletal muscles as the bulbocavernosus and ischiocavernosus. The latter are involved in the final stage of ejaculation.

The parts of the brain which have been mainly implicated in sexual function are in the limbic system. Activity in, or stimulation of, the septum is associated with pleasure and sexual enjoyment. The classic experiments of Olds and Milner (1954) on rat septal and hypothalamic self-stimulation were paralleled in a human subject by Heath (1972). Parts of the thalamus and amygdala are also involved (Kalat, 1984; Katchadourian 1985). Stimulation at some limbic sites may lead to erection or ejaculation in squirrel monkeys (see Bancroft, 1983, for a summary). There is also evidence that transection of the spinal cord may facilitate erection in monkeys, suggesting a possible descending pathway with an inhibitory effect.

The above neurobiological description is necessarily sketchy and incomplete. As Bancroft (1983) has pointed out, the exact details of the parts of the nervous system relevant to sexual function are simply not known either in the brain or spinal cord. There is even uncertainty concerning the neural pathways involved in erection (Wagner & Green, 1981; Rosen & Beck, 1986).

PREVIOUS THEORETICAL CONSIDERATIONS

Although there is no accepted complete neurophysiological theory of orgasm, a number of authors have formulated ideas or theories or made observations which are not without usefulness. These tend to be fragmentary and our summary of them will be approximatively in chronological order.

We distinguish four primarily and usually connected events; namely, erection, emission, ejaculation and orgasm. Emission refers to the nonforceful expulsion of seminal fluid. Based on studies of cats, Semans and Langworthy (1928) claimed that emission was controlled by sympathetic outflow in the first two lumbar roots whereas ejaculation was produced by activity in parasympathetic efferents from the sacral roots S2 – S4. According to Bancroft (1983) these hypotheses may not all be applicable to humans. The latter author alludes to the existence of a central neurophysiological event during orgasm.
The similarity of electrophysiological events during orgasm and those during epileptic discharges has been noted (Mosovich & Tallaferro, 1954; Heath, 1972). In one subject of Heath’s, paroxysmal activity occurred in the septum and spread to the amygdala and thalamic nuclei, but not the neocortex. Slow wave activity at a rate of about 2 per second, preceded orgasm by several seconds.

Masters and Johnson (1966) split the sexual response pattern into four components: excitation, plateau, orgasm and resolution. They have noted various physiological changes associated with each component and these are summarized in numerous places (see for example Katchadourian, 1985). An important distinction is made between ejaculation and orgasm; only the former is necessarily accompanied by a refractory period and orgasm may proceed without ejaculation.

Concerning the details of neurophysiological events during orgasm there has been very little written. In females, Sherfey (1974) proposed that orgasm involved a spinal reflex in which stimulation of stretch receptors in pelvic muscles leads to their ultimate contraction. Mould (1980) proposed that in females, a clonic contraction of pelvic and abdominal muscles occurs after pelvic vasocongestion. An extremely interesting idea was put forward by Davidson (1980). This is called the bipolar hypothesis as it suggests that when sexual excitement reaches a critical level, an orgasmic control center nervous system triggers an orgasm. This happen in two directions, as impulses travel to the brain to give a sensation of orgasm and impulses also travel to the genitals presumably to give rise to ejaculation. In the next section we introduce concepts which are not unrelated to, but are more specific than, Davidson’s ideas.

HYPOTHESES CONCERNING EVENTS PRECEDING, DURING, AND AFTER ORGASM

Let us examine the functions of sexual response as they appear in man. We have:

1. the primary function consists of the physical transfer of sperm from their site of genesis to enable them to unite with ova;
2. the occurrence of (1) is made to occur with high probability to ensure reproduction. This leads to the pleasure associated with sexual behavior which is necessary to ensure it is repeated with sufficient frequency;
3. to ensure that sperm are not accidentally lost or wasted, threshold phenomena are required. These are manifested in the usual prerequisite of an erection and a usually prolonged period of stimulation before ejaculation may occur.

These are complex requirements and we can only be amazed at the efficiency with which they are accomplished. We note that in none of the above references has there been any detailed plausible attempt to explain the nature of the threshold effect nor the refractoriness that follows ejaculation. The theory that we propose explains both of these phenomena.

It is well known (see for example, Kuffler & Nicholls, 1976) that whenever a nerve cell emits an action potential in vivo, sodium enters the cell and potassium is extruded into the extracellular space. In the normal course of events the ionic imbalance that is thus created is only temporary as active transport and other regulatory mechanisms lead to the transfer of ions back to their compartments of origin.

If the level of potassium in the external space is elevated then there is a change in potential as predicted by the well-known Goldman formula. Thus potassium build up depolarizes nerve cell membrane and changes the conductance properties to various
ions. This may lead to further action potentials or in the case of synaptic endings to the release neurotransmitters such as acetylcholine, glutamate and so forth.

If the local clearance mechanisms cannot cope with excess potassium ion concentration, the potassium may diffuse to neighbouring regions leading to the excitation (depolarization) of cells there. This may lead, under the appropriate anatomical and physiological conditions to a wave which propagates and is called spreading depression. This was first observed in the rabbit brain by Leao (1944) and the phenomenon was extensively reviewed by Bures, Buresova and Krivanek (1974). It has been implicated as occurring during certain unconscious states including transient global amnesia. During spreading depression there are elevations in transmitter released from depolarized synaptic terminals; in fact Van Harreveld and Fikova (1970) suggested that the neurotransmitter (glutamate) was the primary agent and the wave of elevated potassium a secondary phenomena. This distinction is not important in the present discussion. However we note that a prerequisite for spreading depression is a region of gray matter dense with cell bodies and dendrites. Also there is a threshold stimulus for its instigation (e.g., a certain level of potassium ion concentration) and a refractory period of a few minutes. It may spread from one region of the nervous system to another.

We now describe a theory of neurophysiological events during sexual activity and in particular explain several phenomena related to orgasm, including the following: (1) ejaculation; (2) threshold phenomena and why the time to ejaculation is variable; (3) emission; (4) orgasm as a pleasurable experience usually associated with ejaculation; (5) orgasm without ejaculation as it occurs in immature males (Bancroft, 1983); (6) the refractory period and in particular, (a) why it must follow ejaculation; (b) why it consists, in part, of the impossibility of an erection; (c) why it ends; (7) interspecies differences. For examples, why cats usually only require one intromission for ejaculation (Clemens & Gladue, 1979).

Within the bounds of the uncertainties in the participating neural circuitry, the following is a plausible sequence of events which explains properties (1)–(7).

An erection occurs due to activity in the parasympathetics, with cell bodies in the lateral horns, emanating from sacral segments S2–S4, with possible sympathetic involvement. Genital stimulation excites receptors such as Meissner’s corpuscles whose axons terminate in the posterior horn in the same sacral segments. Activity in second order neurons with ascending fibres eventually (possibly involving more synapses) excite cells in the septum and other parts of the limbic system. This provides the initially pleasuring sensation of sexual stimulation. Those same second order neurons give off collaterals which synapse with the sympathetics in the thoracolumbar segments. Excitation of the sympathetics leads to smooth muscle contraction which moves the spermatic fluid from the epididymis along the vas deferens to the urethral bulb.

Due to the intense activity of the primary genital receptors and the high density of their terminations in the cord, there is a large increase in the local level of potassium and neurotransmitters which cannot be reduced by the usual clearance mechanisms. Eventually this build-up of chemicals reaches a threshold level at which local propagation is possible as in the initial phase of spreading depression. Thus an elevation in excitatory chemicals is eventually made to occur in the lateral horn due to original increases in the posterior horn. When excess neuroexcitatory chemicals invade the lateral horn the motoneurons innervating the muscles such as bulbocavernosus and ischiocavernosus are suddenly synchronously excited and their contraction leads to ejaculation. Also invaded are the cell bodies of the parasympathetics so that these cells become refractory along with the motoneurons. There is now a period of at least a
few minutes, a typical time course for clearance of massive imbalances, during which active transport mechanisms and so forth restore the original levels of ionic and other chemical concentrations. Thus this time interval during which clearance occurs is the refractory period and no erection may occur during it. Note that it is not necessary for a fully-fledged wave to form as in spreading depression.

The previous paragraph contains an explanation of the fact that erection-ejaculation can occur in a decerebrate creature as there is no limbic or other involvement. However, to explain the intense pleasure and results such as those of Heath (1972) it is only necessary to postulate a concomitant wave like spread in the septum which leads to a massive excitation of neurons in the pleasure centers, similar to an epileptic discharge. In fact one of Heath’s subjects could achieve orgasm by local septal stimulation with the neurotransmitter acetylcholine.

Although the above outline is tentative, it is thought that the general principle does operate; that is, excitation leads to a local build up of excitatory chemicals which lead to invasion of another neighbouring population of cells. Refractoriness is an automatic consequence of the massive depolarization that ensues, similar to that in spreading depression. However the location of these proposed events is not certain. Are there simultaneous wave-like spreads in the limbic system and spinal cord? Or does a wave-like spread in one induce eventually massive activity in the other? In the latter case one can see the similarity with Davidson’s bipolar hypothesis if the primary event is in the cord leading to a transmission of excitation to the limbic system and to the muscles which cause ejaculation.

To explain orgasm without ejaculation in young males we assume that their anatomy is different and that intense activity in the genital receptors leads to a build up of excitatory chemicals in limbic or neighbouring structures. In this case there is perhaps only a weak spinal component and so refractoriness with regard to another erection is less pronounced.

We have attempted to explain most of the known properties or orgasm and ejaculation. To complete the explanation of properties (1)–(7) listed above we have the following. The time to ejaculation is variable because the time courses of build up of excitatory chemicals are variable. This is due to the variability in the existing chemical concentrations, level of metabolic activity, immediate history of activity, intensity and frequency of stimulation, and so forth. These remarks would apply both within an individual and across various individuals.

Emission may occur because there has not been a sufficient build up of local excitatory chemicals are variable. This is due to the variability in the existing chemical lateral horn and cause and cause ejaculation. One then expects a much weaker refractoriness if the above theory is correct. The interspecies differences are presumably due simply to differences in fundamental neuroanatomy and neurophysiology. For example, if the density of receptors is much greater there will be a much quicker elevation in excitatory chemical concentration and hence a faster ejaculation. We have not addressed the role of descending pathways. This completes the attempts to explain ejaculation and orgasm at a neurophysiological level. Although we have concentrated on male orgasm, it seems that some aspects of female orgasm might be similarly explained.

REFERENCES


