Physiology of Cavernous and Spongy Tissues

7

7.1 Their Role in the Erection of the Clitoris

7.1.1 Can These Tissues Be Referred to as "Erectile Tissues"?

The adjective does not apply to the two types of tissue in the same manner.

The **spongy tissue** has no actual erection capacity (this is all the more obvious compared to the male penis in men). Such as we have just observed, this tissue can be saturated with blood and become tumescent. However, due to the absence of any true albuginea, the thinness and the actual structure of the fibrous envelope, which is not constant (it is only present at the level of the bulbs), and especially to the absence of any sub-albugineal venous network, a blocked blood evacuation or a backpressure, such as can be observed at the level of the corpora cavernosa, is not possible.

This does not apply to the **tissue of the corpora cavernosa** where all parts are organised such as to make an erection possible: presence of a thick, extremely resistant and double-layered albuginea, presence of a considerably developed sub-albugineal venous network (Fig. 6.4), vascular network with significantly anastomosed sinus. **The tissue of the clitoral corpora cavernosa is precisely therefore an erectile tissue** and, as a result, there is no doubt concerning the fact that the clitoris has an erection capacity.

This capacity has, however, often been denied, even by eminent specialists, who have referred to the absence of any sub-albugineal venous network (which is completely inaccurate), to the fixity of the clitoral curve related to the presence of the suspensory ligament (but this is forgetting that this connection only really concerns the area of the angle), to the reduced dimensions of the corpora cavernosa and to the fact that these corpora cavernosa do not reach the end of the glans (which, however, corresponds to the same configuration as in the penis). Others, such as Pernkopf in 1943, acknowledged the erectile capacity, while adding that it was an "ineffective erection". Kobelt, whose remarkable work concerning the clitoris has already been mentioned in this study, strongly supported the idea (as from 1844) that the clitoris could enter into erection during the phase of sexual excitation. It is quite certain that, considering the dimensions of the clitoris, this erection remains discrete and limited, resulting however in a slight straightening of the descending portion, an increase in the size of the crura and clitoral body, a rigidity of the descending portion of the body and an extrusion of the glans outside the hood.[1]

7.1.2 Physiology of the Erection of the Clitoris

7.1.2.1 The Biochemical Cascade

This biochemical cascade is well known thanks to the remarkable work completed during recent years by pharmacologists and biochemists, who were searching for new drugs capable of treating cases of male impotence (Bennett AH.).

The process only starts when the phase of sexual excitation occurs, whether this phase is caused by mechanical stimulation (masturbation or other), erogenic stimulation (visual, auditive or other) or during sexual intercourse. This process involves the autonomic nerve endings of the trabecular structures and their capillaries (Fig. 7.1).

Specific parasympathetic nerve endings (nitrergic neurons), under the action of nNOS (neuronal form of the NOS enzyme, nitric oxide synthase), and the endothelial cells of the cavernous sinusoids, under the action of eNOS (endothelial form of the NOS enzyme) (Burnett Al et al.) will then play an eminent part by secreting NO, i.e. nitric oxide (non-adrenergic and non-cholinergic and yet the first neurotransmitter of an erection). At the same time, the sympathetic nerve system is inhibited. The NO will then activate an enzyme, the soluble guanylyl-cyclase (sGC), which will,

[1] However, during the orgasmic phase, the glans may retract under the hood (See chapter 15, The bulbo-clitoral Organ in the Sexual Act), page 129.

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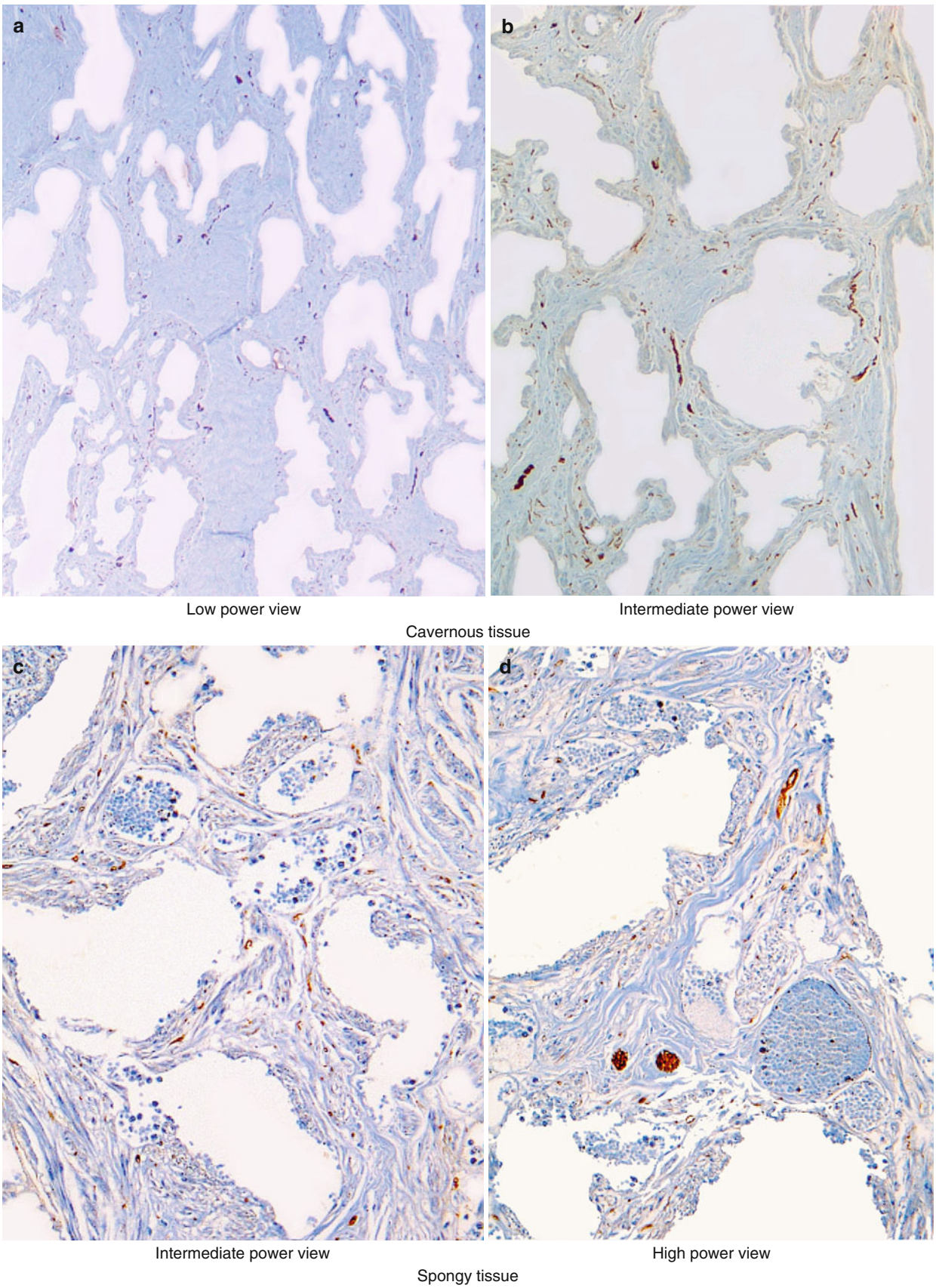

Fig. 7.1 Innervation of the cavernous and spongy tissues (PS100 immuno-staining) (dark-brown-stained nerves). (**a**) Low power view. (**b**) Intermediate power view. (**c**) Intermediate power view. (**d**) High power view

in turn, increase the intracellular concentration of cGMP, cyclic guanosine monophosphate (second neurotransmitter of erection). A hyperpolarisation through the activation of PKG (cGMP-dependent protein kinase) then occurs in the cell. This process activates the calcium-activated potassium channels (BKCa), thus causing a calcium leak and generating in fine, a relaxation of the smooth muscle fibres (Fig. 6.3), which is the starting point of the vascular and tissue phenomena initiating erection.

When sexual excitation decreases, the cyclic guanosine monophosphate is broken down by the type-5 phosphodiesterase (PDE-5), which will gradually cause the return of calcium inside the cell, the retensioning of the smooth muscle fibres and, consequently, the return of the undulations of the collagen fibres (this state being also favoured by the action of elastic fibres) through the restoration of adrenergic sympathetic tonicity stimulating the beta 2 adrenoreceptors of the cavernous tissue. The flaccid state is thus correlated with a state of spontaneous contraction of the smooth muscle fibres (recordable basal myogenic activity). However, there exists, within the clitoral smooth muscle fibres, receptors capable of either causing the relaxation of these fibres or restoring their initial myogenic state. Vasoconstrictive adrenergic alpha 1 receptors and several vasoconstrictive substances (neuropeptide Y, endothelin, PGF-2 alpha prostaglandins) have therefore been identified. Similarly, substances favouring the relaxation of the smooth muscle cells, such as the vasointestinal polypeptide (VIP), PGE 1 prostaglandins, etc., have also been identified.

7.1.2.2 Vascular and Tissue Phenomena

The relaxation of the muscle fibres occurs at two levels:

- Relaxation of the smooth muscle fibres of the superficial clitoral arteries (dorsal arteries) and deep arteries (paraseptal arteries of the corpora cavernosa and their intra-trabecular helicin arteries) by direct action of the parasympathetic fibres on the muscarinic receptors and VIP receptors of these vessels, thus causing their vasodilatation. As a result, a considerable increase of the arterial flow in these vessels occurs, thus allowing the cavernous lacuna to be filled rapidly.
- Relaxation of the smooth muscle fibres of the sinus trabecular walls. These sinuses then open (they become spherical), which is favoured by the relaxation of the collagen fibres of their wall but also by the relaxation of the collagen fibres of the cavernous albuginea. While the corpora cavernosa are being filled, the sub-albugineal veins are pushed back against the internal surface of the albuginea. The corpora cavernosa extend and expand, while their albuginea becomes thin. At the same time, identical phenomena occur in the spongy tissue, and in particular, in the bulbs. They are filled with blood under the effect of the blood flow increase and of the reception of the excess blood from the corpora cavernosa (for which they are used as a spillway), via the pars intermedia network and the direct bulbo-clitoral anastomoses (Fig. 6.8). The cavernous and spongy sinuses are saturated very rapidly, the sub-albugineal veins collapse and the evacuation of intra-sinus blood can no longer occur normally. When the albuginea is at its maximum tension and the filling process is no longer possible (no venous evacuation pathway), rigidity occurs (accentuated by the contraction of the ischiocavernosus muscles) and the clitoris enters into erection.[2]

7.1.2.3 Detumescence

When sexual excitation decreases, the cyclic guanosine monophosphate is broken down by the type-5 phosphodiesterase, which will gradually cause the retensioning of the smooth muscle fibres through the restoration of the noradrenergic sympathetic nerve tonicity and, consequently, the stimulation of the alpha 1 receptors of the clitoral arteries; the vasoconstriction and reduction of their flow; the restoration of the function of the sub-albugineal veins, which can once again combine with the cavernous veins and the emissary veins of the deep dorsal vein to evacuate sinus blood; the return of the undulations of collagen fibres thanks to the action of the elastic fibres arranged as a bridge; and the restoration of the albuginea's usual thickness and diameter. Such as we have just observed, the stimulation of the parasympathetic nerve system inhibits the stimulation of the sympathetic nerve system and, vice versa. Current neuroanatomical work tends to show that it is the hypothalamus which ensures the coordination of the two types of activity of the autonomic nervous system.

[2]The sub-albugineal network merges with the deep dorsal vein of the clitoris via emissary veins, which cross through the albuginea (Fig. 7.2). These veins provide a reduced drainage, which is residual when the sub-albugineal network collapses. This minimum drainage is essential to ensure that the clitoris is not threatened of asphyxiation and that the oxygenation of the tissues can be renewed. If this mechanism does not operate correctly, a pathological phenomenon may appear: clitoral priapism, of which several cases have been reported in medical literature.

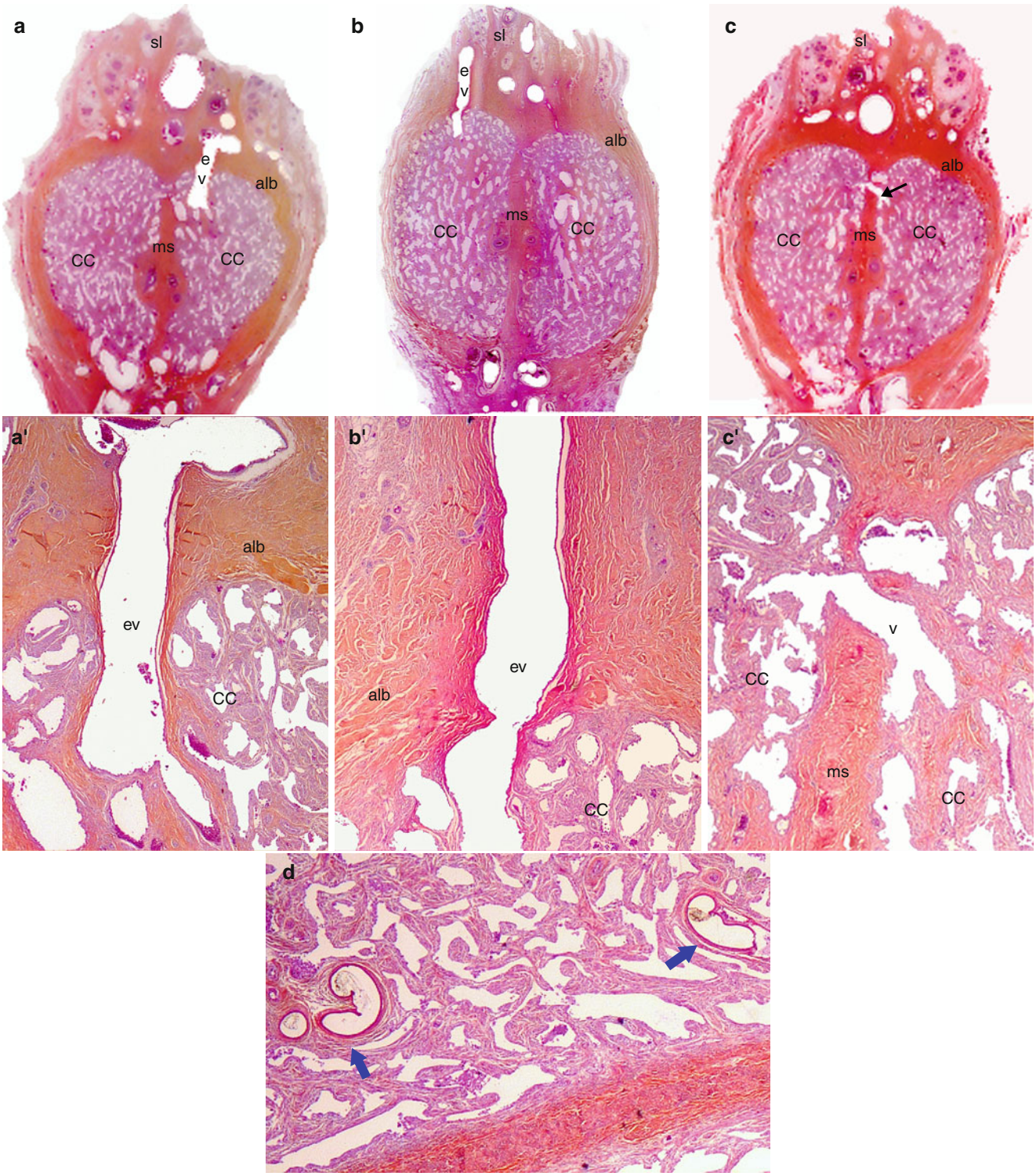

Fig. 7.2 Examples of emissary and draining veins of the corpora cavernosa. (**a**, **b**) Drainage to the suspensory ligament's veins (**a′**, **b′**: microscopic magnifications of **a** and **b**). (**c**) Communicating vein between the 2 corpora cavernosa (**c′**: microscopic magnification of **c**). (**d**) An example of intra-cavernous vein (usual drainage to the deep vein of clitoris). *alb* tunica albuginea, *cc* corpus cavernosum, *ev* emissary vein, *ms* median septum, *sl* suspensory ligament, *v* draining vein, *black arrow* transeptal vein, *blue arrows* they show intra-cavernous veins