Bladder diverticula are of two different types: 1. Those due to abnormal embryonal evolution giving rise to abnormalities, deviations or arrest in development. To this group belong the well-known diverticula of the urachus. But apart from these there exist other, less recognized diverticula probably due to evagination of the embryonal epithelium of the bladder; they are the supratrigional diverticula, those located in the centre of the vesical dome, and some varieties of paraureteral diverticula. A diverticulum is described into which the ureter ends; probably because of excessive ascension of the embryonal ureter. 2. Those which are not due to abnormalities of development or so-called acquired diverticula. There are two varieties in this group: the foetal diverticulum arising in the second half of pregnancy and diverticula formed after birth, which make up the majority. In the genesis of acquired diverticula, both predisposing causes (weak spots of the vesical wall) and determinant causes (any pathology causing prostatism and increase of the intravesical pressure) should be considered. This mechanism of formation is evident in the acquired diverticula after birth, and also in the foetal diverticula, since the urine contained in the bladder is evacuated to the amniotic cavity through actual micturition. It is possible that in this stage there already exist obstacles to urination. It is, therefore, proper to say that the same causes produce the same effects in all stages of life.

We will limit ourselves to the pathogenetic study of the vesical diverticula which will allow us a better understanding of their clinical picture and especially of the best therapeutic approach to each type in particular. Any progress in the study of the causes and genesis of lesions is always favourably reflected in the diagnosis and treatment of those diseases.

Soon after the study of this condition began, two opposing theories appeared concerning their pathogenesis. Several authors such as Hyman, Lax, Maier, Ferris, Watson and Lower, consider these formations to be of congenital origin. Others believe they are acquired as a result of micturitional difficulties.

Actually there exist both congenital and acquired diverticula, but it is necessary to define the meaning of these terms. For Englisch and Rathbun, congenital or true diverticula are those in which all the layers of the bladder can be seen in their walls, that is to say, the mucosa, submucosa and muscular layers. On the other hand, they consider acquired diverticula as those consisting exclusively of the vesical mucosa.

This criterion, rigorously applied, is not acceptable, as some acquired diverticula contain, at least for some time, muscular fibres. Also, the congenital diver-
ticula, containing at their beginning thick muscular walls, often terminate with no muscular fibres. This is due in part to the progressive distention of the diverticula, but mainly to sclerosis which always follows diverticulitis and peri-diverticulitis.

In my opinion, congenital diverticula are those which result from a deviation or arrest in the ontogenetic development of the urinary bladder. This abnormal evolution produces a true diverticulum during the first months of the intrauterine life. Acquired diverticula are those formed in the adult and the elderly, and are often coincidental with micturitional difficulties. However, we should emphasize that these diverticula may also appear in the young and even in the last few months of intrauterine life.

From the above considerations follows that there are three types of diverticula; congenital, due to developmental abnormalities which appear in the first few months of the intrauterine life; fetal, which are acquired during the last few months of intrauterine life; and finally acquired, the most common type, which appear during extraterine life.

**Congenital diverticula**

These are the diverticula developing in the vertex of the bladder at the expense of the urachus which has remained partially patent. They can be compared to the congenital inguinal hernias due to the persistence of the vagino-peritoneal process. The existence of a minute lumen in the urachus not always results in this type of diverticula. There always exists a predisposition to their formation and development and they can appear late when difficulties in micturition occur. Aside from this variety, there is no substantial proof as to the existence of any other type of congenital diverticula, which in the strictest sense of the word, mean abnormalities of development.

As a result of my observations, I described in 1948 a new variety of congenital diverticula; those developing in the midline of the posterior wall of the bladder immediately above the interureteral bar. The enclosed micro-photographs of a few months old foetus show, that in this moment of life there already exists a perfectly developed diverticulum (Fig. 1) consisting of a pouch located in the posterior wall of the bladder (5) and communicating with the vesical cavity (3) through a narrow portion, called the diverticular orifice (4). The cavity is covered inside by mucosa, continuous through the orifice with the vesical mucosa. Surrounding it a thick muscular layer is found with the posterior longitudinal band in the back and the bundles of the circular muscular layer – at the sides.

Above that level the beginning of the diverticulum is a depression in the posterior wall of the bladder.

In the sections below the diverticular neck (Fig. 2), made by scraping the interureteral bar, the perfect separation of the diverticular pouch (5) and the vesical cavity (3) is observed. This demonstrates that the diverticular orifice seen in the anterior slide is located above the interureteral bar.

*Urology and Nephrology* 1, 1969
Fig. 1. 1. Orifice communicating the diverticulum and the bladder. 2. Diverticular cavity. 3. Diverticular neck. 4. Anterior wall of the bladder. 5. Adenomatous medium lobe at the bladder neck.

In subsequent sections done below the one previously described, we can observe that the distance separating the diverticulum from the bladder cavity increases and finally the diverticulum disappears.

If instead of the horizontal sections, sagittal ones were used, we should be able to see a picture resembling the one found in an elderly patient, except naturally for the proportions (Fig. 3). This patient had cystostomy because of chronic...
Fig. 2. 1. Ureter. 2. Anterior wall of the bladder. 3. Vesical cavity. 4. Diverticular orifice. 5. Diverticular pouch. 6. Posterior wall of the bladder.

Fig. 3. 1. Ureter. 2. Diverticular cavity. 3. Vesical cavity. 4. Anterior wall of the bladder. 5. Trigone.
urinary retention due to bladder neck contracture and the progressive prosta-
tism caused a marked hypertrophy of the detrusor, in the section the interstitial
diverticulum (2) limited from behind by the posterior longitudinal band, and in
communication with the bladder cavity (3) is visible.
Therefore, we do not have to force our imagination to establish a close
relationship between both diverticula; foetal and adult.
What is the mechanism of formation of the true congenital diverticula es-
pecially in the two cases just described? No question that they are congenital.

Fig. 4. 1. Internal urethral orifice. 2. Diverticular orifice. 3. Diverticular
pouch. 4. Ampulla of the vas deferens. 5. Ejaculatory duct. 6. Bladder
neck. 7. Prostatic part of the external sphincter. 8. Anterior longitu-
dinal fibres of the detrusor. 9. Anterior circular fibres. 10. Posterior
circular fibres. 11. Posterior longitudinal band. 12. Interureteral bar

When sagittal sections of foetuses in the third month are examined, it some-
times can be observed that in the posterior wall of the bladder, a little above of
what will be trigone, the vesical epithelium starts a cul-de-sac between the latter
and the circular layer already developed and limited behind by the posterior
longitudinal band. It is not difficult to imagine that the cul-de-sac of the primitive
vesical epithelium may form a hernia through the circular muscular layer, giving
rise to this variety of congenital diverticulum.
The primitive bladder epithelium in its normal development never leads to evaginations but produces proliferations inside the vesical cavity. These observations suggest the possibility and probability that real congenital diverticula arise due to an orientational abnormality of the growing primitive vesical epithelium. Instead of growing towards the vesical cavity, it sinks in the mesenchyma, creating a cul-de-sac.

Fig. 5. 1. Internal urethral orifice. 2. Interureteral bar. 3. Ureteral orifice. 4. Diverticular orifice.

The same applies to diverticula located in the midline of the posterior bladder wall but located in a higher plane. An example of this variety is presented in Fig. 4 in which the diverticular opening (2) is located in the centre of the posterior bladder wall, high above the interureteral bar.

Figure 5 shows this diverticulum in an autopsy specimen sagitally cut. We can observe the diverticular pouch (4), separated from the peritoneum by a plane of detachment. I believe that the genesis of this diverticulum is similar to the former case; that is to say, during the fetal life an evagination of the vesical epithelium would be formed through the circular muscular layer and the posterior longitudinal band. The latter does not form at that level a compact bundle and it permits easy passage of vesical epithelium.

However, we have to establish a difference between the diverticulum located above the interureteral bar and the one just described which is located in the upper part of the posterior bladder wall. The latter one is in contact with the vesical peritoneum which explains its tendency to grow. When the diverticulum is located

_Urology and Nephrology_ 1, 1969

Supplied by The British Library - "The world's knowledge"
Fig. 6. 1. Anterior vesical wall. 2. Peritoneum. 3. Diverticulum. 4. Prostate. 5. Seminal vesicles. 6. Internal urethral orifice

Fig. 7. 1. Prostate. 2. Ureter. 3. Left ureteral orifice. 4. Left diverticular orifice. 5. Right diverticular orifice. 6. Right ureteral orifice
Fig. 8. 1. Diverticular stone. 2. Diverticular pouch. 3. Ureter. 4. Seminal vesicle. 5. Prostate

Fig. 9. 1. Levator anus muscle. 2. Seminal vesicle. 3. Juxtaureteral diverticulum. 4. Ureter attached to the diverticular wall. 5. Periureteral muscular sheath. 6. Vesical wall

Urology and Nephrology 1, 1969
just above the trigone, it remains separated from the peritoneum by the posterior longitudinal band; thus, it is an interstitial diverticulum caught among the bundles of the detrusor and having a tendency to become smaller in time. From this study a therapeutic conclusion may also be drawn: supratrigonal interstitial diverticula should be removed intravesically, and the diverticula located in the vesical dome should be removed extravesically; because if removed intravesically, there may be adhesions to the peritoneum which may be injured.

Another case similar in its genesis is the one in Fig. 6. We can observe two diverticular openings symmetrically located on either side of the midline above the trigone. In Fig. 7 each diverticulum is seen containing a large stone. Observe

Fig. 10. 1. Ureter about to end in the diverticular cavity. 2. Diverticulum

that the ureter is not in contact with these diverticula. Also note that both are separated by the posterior longitudinal band. I believe that the genesis of this diverticular variety is similar to that previously described, that is the vesical epithelium is evaginated between the third and fourth months of development. The surgical approach should be intravesical.

There is another variety whose importance deserves detailed description. I believe it is interesting to show a developmental anomaly in which the ureter ends in a congenital diverticulum. This was an adult male with bladder cancer who died without surgery. Sagittal autopsy section (Fig. 8) reveals the existence of a vesical diverticulum (2), next to it the juxtapelvesical ureter with its own musculature (3) in normal condition and also muscular bundles of the periureteral sheath which as always are separated from the ureter by an interstice.
On further examination of sections taken downwards it is observed (Fig. 9) that as the ureter almost penetrates the vesical wall, it is about ending in the diverticulum (4).

Figure 10 shows the ureter ending in the internal wall of the diverticulum. The intrinsic muscular fibres of the ureter end as always in the edges of the ureteral orifice.

Figure 11 shows the diverticular opening in the vesical wall, in the place where the ureteral orifice is normally located (1).

Further sections show that the bundles of the periureteral sheath progressively increase, forming the interureteral bar and the posterior half of the trigone.

This diverticulum is a developmental anomaly probably due to an excessive ascension of the ureter which would pull up a portion of the primitive epithelium of the bladder. Actually this would be an exaggeration of a normal happening since, as we know, the ureter derives from the wolffian duct from which it ascends towards its final location. It is interesting to know this variety because in intravesical diverticulectomy the ureter is involved and should be reimplanted into the bladder.

Acquired diverticula

These are the diverticula which are not produced by developmental anomalies. In the majority of the cases, they are produced during extrauterine life: but they can also originate in foetal life by similar mechanisms, that is, by mechanical causes.

*Urology and Nephrology* 1, 1969
We should consider two types of acquired diverticula: those formed during the extrauterine life and those originating during foetal life.

In the formation of these diverticula, both predisposing and determinant causes must be considered, regardless of a foetal or an extrauterine appearance.

The predisposing factors are anatomical, that is, there exist weak spots in the vesical wall. The determinant causes are all the pathological conditions causing micturitional difficulties and increase of intravesical pressure.

**Diverticula acquired after birth**

These can be located in any region of the urinary bladder. However, they have not been observed in two areas, the trigone and the anterior wall of the bladder. It is easy to understand why diverticula are never seen in the trigone; its great density and its partial adherence to the prostate are the factors which prevent the formation.

It is more difficult to understand why diverticula do not develop in the anterior wall of the bladder, but in exceptional cases. In my opinion this extremely rare occurrence is due to the anatomical constitution of this wall which is formed by three superimposed muscular layers: external longitudinal fibres, circular fibres, and plexiform or internal longitudinal fibres. Actually it is only in this area that the internal longitudinal fibres condense to form a true layer. In the other areas its existence is uneven. For this reason, although this is not the area richest in muscular fibres, it is the one which offers the greatest resistance to pressure. The resistance of a muscular wall depends as much or more on its architectonic disposition in various planes and directions, as on its quantity.

Apart from these, diverticula may appear in any other area of the bladder. However, there is a preferred site, corresponding exactly to the weak region of the detrusor. In fact, diverticula are principally observed in the posterolateral aspects of the bladder, near the parietal course of the ureter, generally above and outside of the structure.

There are two factors which explain the appearance of diverticula in the posterolateral-juxtaureteral region of the bladder.

1. **Architectonic disposition of the vesical musculature**

There is a large, weak zone in the bladder wall; it is a triangular-shaped area, limited behind by the strong posterior longitudinal band and in front by the plane of anterior longitudinal fibres. The vertex of the triangle corresponds to the meeting point of both the longitudinal formations; the base is the vesico-prostatic junction.

In well developed bladders the area of such a triangle is superficially formed by a continuous layer of external longitudinal fibres corresponding to the lateral group (Fig. 12); inside there exists another layer of circular fibres which are perpendicular to the former group. The plexiform layer, although very slightly devel-
oped, exists at this level. When this normal disposition is present, diverticular formation is very difficult, even with micturitional difficulties. However, not always the same anatomical disposition is found. There exist bladders in which the area of the lateral triangle only presents a few isolated muscular bundles in its superficial layer. In these cases, the vesical wall is formed in some points only by muscular bundles of the circular layer. When this occurs, all necessary conditions exist for diverticular formation by normal micturitional pressure, or by prostatism with increase of intravesical pressure.

2. Existence of a vesical hilum

The posterior part of the triangle or zone of least resistance is where the ureter along with vessels and nerves enters the bladder; so as to become in a way the hilum of the urinary bladder. Therefore it is understandable that this is the ideal location for the vesical mucosa and submucosa to go through and to form a diverticulum. In these cases the diverticulum is a simple hernia of the mucosa through the vesical musculature.

By this mechanism the majority of the paraureteral diverticula are formed. Figure 13 shows an example of this diverticular variety seen in a patient with long-standing prostatic adenoma; it can be identified in the interstitial phase produced by dissociation of the muscular bundles at this region. As usual, it is a cul-de-sac located laterally near the ureter. It is presumed that in time it would become extravasical. This is an example of acquired diverticula by increase of the intravesical pressure and affecting the weak zone of the bladder wall. Note its proximity to the ureter in order to avoid injuring it.
When the diverticulum is formed by the mucosa, as in Fig. 13, it is a hernial formation: that is a progressive process, and therefore its extirpation is on principle indicated. There are authors who believe that only the large diverticula should be removed; however, removal of the large diverticula leaving the smaller one untouched, would allow the latter to produce the same pathological phenomena originated by the progressive growth of these hernial formations.

Fig. 13. 1. Diverticulum ending in the vesical cavity

It is obvious that prostatism is an important factor in diverticular formation which points out the necessity of diagnosing and treating any obstructive condition to urinary outflow. Therefore when diverticula are removed intravesically, the vesical neck should be palpated and inspected because if diverticulectomy is not accompanied by removal of the obstruction, surgery will not be completely successful and urinary retention will probably persist to some degree. Although prostatism is an important determining factor in diverticular formation, its importance should not be overemphasized, since obstruction to urination is common and vesical diverticula are not. Also, there exist many cases in which an obstacle to urinary outflow cannot be demonstrated, not only in young individuals, but also in adults and in elderly. In these cases the normal intravesical pressure is enough to produce the diverticular hernia because of congenital weakness of the vesical wall.

In women vesical diverticula are uncommon, which is substantiated by the fact that obstacles to urination are less frequent in females.

Apart from these considerations, there are two mechanisms of formation for the acquired diverticula: the first is found in those initiated by a wide depression in one or several weak spots in the vesical wall (external paraureteral

Urology and Nephrology 1. 1969
region, Fig. 14). At the beginning, that depression broadly communicates with the vesical cavity; later on, the intravesical pressure, pushing the weak muscular bundles at the bottom of the hernia, atrophies and dissolves them. At the same time, the juxtapapillary muscular bundles tend to get together at the level of which it will later be the definite diverticular orifice as vestige of its origin. Diver-

Fig. 14. 1. Ureter. 2. Urachus. 3. Small diverticulum. 4. Vertex of the triangle. 5. Anterior side of the triangle. 6. Posterior side of the triangle. 7. and 8. Section of the lateral longitudinal fibres leaving the circular fibres which form the deep plane of the triangle. 9. Muscular fibres inserting to the pelvic aponeurosis. 10. Vessels and nerves of the vesicoepiploic junction.

ticula of this origin are characterized: 1. by place of implantation (weak areas of the vesical wall or posterolateral regions of the bladder above and outside of the ureteral orifice), 2. by having a wide neck, and 3. by having its own vessels and nerves demonstrable by dissection, which is logical since they represent a part of the vesical wall. As an example of the origin and evolution of this so common diverticular variety we present Fig. 14 showing a well developed, wide-necked diverticulum on the left side originated in the weak zone of the lateral vesical wall and in which some muscular fibres with a tendency to disappear are seen.

On the right side, a similar diverticular formation appears in its initial phase: it is all the posterolateral wall that yields to the intravesical pressure, simulating

Urology and Nephrology 1, 1989
an evagination (3). The muscular bundles are evident but weak and with a tendency
to atrophy. The position of the ureter far away from the midline, and as if dragged
up by the diverticulum, must be emphasized.

In cases similar to the one described above, that is in the initial phase of this
diverticular variety, if the trigone is examined (Fig. 14), the ureteral orifice is seen
to be displaced a little outward but always in a much smaller proportion to the
point of ureteral entrance in the vesical wall.

The second mechanism of formation can be compared to the way in which
the epigastric hernias begin and develop. These start by peritoneal invagination
following the areolar tissue and nerves at the time they perforate the linea alba.

The urinary bladder contains many vessels and nerves which go through
the vesical muscle mainly at the site described as vesical hilum. In these cases the
diverticular evolution is such that only the mucosa penetrates, originating a mi-
nute cul-de-sac as can be seen in Fig. 13, and constitutes the interstitial diverticula.
However, in time a surprising growth may be observed.

Until they are fully developed, the characteristics of this variety are: 1. a
narrow but long neck, 2. a lack of demonstrable vessels and nerves by dissection.
3. a general appearance at the age of prostatism, 4. they are usually multiple.
5. they are located in any area of the bladder except the trigone and the anterior
vesical wall, and 6. they lack muscular fibres.

They can be reproduced experimentally in cadavers by a simple maneuver:
that is to cut the muscular fibres in a point with the bladder moderately distended
and then, by squeezing with our hand a hernia of vesical mucosa is observed to go
through the created rent.

It has been possible experimentally to make diverticula in dogs, by establish-
ing an obstacle to micturition and weakening the vesical wall at a certain point.

Vesical eventions

Beside the acquired diverticula, there are similar formations originated
by the same causes and identical mechanisms.

Sometimes the weak portion of the bladder is enlarged, especially in the
space between the posterior and anterior longitudinal bands; then, by the normal
intravesical pressure or by a pressure increase in cases of prostatism, a wide but
shallow depression is formed affecting the entire area of the weak region, as can
be seen in Fig. 15. If this specimen is examined by transillumination, the depression
will be seen as formed only by the mucosa and submucosa, reinforced by weak
muscular bundles. The perimeter around this translucent zone differs in the
strength and solidity of its muscular bundles. These formations, determining a
clear symmetry of the bladder, probably persist as such during the entire life, or
increase slightly without ever becoming a true diverticulum. It is very difficult to
establish the limit between the true diverticula and these wide and shallow depres-
sions of the posterolateral wall of the bladder. Sometimes transitional cases

Urology and Nephrology 1. 1969
Fig. 15. 1. Interstitial juxtaureteral diverticulum. 2. Ureter. 3. Fibres of the ureter inserting to the ureteral meatus.

Fig. 16. 1. Right posterolateral initial diverticulum. 2. Ureteral meatus. 3. Internal urethral orifice.
Fig. 17. 1. Diverticular pouch. 2. Muscular fibres of the detrusor attached to the diverticulum. 3. Initial diverticulum of the posterolateral vesical wall. 4. Ureter

Fig. 18. 1. Internal urethral orifice. 2. Displaced ureteral meatus. 3. Diverticular pouch in the initial stage

Urology and Nephrology 1, 1969
Fig. 19. On the left side, sagittal section of the bladder and prostate: 1. Internal urethral orifice; 2. Urethral meatus; 3. Initial diverticular pouch. On the right side, the same specimen as seen by transparency.
are found as the one seen in Fig. 16; but it is also possible that some persist throughout life without producing substantial changes. This process is similar to the eventration of the abdominal wall, and should be considered as such. The existence of vesical eventration necessarily changes bladder function, since upon contraction, the urine going into these depressions causes a loss in the contractile capacity of the detrusor; this in turn produces a hypertrophy of the muscle in order to compensate for that functional deficiency. This situation may exist alone or may coincide with an obstruction to urination (bladder neck contracture, prostatic adenoma or urethral valves). Removal of obstructive lesions of the bladder neck and prostate usually results in a cure; although sometimes persistent phenomena of prostatism occur as chronic urinary retention which is due to the presence of vesical eventration. When vesical eventration is found alone, it causes vesical prostatism which should be considered in clinical urology when causes and evaluation of certain vesical retentions are studied.

Foetal diverticula

Some authors consider the existence of diverticula in foetuses and newborns, which at first sight constitutes a decisive argument in favour of their congenital origin because of deviation of the development.

Going through the literature I have found a few descriptions not enough studied from the anatomo-pathological point of view.

Fig. 20. 1. Longitudinal fibres. 2. Circular fibres. 3. Vesical cavity. 4. Trigone. 5. Diverticular orifice. 6. Diverticular cavity. 7. Seminal vesicles.

Urology and Nephrology 1, 1989
In my opinion, what happens during foetal life is that simple depressions are produced in the weak spots of the vesical wall, in persons with a defective constitution of the lateral triangle of the bladder, due to defective development of the longitudinal lateral fibres.

What we see at this stage of life is the initial phase of the diverticula. Nevertheless we sometimes find very well developed foetal diverticula, as can be seen in Fig. 20 corresponding to a full-term foetus.

In fact, we can see here the diverticular pouch (6) located outside the trigone (4) and communicating with the vesical cavity (5). But its mechanism of formation is similar to that during extrauterine life. That is the intravesical pressure produces a depression in the weak area of the posterolateral wall of the bladder, forming a pouch communicating with the vesical cavity, its walls being formed by all the layers of the bladder. I therefore think that these diverticula develop by mechanism similar to that of diverticula acquired during extrauterine life.

If we admit that most of the bladder diverticula are acquired and formed in the adult and old age, it seems quite inconceivable that they can also be found, even in their initial phase, in intrauterine life. But this is only apparent, as in reality foetal life there are circumstances similar to those found in extrauterine life. It is a well known fact that in the second half of pregnancy the bladder fills with urine which will be emptied in the amniotic cavity. And it is also possible that at this stage there may be difficulties of micturition with a consequent increase of intravesical pressure and may be the cause of these deformities in the way of pouches, which in reality are initial diverticula, comparable to what occurs in the adult with difficulties of micturition.

In my opinion this is a mechanical problem with the same principles applicable to both intra- and extrauterine life. Moreover, the same causes should exert a more marked effect on the foetus, if we consider that during foetal life the organs are more malleable and more susceptible to any influence.

References


Urology and Nephrology 1. 1969