Although the anatomical structure of the ano-rectum provides the \textit{framework} for continence, the interrelated \textit{functions} of the various parts are what determines the continence status of an individual. It is not possible to isolate any constituent organ or part of the distal bowel from its immediate neighbours in regard to the efficiency with which defaecatory control is exercised. An excellent anal sphincter muscle can be totally negated, not only by colonic overactivity (e.g. in severe diarrhoeal states), but also by a sluggish inert rectum that allows faecal impaction to occur. In the same manner, a colon or rectum of impeccable performance are negated by a patulous or damaged anal sphincter.

While the surgeon faced with an incontinent patient may be primarily concerned with the integrity of the anatomical and physiological structure of the ano-rectal organ, he must always remember the complex interdependency of the intricate neuromuscular and reservoir functions of different parts of the alimentary tract; and both of these must be placed in the context of the whole person. Brilliant ano-rectal surgical procedures are misplaced both in their intent and in their effect in a patient suffering incontinence because of senile dementia.

This section attempts to provide a logical programme for the diagnosis of the causes of incontinence, and the application of the outcome of this process for the management of an individual patient with incontinence. The key to a successful surgical result lies in appropriate matching of the clinical problem with the correct operation. While the surgeon has to be aware that any repair must be carried out with due consideration of the anatomy, the final test of his operation will be physiological, i.e. the completeness (or otherwise) of defaecatory control. Such is the subtlety of ano-rectal function that the surgeon needs to combine physiological knowledge, clinical flexibility and sound judgement with an impeccable surgical technique when he is seeking to alleviate incontinence by surgical opera-
tions involving the muscles of the pelvic floor and anal sphincters.

**Patho-physiology of Continence**

[3,4,5]

**Proximal Alimentary Tract**

Disorders of the proximal gastro-intestinal tract that may be associated with incontinence are shown in Fig. 2.1 a, b. Explosive diarrhoea is the principal underlying factor that is common to all these varied conditions.

**Colon** [6,11]

It is impossible to consider continence of the ano-rectum without reference to the colon. If the colon transmits loose stools to the rectum in an uncontrolled and/or violent way, normal ano-rectal physiological mechanisms are not invariably able to guarantee continence. To provide crude illustrations of such colon-originated breakdowns of the continence mechanism one can consider the case of the patient with massive diarrhoea (e.g. from dysentery or cholera) who is frequently completely incontinent; similarly surgeons using massive intestinal purgation to effect a clean colon prior to surgery also reproduce the circumstances in which extreme intestinal hurry coupled with watery stools causes incontinence despite a physiologically intact ano-rectum.

Normally the colon works quietly, by low pressure activity occurring in an intermittent manner. This type of activity occurs over short lengths (5.0 cm) and is called “segmentation”; it is responsible for kneading and turning over the faecal content (Fig. 2.2). Excessive segmental contractions can cause slowing of faecal transit, and the extra time spent by the faecal stream in the colon allows increased resorption of water. Such delayed colonic passage results in a type of constipation associated with scybaloous small faecal pellets. Such tiny hard faecal “pebbles” are unable to initiate a normal defaecatory response by rectal distention, and the patient frequently responds to this situation by excessive abdominal straining. Every so often segmental activity of the normal colon is replaced by “mass movements” which are now known to be peristaltic in nature: these result in abrupt onward emptying of long (30–45 cm) lengths of colon, and when the contents of the pelvic colon are thus emptied into the rectum by such activity, the sudden rectal distention so produced triggers defaecation. If the colon becomes inert or paralysed (e.g. by excessive use of drugs – the “cathartic colon” syndrome) the rectal wall is not stimulated by the normal rapid stretching that accompanies such emptying of the pelvic colon (and which is a feature of the well-known gastro-colic reflex that commonly occurs at breakfast-time), and a loaded (impacted) rectum that does not empty efficiently can become a cause of “spurious” diarrhoea due to overflow faecal incontinence.

Abnormal constituents appearing in the faeces (e.g. laxative drugs such as senna or bisacodyl; blood; bile) can cause prolonged or excessive peristaltic activity. Certain pharmacological enemas contain material that directly stimulates such hyperactivity (e.g. sodium acid phosphate, sodium picosulphate, oxyphenisatin) which can overwhelm the anorectal control mechanisms.

Stress, neurological disorders and psychiatric disturbance can each inhibit normal colon transit mechanisms. Paraplegia can be complicated by massive colo-rectal dysfunction. Some drugs used in the treatment of mania, depression or anxiety (e.g. diazepam, lithium carbonate) can produce severe colonic faecal retention by either inhibiting peristaltic activity or by promoting excessive segmental contraction. If the rectum becomes overloaded, incontinence can result. Subnormal mental states are commonly associated with both constipa-