Chapter 16: Epistaxis

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History

Epistaxis is mentioned in medical literature dating back to very early times. Hippocrates (fifth century BC) was probably the first to appreciate that pressure on the alae nasi was an effective method of controlling nose bleeds, although in some cases he resorted to nasal packing and the application of cold fomentations to the shaved head. He regarded the complaint as being primarily of young persons, and was the first to describe vicarious menstruation.

Ali Ibn Rabban Al-Tabiri (AD 850) devoted a chapter of his massive work *The Paradise of Wisdome* to epistaxis. In it he wrote: 'The complaint of nose bleeding is due to swelling of a vein and its rupture, or perhaps a reduction in the force which confines the blood within.' He implied that some of the medications inserted into the nose owed their efficacy as much to their temperature as to their pharmacological properties.

Morgagni (1769) recognized 'the extremely turgid blood vessels about that part where the alae nasi are formed with the bone, about a finger's breadth more or less from the bottom of the nostril'. He was reported to have stopped nose bleeds by introducing his finger and 'pressing that part whereupon the blood ceased to flow, so that it was not even discharged by the posterior nostril into the fauces'. Morgagni drew his inspiration from his former teacher Valsalva and for this reason Little's area is referred to as 'Locus Valsalvae' in Italian circles. Morgagni's records also contain the suggestion, previously entertained by Valsalva, that nasal haemorrhage might be arterial in origin for it was his practice to 'syringe the nose with cold water and to apply the spirit of wine, especially to contract the mouths of swollen arteries'.

Mahomed (1880-1881) who pioneered the development of the sphygmomanometer stated that 'the frequency with which severe epistaxis occurs in old people with high arterial pressure is striking and for them very fortunate for if their noses did not bleed their brains would'. In 1879, Little published his case reports in the *Hospital Gazette* (Rainey, 1952) in which he identified the site of bleeding as being at the caudal end of the septum, and a year later Kiesselbach made similar observations. However, even after the introduction of modern histological methods, investigations into the mechanism of epistaxis were few and relatively uninformative, so that until recently very little was known about the pathology of nasal blood vessels.

The first attempts at arterial ligation were in 1868 (Bartlett and McKittrick, 1917) when Pilz of Breslau tied the common carotid artery, and it was much later that external carotid ligations were performed for the control of nose bleeds. Seiffert (1928) introduced ligation of the internal maxillary artery via a transantral approach and Goodyear (1937) was the first to tie the anterior ethmoidal artery.
Vascular anatomy of the nose

Textbook descriptions of the vascular anatomy of the human nose are based on Zuckerkandl's original and comprehensive studies of the subject (1982). The nose is vascularized by the internal and external carotid arteries via their respective branches, there being a confluence of the two systems, particularly at the caudal end of the septum where a number of arteries anastomose with each other (Little's area).

With the exception of Little's area, the middle turbinate has for a long time been regarded by clinicians as the dividing line between the internal and external carotid distributions, with a corresponding imaginary line of demarcation at the same level on the nasal septum (Weddell et al, 1946). This landmark has served as a guide in deciding which of the two areas is responsible for the epistaxis, and has allegedly helped the surgeon to decide which artery to ligate in severe cases of epistaxis.

The dividing line between the two carotid distributions may not, however, coincide exactly with the level of the middle turbinate. The work of Zuckerkandl (1892) and Burnham (1935) indicates that the blood supply to the turbinate is derived exclusively from the external carotid artery and that anastomosis between the two carotid distributions occurs above and anterior to its attachment to the lateral nasal wall, and not within it. They also described an artery to the superior turbinate and meatus, with a corresponding vessel on the septum, both of which originate from the nasopalatine branch of the sphenopalatine artery (external carotid).

Shaheen (1967) confirmed the presence of a branch from the nasopalatine artery supplying the superior meatus, turbinate and corresponding septum by X-raying the excised nasal fossae of cadavers which had been previously injected with barium-gelatin mixtures. It would therefore seem that the area designated as receiving blood from the internal carotid artery is smaller than previously supposed. Certainly the gross disproportion between the diameters of the anterior ethmoidal artery and the sphenopalatine at their points of entry into the nose would corroborate this view. The surgeon who lacerates the anterior ethmoidal artery in an external ethmoidectomy rarely has difficulty with haemorrhage; similarly those who deliberately ligate this vessel for epistaxis are always impressed by its small size. By contrast the terminal segment of the internal maxillary artery is a much larger vessel. The calibre of the posterior ethmoidal artery is also small, so that its contribution to the nasal blood supply is unlikely to be significant even if it varies reciprocally in size with the anterior ethmoidal vessel as suggested by Batson (1935).

It is noteworthy that the anterior ethmoidal artery was found to be absent unilaterally in 14% of cadaver dissections, and bilaterally in 2.5% of cases, the canal being either imperforate or filled with fibrous tissues or nerves (Shaheen, 1967).

This supports the contention that these vessels contribute very little to the blood supply of the nose, even if a somewhat larger posterior ethmoidal artery is found doubling for the missing anterior vessel and running a similar course to it, as sometimes happens. In this connection, the surgeon who sets out to ligate the ethmoidal vessels should be aware that, when the anterior vessel is missing, the posterior ethmoidal artery may arise directly from the circle of Willis and may, therefore, not be encountered in the orbit at all. This arrangement
conforms much more to the state of affairs in early embryonic life when the posterior ethmoidal artery is the dominant vessel of the nose, dwarfing not only the anterior ethmoidal artery but the nasopalatine vessel as well (Shaheen, 1967).

Burnham (1935) in his description of the anatomy of the lateral nasal wall claimed that the arteries to the inferior and middle turbinates and their respective branches lay partly embedded in the bone of these structures. In the case of the inferior turbinate, he found the bony canals containing the branches of the inferior turbinate artery extended along the central three-fifths of the bone. The middle turbinate artery and its branches were protected by a bony covering in the posterior half of the concha. Thus a considerable segment of both of these arteries and their branches is unlikely to give rise to epistaxis even if rupture occurs. By the time the arteries have emerged from their bony channels to lie beneath the mucous membrane, they will have diminished considerably in size.

Ogura and Senturia (1949) found in a series of patients with epistaxis that the bleeding point arose on the lateral wall in 28 out of 88 cases, and other authors have similarly implicated the lateral wall as a common site for bleeding. Shaheen (1967), on the other hand, was unable to find any cases of bleeding from the lateral wall of the nose in 117 cases, and his anatomical dissections and serial sections of the nose confirmed the findings by Burnham (1935).

The vast majority of patients who suffer from arterial epistaxis bleed from the nasal septum, and chiefly from the area where anastomosis of the nasopalatine, greater palatine, anterior ethmoidal, and coronary arteries takes place. This plexus was originally described by James Little and it is important to note that bleeding from it is arterial in origin, and not venous as some reports suggest. The venous bleeding, which is common in young persons, arises from the vein which lies immediately behind the columella at the anterior edge of Little's area. It runs vertically downwards and crosses the floor of the nose obliquely before joining the venous plexus on the lateral wall of the nose.

The dynamics of the nasal circulation depend to a large extent on the presence of arterioarterial anastomoses between the various arteries which contribute to the vascular supply of the nose. The branches of the anterior and posterior ethmoidal arteries join in a series of arcades in the upper one-third of the nose and the branches of the sphenopalatine artery anastomose with those of the ethmoidal arteries above the level of the middle turbinate. Opposing heads of pressure meet in the anastomoses with a sharp interface between the two, which can be displaced by dropping the pressure in one or other of the opposing systems. Shaheen (1967) demonstrated, by means of dye injections into the carotid vessels of live humans, that the dispersion of dye in the nasal mucous membrane could be affected by dropping the pressure in the system not being injected. For instance, dye injected into the internal carotid artery failed to appear in the nose, confirming the poor circulation of the ethmoidal vessels, but when the external carotid was occluded at the time of injection the entire upper half of the nose was suffused with dye from above downwards. The rapidity with which such dye displacement takes place, confirms the importance of the arterioarterial anastomoses in the nose.
The importance of possible anastomoses across the midline must also not be overlooked, either at the nasopharyngeal end or between the two anterior ethmoidal arteries at the crista galli.

These observations could well explain the many documented reports of failed ligation in which surgeons assumed, probably incorrectly, that they had tied the wrong vessel simply because bleeding has not stopped after ligation.

The arteriovenous anastomoses which are present at the anterior end of the inferior turbinate and septum at a microscopical level are probably of little importance in the aetiology and persistence of epistaxis, but their precise role is as yet far from clear.

**Clinical manifestations**

The prevalence of epistaxis in random samples of the population was found in one study to be between 10 and 12% (Shaheen, 1967). The age distribution shows an increase in frequency between the ages of 15 and 25 years, and later from 45 to 65 years, but with little difference between the sexes.

In only a small number of cases can epistaxis be attributed to a well-defined primary cause, such as a blood dyscrasia, a blood vessel abnormality, or local nasal pathology. In the majority of cases bleeding arises from an artery or a vein without any obvious abnormality to account for it; hence the terms 'spontaneous' or 'idiopathic epistaxis' which have been coined to cover this, the commonest category of epistaxis.

Certain contributory factors may be implicated in the onset of bleeding in cases of so-called 'spontaneous epistaxis', such as nose blowing, sneezing, coughing, straining, pregnancy, coryza and sinusitis. They all share one thing in common, namely a sudden rise in vascular pressure.

Venous epistaxis from the retrocolumellar vein tends to occur in subjects under the age of 35, whereas arterial epistaxis occurs in the older age groups. The duration of bleeding, as might be expected is short-lived in venous epistaxis, and quite prolonged in bleeding of arterial origin (*Table 16.1*).

**Table 16.1 The striking difference in duration between venous and arterial epistaxis**

<table>
<thead>
<tr>
<th>Subjects</th>
<th>10 min or under</th>
<th>Over 10 min and under 2 h</th>
<th>Over 24 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects over 35 years</td>
<td>10</td>
<td>18</td>
<td>44</td>
</tr>
<tr>
<td>Subjects under 35 years</td>
<td>28</td>
<td>13</td>
<td>4</td>
</tr>
</tbody>
</table>

Furthermore, there is an inverse relationship between the frequency and duration of epistaxis, the more severe arterial haemorrhages recurring rarely more than once or twice. No correlation can be established between the prevalence of epistaxis in random samples of the population, and their blood pressure status, although there is some correlation between the
severity of epistaxis and the degree of vessel wall disease as judged by retinoscopy (Shaheen, 1967). The finding of a high proportion of subjects with high blood pressures in hospital practice (Table 16.2) signifies, not that hypertension causes epistaxis, but rather that patients with higher blood pressures have more severe or persistent bleeding and are therefore eligible for hospitalization.

Table 16.2 The blood pressure distribution of subjects attending hospital with epistaxis. Pressure adjusted to a standard reference age for both sexes

<table>
<thead>
<tr>
<th>Diastolic pressures (mmHg)</th>
<th>Number of subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>65</td>
<td>2</td>
</tr>
<tr>
<td>70</td>
<td>0</td>
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<tr>
<td>75</td>
<td>3</td>
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<tr>
<td>80</td>
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<td>7</td>
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<tr>
<td>110</td>
<td>5</td>
</tr>
<tr>
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<td>6</td>
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<td>3</td>
</tr>
<tr>
<td>130</td>
<td>1</td>
</tr>
<tr>
<td>135</td>
<td>0</td>
</tr>
<tr>
<td>140</td>
<td>2</td>
</tr>
</tbody>
</table>

The pathology of nasal arteries

Examination of the medium and smaller nasal arteries of persons dying in middle and old age has shown that these are subject to a progressive replacement of the muscle tissue in the tunica media by collagen (Shaheen, 1967). This change varies from interstitial fibrosis to almost complete replacement of the muscle by scar tissue. It seems that persons giving a history of epistaxis exhibited the more severe changes, but this is not to say that these changes are necessarily responsible for vessel rupture. They could, however, account for the lengthy duration of arterial haemorrhages, presumably because of a failure of the vessel to contract down in the absence of sufficient muscle in the tunica media.

It is also apparent that larger vessels of the calibre of the maxillary artery are prone to calcification (Mönkenberg’s sclerosis). The resulting lack of elasticity could well contribute to the pathogenesis of small vessel rupture by the creation of a local systolic hypertension.

The precise mechanism of bleeding is thought to be a dissecting aneurysm of the nasopalatine artery or one of its branches, but the factors initiating the process have, so far, not been identified.
It is also a mystery why bleeding should occur from the retrocolumellar vein in young subjects. Careful inspection of the site shortly after a bleed sometimes reveals a tiny area of local ballooning overlying the vein, and this could possibly signify an area of vessel wall weakening, perhaps as a result of localized ischaemia.

Clinical management of spontaneous epistaxis

The young person with recurrent bleeding

After taking a careful history to establish that bleeding is not secondary to systemic disease, the nose is examined for signs of recent bleeding and for local abnormalities. In the absence of any obvious local disease, attention is turned to the septum which will often reveal an engorged vein at the anterior end of Little's area just behind the columella. If bleeding has been quite recent, a microaneurysm may be seen in the mucosa overlying the vein. Topical anaesthesia with 5% cocaine followed by cauterization with a silver nitrate stick will suffice to control most cases of epistaxis.

Some cases are particularly obstinate and require more than one application of a caustic agent, in which case it may be more effective to use trichloroacetic acid. Great care must be taken to ensure that none of the acid comes into contact with the nasal vestibule, as this will leave a particularly painful burn. There are some patients who bleed in spite of seemingly adequate attempts at cauterization, and the best policy is to coagulate the offending vessel with diathermy under general anaesthesia. Galvanocautery under local anaesthesia is not to be recommended in children, and even adults find the experience unpleasant. The sight of the heated filament, the sensation of heat within the nose, and the smell of charred flesh are off-putting to all but the hardiest individual.

Treatment of epistaxis in the young

Pinching the nostrils is the time-honoured method of stopping venous bleeding from the caudal end of the septum. Once bleeding has ceased, the nose can be cocainized and cauterized, although the vessel may bleed during the process of applying the caustic agent. Perseverance is required until bleeding finally stops.

Treatment of recurrent epistaxis in older subjects

Epistaxis in older persons does not recur with the same frequency as it does in younger people. Some patients only have the one major bleed, and when examined afterwards there may be very little to see. In such cases, there is nothing to be gained by cauterizing Little's area, unless it is certain that bleeding previously originated from this part of the septum.

Assessment of the cardiovascular system is important, however, and the patient should be referred to a physician if any abnormality such as hypertension is discovered. This is not so much to prevent further epistaxis, as to protect the individual from the harmful effects of the raised blood pressure.
Management of epistaxis in the elderly

Observations of the patient's pulse, blood pressure, and general condition are made in order to gauge the extent of blood loss. Estimation of the packed-cell volume in conjunction with the haemoglobin will also guide the clinician as to the need for replacing lost blood. At the same time the bleeding and clotting times and platelet count should be investigated to exclude a blood dyscrasia.

The nose is examined, preferably with the patient sitting upright in a chair. A plastic cover is draped around the neck and a bowl placed in the hands. Inspection of the nose may show a spurting blood vessel on the nasal septum, but usually the site of bleeding is smothered in blood. Cocainizing the nose with a 5% spray serves two purposes, namely to allow the introduction of a catheter so that the blood can be sucked away, and to stop bleeding by vasoconstriction. This gives the examiner the opportunity of locating the site of bleeding, but if this has stopped, suspect areas can be gently rubbed with orange sticks loaded with cotton wool in an attempt to cause further bleeding.

The bleeding point is cauterized if accessible, but quite frequently it is situated far back on the septum or behind a spur so that cauterization is technically impossible.

If the bleeding persists, the nose should be packed, preferably with ribbon gauze medicated with a suitable antiseptic such as bismuth-iodine-paraffine (BIPP). This can be left undisturbed for several days without fear of the patient developing complications. The old-fashioned method of controlling epistaxis by sitting the patient up with a cork between his teeth (Trotter's method) and allowing him to bleed until he becomes hypotensive is to be deprecated. Death from coronary thrombosis secondary to hypotension is a well-recognized complication of epistaxis and regularly appears in the Registrar-General's mortality statistics. It must be emphasized that old patients with poor hearts and circulations do not tolerate severe and prolonged blood loss.

Some clinicians prefer to insert an inflatable balloon in preference to ribbon gauze which is abrasive to the nasal mucosa. However, inflatable balloons will not adapt themselves readily to the irregular contours of the nose, and are therefore less reliable in the control of epistaxis.

After two to three days the packing can be removed, and in most instances bleeding will have stopped completely. If recurrent bleeding occurs, however, the pack will have to be re-inserted. Patients in this category, together with those suffering from prolonged epistaxis and those who are generally unfit, should always be hospitalized. They should be nursed sitting up and sedated to allay their anxiety and lower their blood pressure. The choice of sedative is a matter of individual preference, but opiates such as Omnopon (morphine) are popular, although they may sometimes cause vomiting. Diazepam by injection is also effective. If bleeding persists in spite of adequate packing, serious consideration must be given to the need for arterial ligation. There is some evidence to suggest that submucous resection of the septum may be helpful when bleeding originates from behind a prominent septal spur.
Postnasal packing may be helpful in those cases where anterior packing alone has failed to control the bleeding. It is particularly indicated in the control of haemorrhage following adenoidectomy. It is normally undertaken under general anaesthesia but can be accomplished under local anaesthetic in the cooperative patient. A small catheter is passed through each nostril from anteriorly backwards into the oropharynx. These are then drawn out through the mouth, and tied to two tapes which are secured to the pack. The catheters and attached tapes are then pulled forward through the nose, and tied across a bolster or dental roll which protects the columella. A piece of thread, previously attached to the lower edge of the pack is brought out through the mouth and secured to the cheek with adhesive tape. The anterior nasal cavities can then be packed with BIPP. The patient should be covered with a suitable broad-spectrum antibiotic and, after the bleeding has been controlled for a few days, the anterior packing removed. After cutting the tapes knotted across the columella, the pack can be removed through the open mouth, by pulling downwards on the lower central thread.

**Arterial ligation**

The patient who continues to bleed every time the pack is removed or who keeps on bleeding with the pack *in situ* will generally have to be transfused. If, over a period of 4-5 days, bleeding has not stopped, arterial ligation should be performed. In the absence of definite knowledge about the whereabouts of the bleeding point, it is reasonable to interrupt the external carotid system, since this supplies as much as 90% of the nasal mucosa. Bleeding from the ethmoidal region is in fact very uncommon and is rarely of a severity to merit arterial ligation, in spite of the occasional report describing severe ethmoidal bleeding.

Although interruption of the internal maxillary artery has become fashionable, it is by no means certain that this is necessarily more effective than ligation of the external carotid artery. Being nearer to the source of bleeding, the drop in blood pressure in that part of the nose supplied by the maxillary artery is greater than after tying the external carotid. From this, dividing the maxillary artery should, in theory, be more effective, but, as mentioned previously, the drop in pressure almost certainly encourages the displacement of blood from other areas of the nose through arterial anastomoses, with the possibility of bleeding persisting. Pearson, Mackenzie and Goodman (1969) also pointed out that continued haemorrhage after maxillary artery ligation could result from retrograde blood flow by way of arterio-arterial anastomoses between branches of the maxillary artery in the pterygopalatine fossa. Such distortions of flow would by-pass the interrupted segment of the maxillary artery thereby contributing to the persistence of haemorrhage. The descending palatine artery was particularly singled out by them as being a possible source of retrograde bleeding into the final portion of the maxillary artery, the moral being that not only should the main trunk be ligated as close to the nose as possible, but that as many branches as possible should be interrupted. By contrast, ligation of the external carotid artery does not produce quite the same blood pressure reduction distally and is, therefore, less effective in controlling bleeding, although blood flow from the ethmoidal to the nasopalatine areas may, in fact, be less. If bleeding persists after ligation of the maxillary artery, it is logical to proceed to interruption of the anterior ethmoidal artery with the prospect of arresting the bleeding permanently. The addition of ethmoidal artery ligation to external carotid ligation for persistent haemorrhage is less likely to be as effective, since the cause of the persistent bleeding in this case is probably inadequate drop of pressure in the distal external carotid, rather than displacement of blood from one area of the nose to another. In cases of hypertension, it would be
reasonable to ligate the maxillary and anterior ethmoidal arteries empirically at the same sitting.

**Ligation of the internal maxillary artery**

This operation is usually performed under general anaesthesia. A sublabial incision is made and then as large an opening as possible is made in the anterior antral wall without compromising the infraorbital nerve. The thin posterior bony wall of the antrum is shattered gently with a gouge and removed piecemeal with punch forceps to reveal the underlying periosteum on the posterior wall of the maxilla. This is incised horizontally from side to side and the fat of the pterygopalatine fossa teased out with long straight artery forceps until the tortuous maxillary artery is seen. The artery is divided between clips as close to the sphenopalatine foramen as possible and clips are placed on any large adjacent branches. The creation of an antrostomy is optional.

**Ligation of the anterior ethmoidal artery**

This is performed through an external ethmoidectomy incision. After ligating branches of the angular vein, the incision is continued down to the periosteum which is then incised in the line of the incision. The periosteum is elevated posteriorly, first off the lacrimal fossa, then the lamina papyracea of the ethmoid. The medial orbital periosteum is retracted laterally together with the lacrimal sac and held out of the way by a self-retaining retractor (Talbot). The artery is identified as a funnelling of orbital periosteum into the ethmoid labyrinth at the junction of the medial and superior walls of the orbit about half-way back from the orbital margin. It is coagulated and divided, and the incision closed without drainage.

**Other methods of treatment**

On the assumption that the application of cold stimuli to the nose is likely to reduce or arrest epistaxis, the use of cryotherapy has been advocated to control intractable bleeding (Hicks and Norris, 1983).

More recently therapeutic embolization under selective angiographic control has been carried out with apparent satisfaction (Vanwyck, Vinuela and Heeneman, 1982).

**Unusual causes of epistaxis**

**Osler's disease**

This is a familial hereditary complaint in which sufferers develop prominent telangiectatic formations recognized as red spots on the lips and the mucous membrane of the mouth, especially the tongue, as well as telangiectases on the face and in the nose. The defects in the nose are liable to cause severe epistaxis, and bleeding is rarely from one site alone. The condition may be complicated by the presence of lesions in the gut which may bleed, or arteriovenous malformations in the lungs.

Harrison (1957) has shown that high doses of oestrogen will lessen the frequency and severity of nose bleeding, probably by inducing a squamous metaplasia of the nasal mucous
membrane. However, this form of treatment is inappropriate in male patients, and could well be dangerous if prescribed over a long period of time.

Saunders (1963) advocated excising the mucous membrane of the anterior part of the septum and lateral nasal wall and its replacement by a split-skin graft which is laid on the perichondrium and sewn into position. This treatment is temporarily effective but bleeding usually recurs months or years later. The carbon dioxide laser has been used in recent years to destroy the lesions which are visible in the anterior part of the nasal fossa and, if necessary, access may be improved by resorting to lateral alar incisions. The lesions nevertheless gradually make their reappearance and bleeding eventually recurs. Radiotherapy is also successful for a time in controlling the nose bleeds.

Haemorrhage in this condition may sometimes be unusually severe and may require repeat transfusions to keep up with the blood loss. In some patients the disease runs a relatively mild course, whereas in others it may become increasingly debilitating, with the development of chronic anaemia and hypertension.

Bleeding diatheses

Epistaxis may be a manifestation of a clotting defect, increased fragility of capillaries, or a deficiency in platelets. A history of prolonged bleeding after trauma or dental extraction is suggestive or, alternatively, bruising or bleeding into joints. When suspected, the patient should be stripped and a search made for signs of purpura, bruising and swollen joints; a Hess's test and platelet count are carried out, and the bleeding and clotting times are measured. If required, specific tests for deficiencies of the factors responsible for coagulation can be performed to rule out such diseases as Christmas disease and haemophilia. In elderly subjects Waldenström's macroglobulinaemia should be excluded.

The treatment depends to a large extent on the individual cause of the blood dyscrasia, but in the short term, blood transfusion may be necessary. Particular caution should be exercised in packing the nose of patients with bleeding diathesis as clumsy handling will result in trauma to the mucous membranes, leading to further haemorrhage and nasal packing in such cases may well do more harm than good.

Nasopharyngeal angiofibroma

This arises in male adolescents and is thought, by some, to be a vascular malformation. It is characterized, not infrequently, by severe haemorrhages resulting in anaemia, local sepsis, and debility. The diagnosis is confirmed by arteriography and the correct treatment is surgical removal.