# Arterial epistaxis*

By O. H. Shaheen (London)

In his massive work entitled *The Paradise of Wisdom* (A.D. 850) Al-Tabiri stated that 'nose bleeding is due to the swelling of a vein and its rupture, or perhaps a diminution of the force which confines the blood within that vessel'. It seems extraordinary that 1,100 years later we are still ignorant of such fundamentals as the aetiology of arterial epistaxis. Furthermore, we have come to accept the teachings of our forebears without questioning their validity, probably because the subject is not sufficiently exciting to arouse one's curiosity. Statements such as: 'Hypertension causes epistaxis' or 'The pathological process responsible for vessel rupture is atheroma', are commonplace but inaccurate. A re-appraisal of the more basic aspects of arterial epistaxis and its surgical treatment is therefore timely, and may lead to further discoveries in the future.

## Hypertension and epistaxis

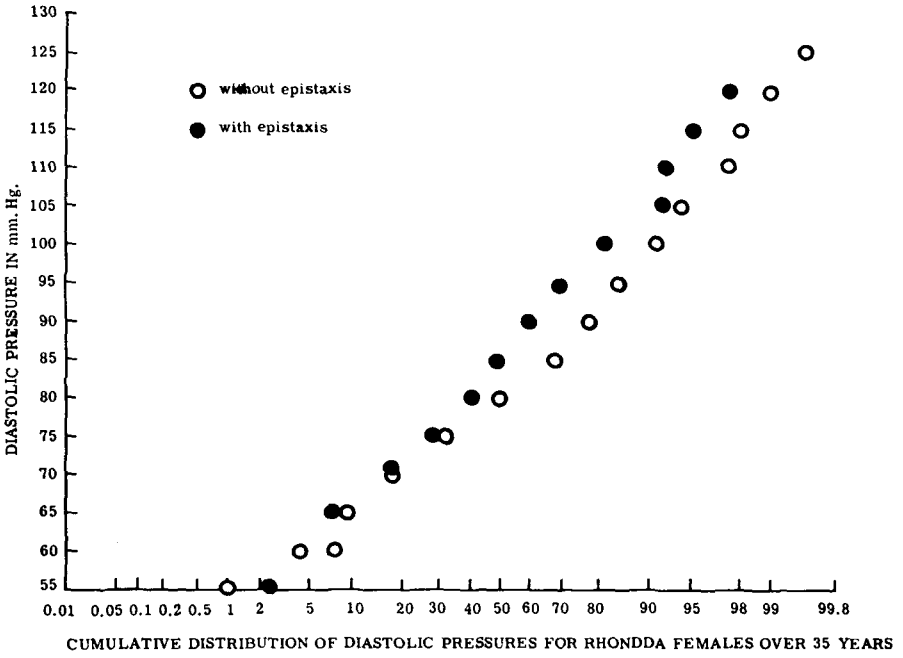
Hypertension has for a long time been held responsible for epistaxis, probably because many hospital patients with epistaxis are found to be hypertensive. Mitchell (1959) specifically investigated the alleged relationship in a retrospective study performed on a hospital population. He compared the blood-pressure levels of patients admitted with epistaxis with those admitted for other nasal disorders such as polypi and septal deflections and found that the former group had significantly higher pressures than the latter. His finding of a 4·5 per cent incidence of epistaxis in a group of hospital hypertensives was put forward as a further argument that hypertension causes epistaxis.

However, this figure is considerably lower than the prevalence of epistaxis in random samples of the population of the Rhondda Fach over the age of thirty-five, which was found to be approximately 13 per cent (Shaheen, 1967). Furthermore, experimental animals made hypertensive by occlusion of one or both renal arteries do not develop epistaxis, nor do animals such as the turkey or giraffe who normally have a high arterial pressure in their heads. In fact, the only animal to develop epistaxis with any frequency is the racehorse, usually during or just after races, and this is thought to be an inherited recessive characteristic (Moorehouse, 1933).

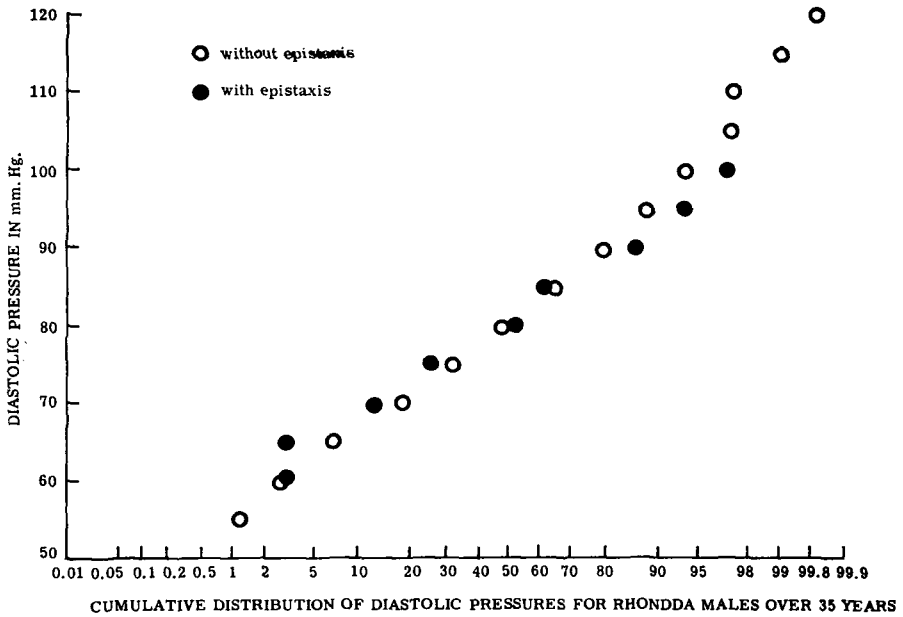
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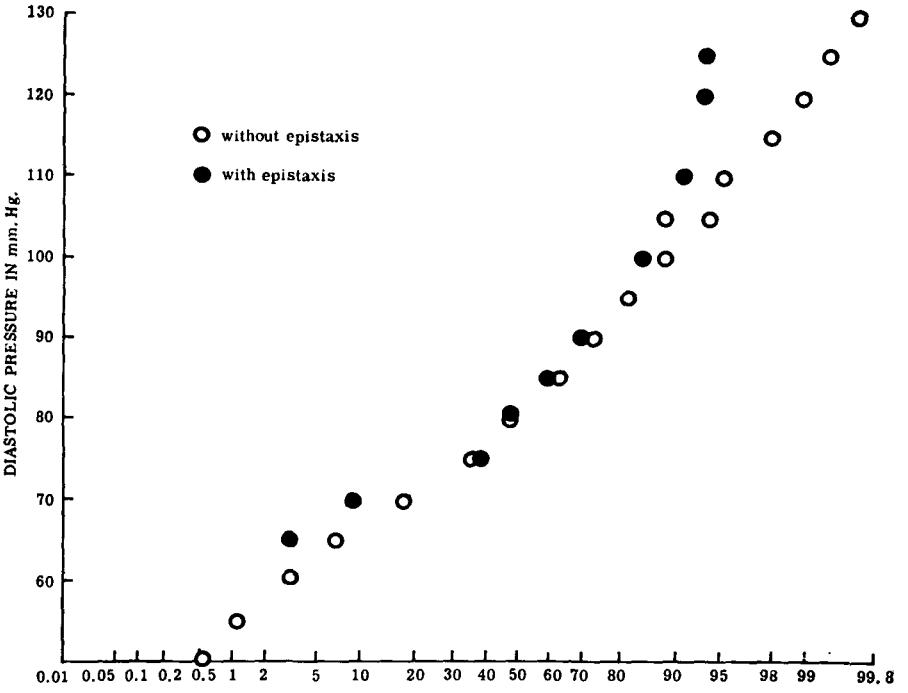

CUMULATIVE DISTRIBUTION OF DIASTOLIC PRESSURES FOR RHONDDA FEMALES OVER 35 YEARS



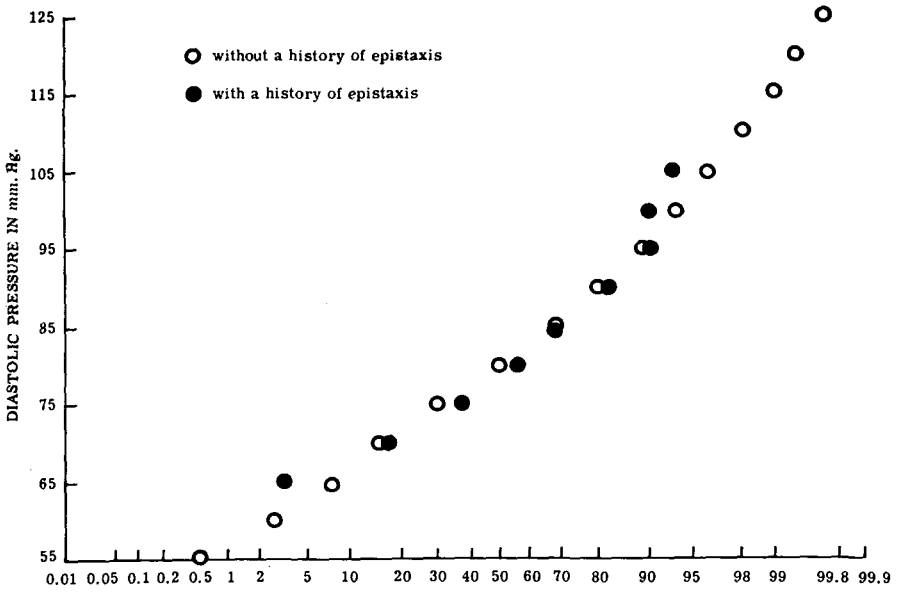

CUMULATIVE DISTRIBUTION OF DIASTOLIC PRESSURES FOR RHONDDA MALES OVER 35 YEARS



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CUMULATIVE DISTRIBUTION OF DIASTOLIC PRESSURES FOR FEMALES OVER THE AGE OF 35 IN THE VALE OF GLAMORGAN




CUMULATIVE DISTRIBUTION OF DIASTOLIC PRESSURES FOR MALES OVER THE AGE OF 35 IN THE VALE OF GLAMORGAN

FIGS. 1, 2, 3 and 4.

The cumulative distribution of diastolic blood pressures of females and males in the Rhondda Fach and Vale of Glamorgan, both with and without a history of epistaxis.



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# O. H. Shaheen

In 1964 a survey by the Epidemiological Research Unit (MRC) investigating the effect of age and environment on the distribution of blood pressures in South Wales provided the writer with the opportunity of looking into the alleged relationship of epistaxis to hypertension. Individuals over the age of five years were selected by random sampling and their first-degree relatives included in the survey as the unit was also interested in the pattern of inheritance of hypertension. Blood-pressure measurements were carried out on 909 subjects in the Rhondda Fach and 1,142 in the Vale of Glamorgan. Subjects were visited in the home by two members of the unit and blood-pressure measurements made under standard conditions. The subjects whose blood pressures were measured were asked two questions: the first related to a history of epistaxis; and the second to its severity by asking if the subject had received medical attention for nose bleeds.

The cumulative distribution of blood pressures was plotted on arithmetical probability paper for subjects of both sexes with a history of epistaxis and those without (Figs. 1, 2, 3 and 4) and the results showed no significant difference between the two groups.

There were twenty-nine individuals over the age of thirty-five who gave a history of epistaxis requiring medical attention. The mean diastolic blood pressure in these subjects was higher than those subjects with milder bleeding, whose averages did not differ significantly from those without a history of epistaxis. The conclusion to be drawn is that hypertension does not influence the prevalance of epistaxis, but the severity of epistaxis may well be related to the blood pressure status of the individual.

In a second investigation, the writer also looked into certain facets of epistaxis in seventy-two subjects over the age of thirty-five years attending hospital for epistaxis. The age distribution is depicted in Figure 5 and the age-diastolic pressure distribution in Figure 6. The figures which are encircled indicate those subjects with arterial pressures in excess of the mean plus one-and-a-half standard deviations for the particular age group and sex. It is immediately apparent that three-quarters of the patients had blood pressures which were lower than this arbitrary yardstick for diagnosing hypertension. The relatively high proportion of hypertensive patients (25 per cent) does not necessarily imply that patients with epistaxis are more inclined to be hypertensive but that patients attending hospital are probably the ones with severe nose bleeds and the severity (not the frequency) of epistaxis may be related to hypertension. An attempt to relate the height of the blood pressure to the severity of epistaxis as expressed by the duration of epistaxis using the coefficient of correlation was however notably unsuccessful. A much more significant correlation was, in fact, found between increasing arterial degeneration in the optic fundus and the severity of epistaxis as measured by the duration using the $X^2$ test ($p \cdot 02$–$\cdot 01$).



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# Arterial epistaxis

## The anatomical sites of arterial epistaxis

It has been customary since Zuckerkandl's (1892) time to subdivide the arterial distribution in the human nose into two main areas which are derived from the internal and external carotid arteries respectively. Clinicians have chosen the middle turbinate as the dividing line on the lateral wall with a corresponding imaginary line of demaracation at the same level on the nasal septum (Weddell *et al.*, 1946; Cahn, 1955). This landmark has allegedly helped the surgeon to select the appropriate vessel for ligation.

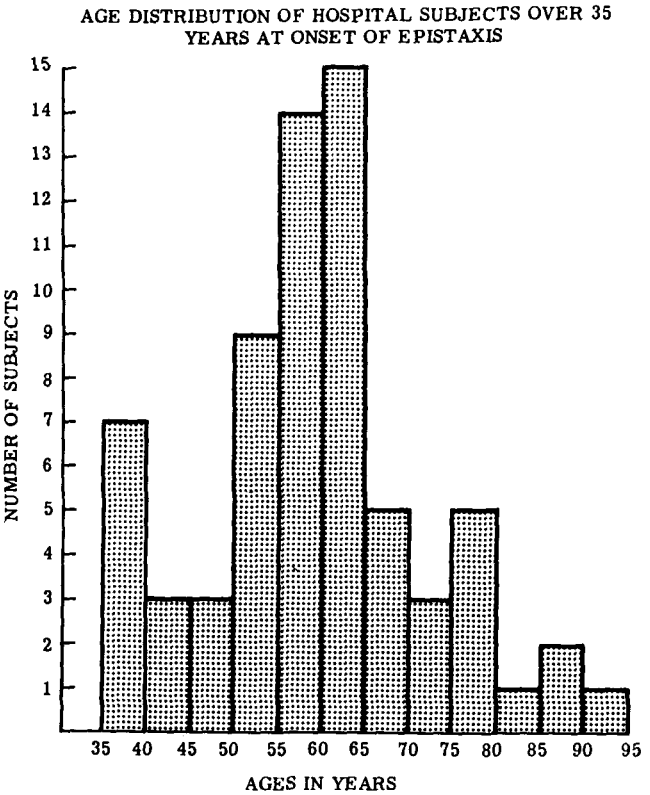



FIG. 5.

The age distribution of subjects over the age of thirty-five attending hospital with epistaxis.

The work of Zuckerkankl (1892) and Burnham (1935) indicates that the blood supply to the middle turbinate is derived exclusively from the maxillary artery (external carotid), and that a vessel from the same source supplies the posterior part of the superior turbinate and meatus with a corresponding vessel on the septum (Fig. 7). If their findings are accurate the area designated as receiving blood from the internal carotid artery must be smaller than generally supposed. The gross disproportion between the



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DIASTOLIC PRESSURES IN mm. Hg.

| AGE GROUPS | TOTALS | 60 | 65 | 70 | 75 | 80 | 85 | 90 | 95 | 100 | 105 | 110 | 115 | 120 | 125 | 130 | 135 | 140 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 35 - 39 | 6 | 1 | | | 1 | | | 1 | | (2) | (1) | | | | | | | |
| 40 - 44 | 1 | | | | | | | | | 1 | | | | | | | | |
| 45 - 49 | 6 | 1 | | | | | 2 | 1 | | (1) | | (1) | | | | | | |
| 50 - 54 | 11 | 1 | | | | | 1 | 4 | | 1 | (1) | (2) | | | | | (1) | |
| 55 - 59 | 14 | | | | | 2 | 1 | 1 | | 5 | | | | (3) | | (1) | | (1) |
| 60 - 64 | 15 | | | | | 2 | 1 | 1 | | 3 | 3 | 3 | 1 | | | (1) | | |
| 65 - 69 | 6 | | | | | 2 | | | | 1 | | | 1 | | | (2) | | |
| 70 - 74 | 4 | | | | | | | | | 1 | | 1 | (1) | (1) | | | | |
| 75 - 79 | 4 | | | | | 1 | | 2 | | 1 | | | | | | | | |
| 80 - 84 | 2 | | | | | | | 1 | | 1 | | | | | | | | |
| 85 - 89 | 2 | | | | | | 2 | | | | | | | | | | | |
| 90 + | 1 | | | | | | | | | 1 | | | | | | | | |
| TOTALS | 72 | 3 | | | 1 | 7 | 7 | 11 | | 18 | 5 | 7 | 3 | 4 | | 4 | 1 | 1 |

AGE - DIASTOLIC PRESSURE DISTRIBUTION FOR MALES AND FEMALES ATTENDING HOSPITAL WITH EPISTAXIS

FIG. 6.

The age-diastolic pressure distribution of subjects over the age of thirty-five attending hospital with epistaxis. The figures surrounded by a circle indicate a pressure in excess of the mean plus one-and-a-half standard deviations for the age and sex in question.

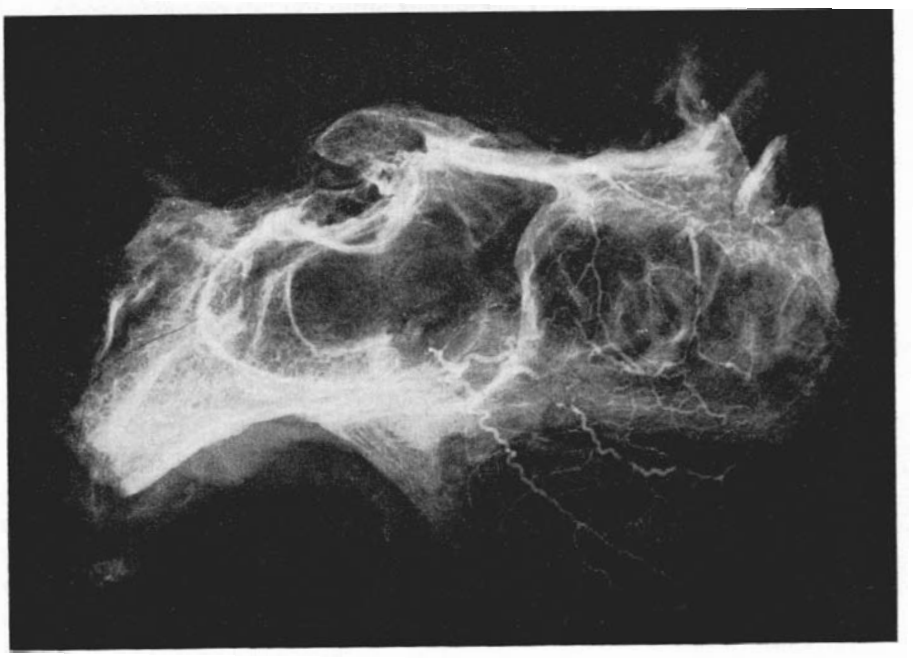


FIG. 7.

A radiograph of the upper posterior part of the nasal cavity of a cadaver whose arteries were injected with barium. The two main subdivisions of the long nasopalatine artery are seen, from the upper branch of which is a vessel which supplies the superior turbinate and anastamoses with the posterior ethmoidal artery.

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diameters of the anterior ethmoidal artery and the sphenopalatine at their points of entry into the nose (Figs. 8 and 9) would tend to corroborate this contention. This must mean, therefore, that the chances of epistaxis originating from the ethmoidal area must be very small.

In the seventy-two patients over the age of thirty-five years personally examined, the site of haemorrhage was determined in approximately 50 per cent of cases. Figure 10 depicts diagrammatically the position of the bleeding point in those instances in which it was possible to identify it with certainty, many of which were arterial spurters. There were no cases of bleeding from the lateral nasal wall, and few were found to originate from the anterior ethmoidal area.

### Arterio-arterial anastamoses

The mechanics of the arterial circulation in the nose have never been properly studied. We lack knowledge regarding the behaviour of the arterial circulation and the artero-arterial anastamoses between the internal and external carotid circulations in the nose. McDonald and Potter (1951) demonstrated the presence of opposing heads of pressure between the carotid and vertebral circulations in the posterior communicating artery of the circle of Willis of rabbits. They injected methylene blue into the internal carotid artery or the vertebral artery of rabbits, and examined the sharp interface of dyed and undyed blood in the posterior communicating artery. Their findings led them to believe that a balance of pressures exists between the two opposing systems, but that lowering the arterial pressure in one or other homolateral vessel causes the interface of blood to be displaced. Thus, if dye is injected into the internal carotid artery the sharp interface normally occurs halfway along the posterior communicating artery of the circle of Willis, but if the homolateral vertebral artery is simultaneously occluded, the interface moves in a backward direction into the basilar artery. Similarly, occlusion of one carotid or vertebral artery caused dye injected on the contralateral side to cross the mid-line of the circle of Willis. The arterial circulation of the nose may behave in an analagous way. Lowering the pressure in the external carotid artery by ligating it should cause a displacement of blood from the upper internal carotid area of the nose to the lower external carotid area across the anastamoses linking the two areas. This state of affairs could explain instances of persistent bleeding after external carotid ligation, many of which were thought at the time to be examples of ligating the wrong vessel.

The injection of a dye into the carotid arteries of live humans was used by the speaker as a method of outlining the extent of individual arterial contributions to the nose, and also studying the speed with which intranasal anastamoses function. Twenty-four patients with advanced malignant neoplasms of the head and neck not affecting the nose or nasopharynx



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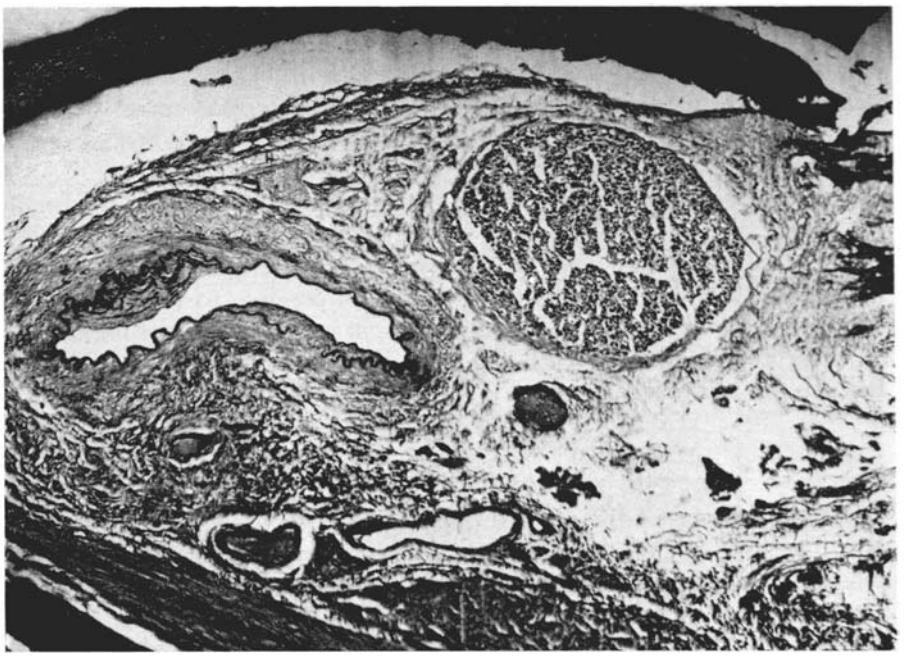


FIG. 8.
Anterior ethmoidal artery and nerve. Elastic—Van Gieson (×80).

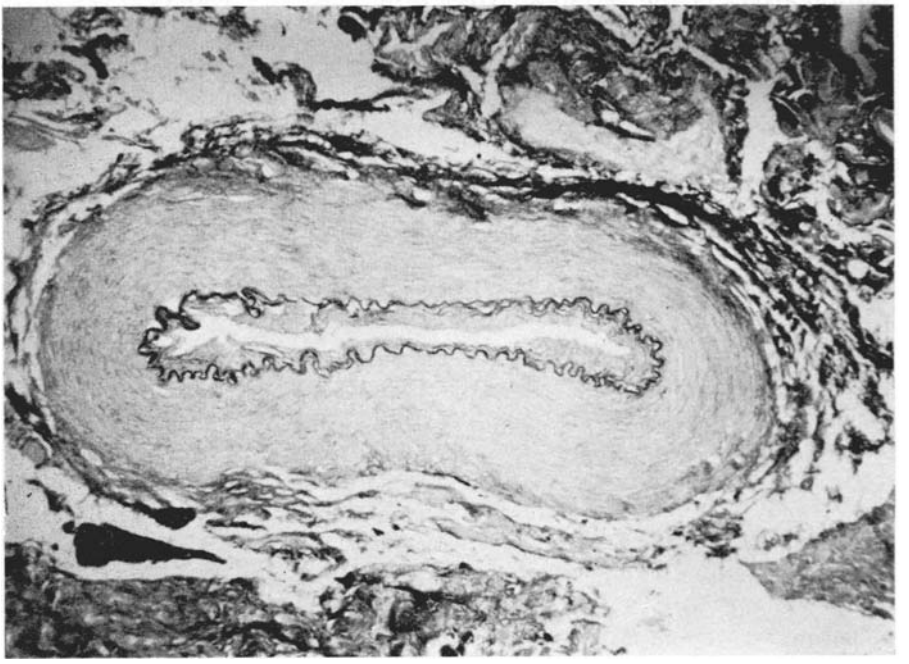


FIG. 9.
Nasapalatine artery at its point of entry in the nose. Elastic—Van Gieson (×80).



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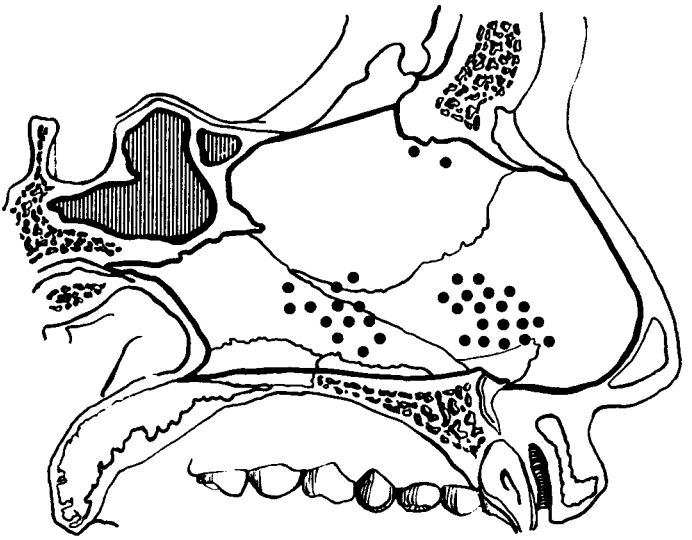


FIG. 10.
The position of identifiable bleeding points.

were selected for study as they were to undergo open regional intra-arterial cytotoxic therapy, the agent in question being ethoglucid. For this technique a tracer dye such as methylene blue or disulphine blue is slowly injected into an artery in order to determine which vessel supplies the area of the neoplasm. The procedure was simply adapted to the exercise in question by looking into the uncocainized nose after injection of the dye. The procedure was carried out under general anaesthesia, the neck being opened to expose the carotid bifurcation and the external and internal carotid arteries. In a small number of cases, injection of dye was performed by retrograde cannulation of the superficial temporal artery. In eight cases, dye was injected into the internal carotid artery just above the bifurcation. In two of these, the injection of the dye was not accompanied by occlusion of the external carotid artery, and in both cases although dye appeared in the periorbital skin no dye was actually seen in the nose. In six cases, injection into the internal carotid was accompanied by simultaneous occlusion of the external carotid artery simulating the effect of ligation, and in all six there was an immediate and dramatic suffusion of the upper face with dye and a down-flow of dye from the cribiform plate to involve the upper third to one-half of the septum and lateral nasal wall (Fig. 11). In thirteen cases dye was introduced into the external carotid artery. In eight of these the facial artery was left patent, and in three of these cases the whole of the septum and lateral nasal wall took up the dye although staining was less intense above the level of the middle turbinate (Fig. 12). In the remaining five cases the dye stained the lateral wall and septum two-thirds



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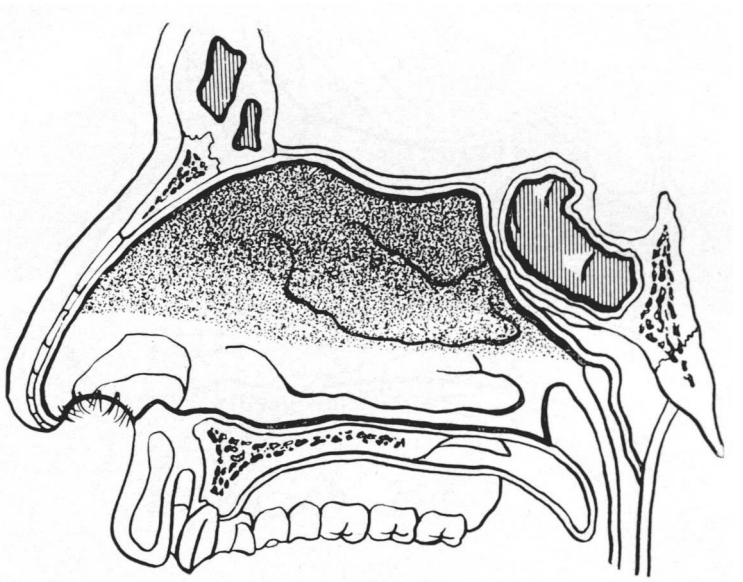


FIG. 11.

A diagram of the right lateral nasal wall depicting the down-flow of dye resulting from the injection of dye into the internal carotid artery with simultaneous occlusion of the external carotid artery. (Six cases.)

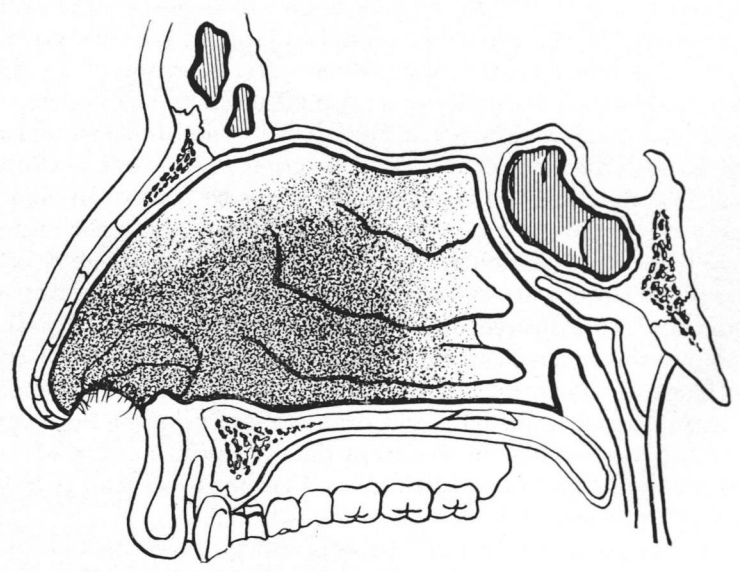


FIG. 12.

A diagram of the right lateral nasal wall depicting the extent of dye suffusion after injecting dye into the external carotid artery without occluding the facial artery. (Three cases.)



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of the way up (Fig. 13). In five cases of external carotid injection, the facial artery was occluded. In these the whole of the septum and lateral nasal wall took up the dye, although the intensity of staining was less above the level of the middle turbinate (Fig. 14). In the three cases in which dye was

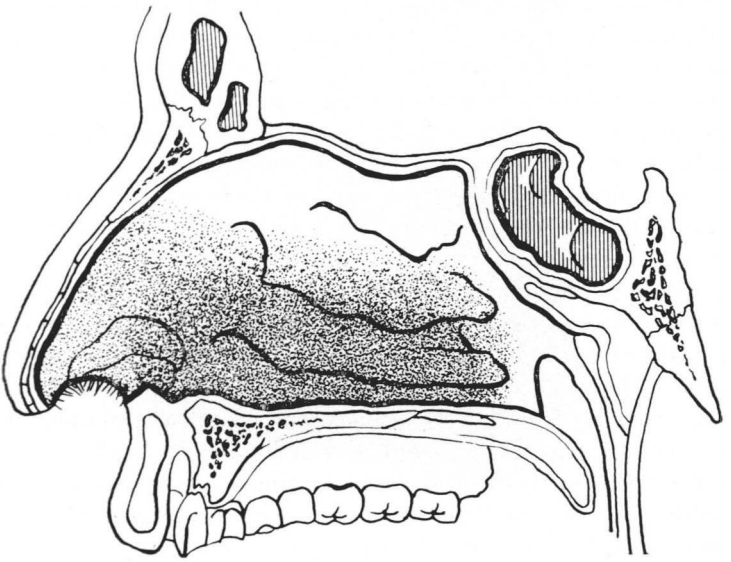


FIG. 13.
A diagram of the right lateral nasal wall depicting the extent of dye suffusion after injecting dye into the external carotid artery without occluding the facial artery. (Eight cases.)

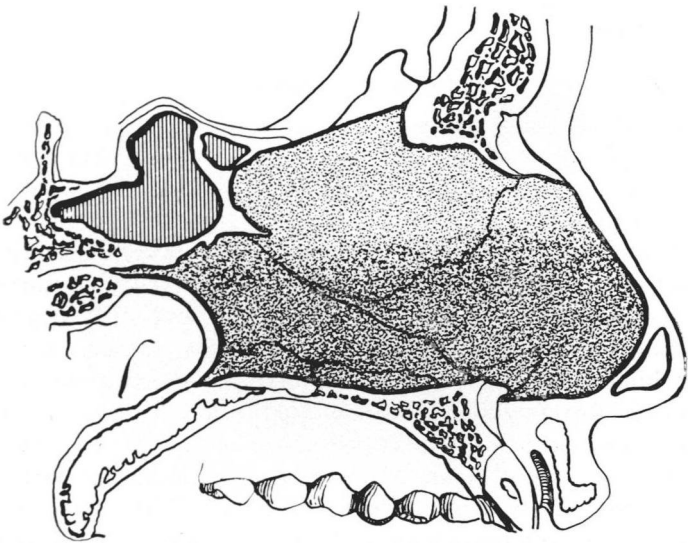


FIG. 14.
A diagram of the right side of the nasal septum depicting the extent of dye suffusion after injecting dye into the external carotid artery with the facial artery occluded. (Five cases.)



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injected into the internal maxillary artery by retrograde cannulation of the superficial temporal artery, the entire nasal cavity with the exception of the cribiform plate took up the dye well.

Clearly, these investigations show that the ethmoidal arteries supply only a very small area of the nose. They also show that occlusion of the external carotid artery causes an immediate displacement of blood from the internal carotid distribution to the external carotid of the nose, sufficient to explain those instances of failed carotid ligation. The effect of occlusion of the facial artery on injections of dye into the external carotid was probably to draw blood from the orbital branches of the internal carotid into the face, so that dyed blood from the lower half of the nose replaced blood which would normally fill the ethmoidal areas and which was being diverted to the face.

The effect of intranasal anastamoses has also been observed by the writer in two cases of maxillary artery ligation for epistaxis. In one, a male of fifty-two years, the bleeding point was identified on the table immediately prior to ligation by injecting dye into the maxillary artery. The dye was seen emerging from the bleeding point which was unquestionably in the territory of the external carotid low down and far back on the septum. However, even after ligation bleeding continued for some time afterwards, presumably via the anastomoses joining the ethmoidal with the sphenopalatine vessels. In a second case of maxillary artery ligation in a female of forty-two years, the clip on the distal half of the divided internal maxillary artery slipped off accidentally. Vigorous retrograde bleeding from this half of the vessel was observed, indicating that blood flow was proceeding in the reverse direction.

### The pathology of nasal arteries

Earlier mention was made of the apparent relationship between the duration of arterial epistaxis and vessel degeneration in the optic fundus. The writer was interested to find out exactly what the nasal vessels of subjects over the age of thirty-five years were like, and whether the vessels of subjects with a history of epistaxis were in any way distinctive or not.

Naso-ethmoidal blocks (Figs. 15 and 16) were taken from thirty middle-aged and elderly subjects (twenty-one males and nine females) post-mortem. Subjects were divided into three groups, namely: normotensive subjects who had not suffered from epistaxis, of which there were eleven; hypertensive subjects who had not suffered from epistaxis, of which there were eight; and subjects with a history of epistaxis irrespective of the blood-pressure status of the individual (eleven cases). Information about a history of epistaxis was obtained from relatives, the patient's general practitioner, and from hospital records. The diagnosis of hypertension was made from the hospital records and the weight of the heart at post-mortem. The blocks were decalcified in 5 per cent formic acid and decalcification



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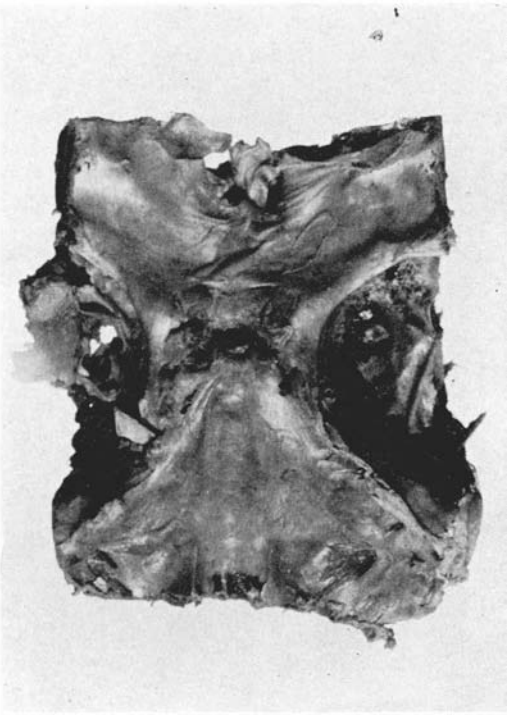


FIG. 15.
A naso-ethmoidal block taken at post-mortem seen from above. The pituitary fossa is in the centre for orientation.

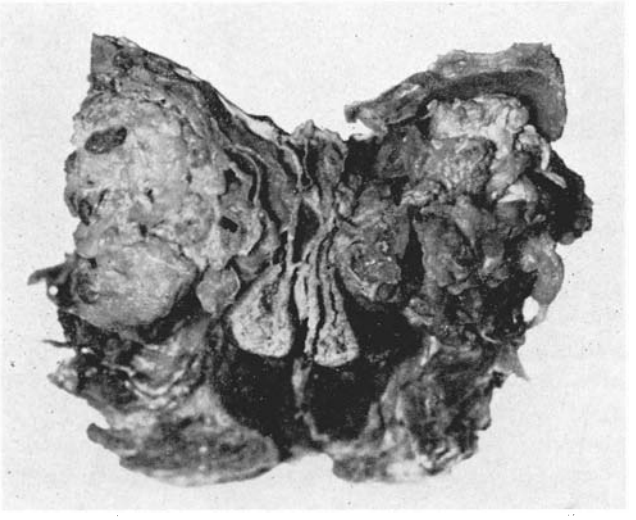


FIG. 16.
A naso-ethmoidal block taken at post-mortem seen from the front. The nasal fossae, turbinates, and the medial half of the orbits are seen.



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tested for chemically. The maxillary and anterior ethmoidal arteries of each side were dissected out and examined microscopically, as were representative full thickness portions of the nasal interior from various sites designated. The most striking finding in this investigation was the change in the muscular tunica media of arteries of intermediate and small size. This can best be described as a fibrous change varying in degree from an interstitial fibrosis to a segmental collagenous replacement of the muscle, amounting in some cases to half the circumference of the artery (Figs. 17, 18, 19 and 20). These changes were not confined to any one segment of the nose, being

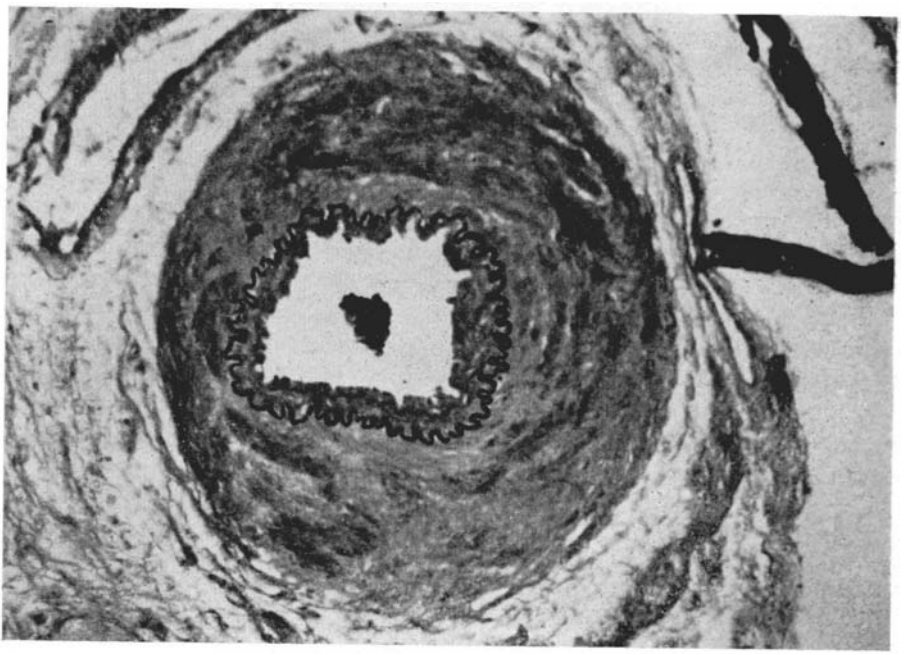


FIG. 17.
A transverse section of a nasal artery showing interstitial fibrosis of the tunica media. Elastic—Van Gieson. (×110.)

equally distributed amongst the various sites sampled. A system of grading the severity of this change was adopted (Fig. 21) which suggested that the more severe changes were seen in subjects with a history of epistaxis. It would be naive to say that this fibrous replacement of the muscle is the cause of arterial rupture, but it may well be the reason for the persistence of arterial epistaxis if the muscle in the arterial wall is incapable of contracting properly. Actually the cause of rupture is probably a dissecting aneurysm of one of the septal arteries as seen in Figure 22 which is taken from a patient who succumbed shortly after an epistaxis, and whose nasal cavity was serially sectioned.



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FIG. 18.

A transverse section of a nasal artery showing segmental loss of muscularity with replacement by collagen in about a third of the vessel-wall circumference. Elastic—Van Gieson. (×90.)

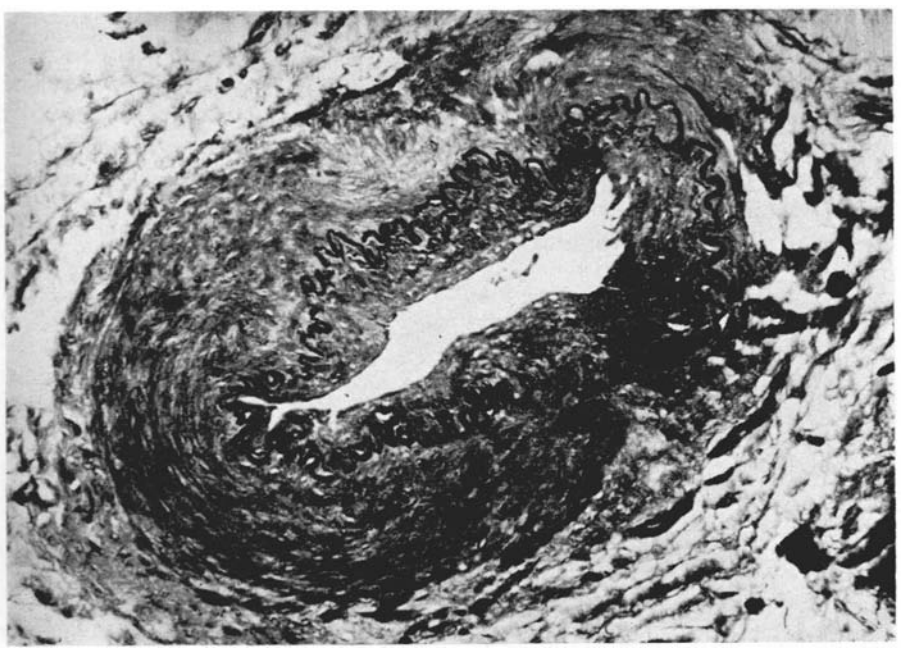


FIG. 19.

A transverse section of a nasal artery showing segmental loss of muscularity with replacement in about one-half of the vessel-wall circumference. Elastic—Van Gieson (×128.)

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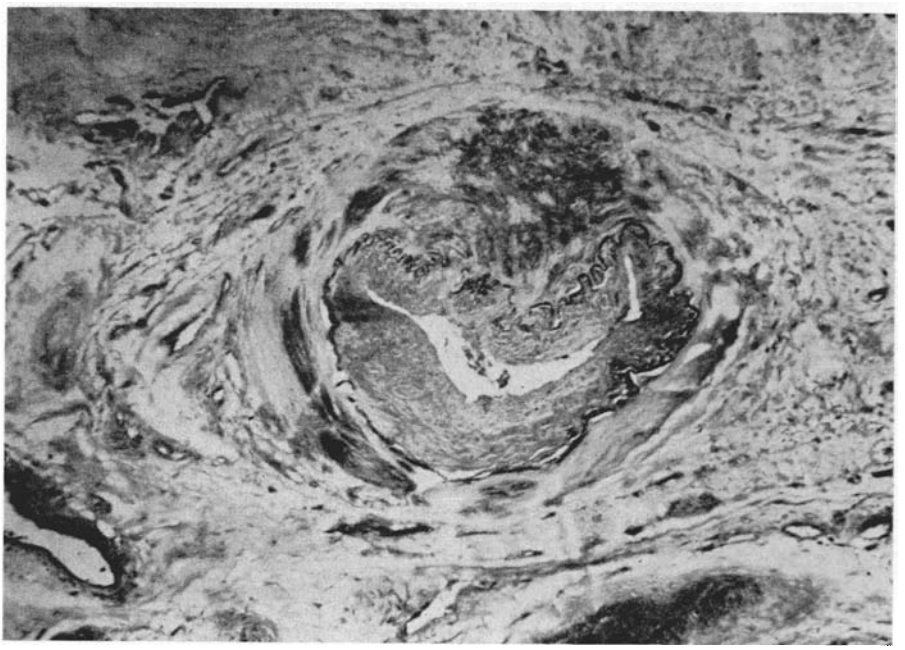


FIG. 20.

A transverse section of a nasal artery showing loss of three-quarters of the muscle coat and replacement by collagen. Elastic—Van Gieson. (×80.)

In the case of the maxillary and anterior ethmoidal arteries true atheroma was rarely found in this study, but calcification of the internal elastic lamina or tunica media known as Monckeberg's sclerosis was frequently seen. This change may be yet another factor responsible for the persistence of nose-bleeds by creating a high local pulse pressure in the vessel which is transmitted to smaller arteries which in turn are incapable of closing down once bleeding has occurred.

NORMOTENSION - NO EPISTAXIS

| Grade I | Grade II | Grade III | Grade IV |
|---|---|---|---|
| 5 | 2 | 2 | 2 |

HYPERTENSION - NO EPISTAXIS

| Grade I | Grade II | Grade III | Grade IV |
|---|---|---|---|
| 3 | 4 | 1 | 0 |

EPISTAXIS

| Grade I | Grade II | Grade III | Grade IV |
|---|---|---|---|
| 0 | 5 | 3 | 3 |

FIG. 21.

The grading of changes in the tunica media of nasal arteries examined after death in these groups of subjects.



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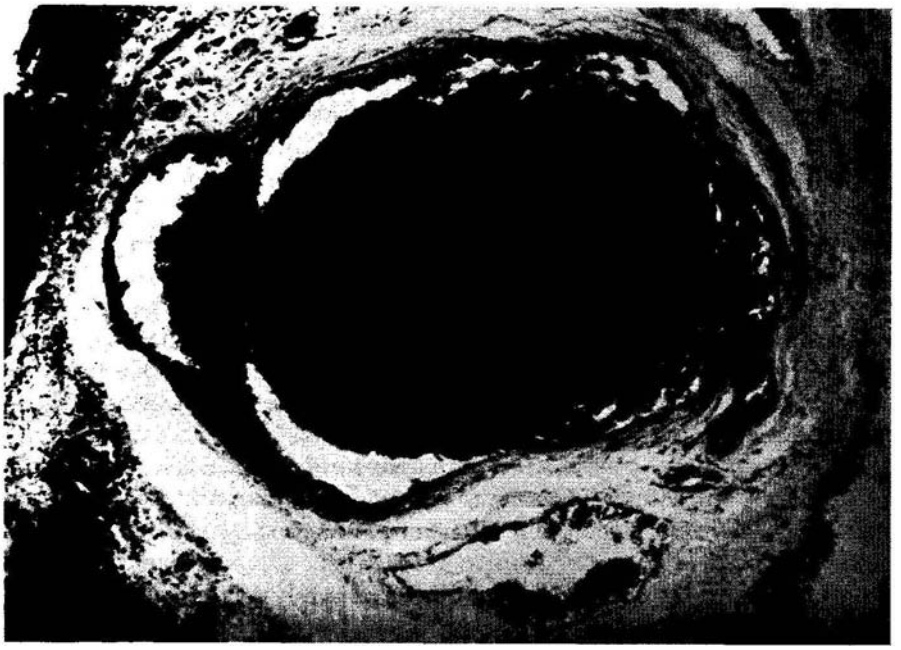


FIG. 22.

A dissecting aneurysm of the nasopalatine artery of a patient suffering from epistaxis. Haematoxylin and Eosin ( × 120.)

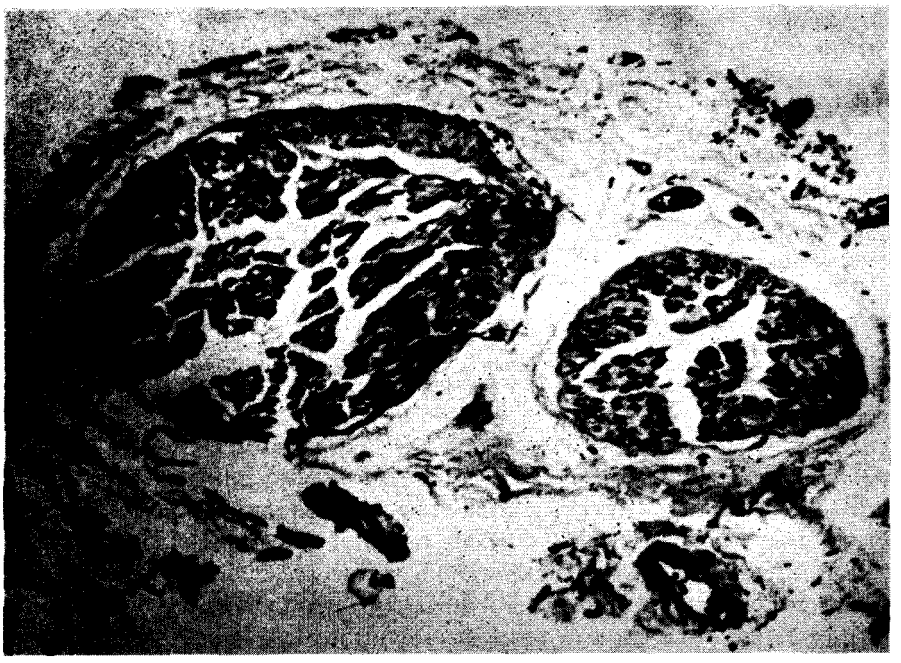


FIG. 23.

A periosteal cuff passing through the anterior ethmoidal foramen and seen in transverse section showing two ethmoidal nerves, the artery being absent. Elastic—Van Gieson. ( × 120.)

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**Summary**

If arterial ligation is contemplated and the site of bleeding has not been identified it is reasonable to ligate the external carotid or maxillary artery, on the basis that this vessel supplies the major part of the nose. If bleeding persists in spite of ligation, this does not mean that the wrong vessel has been tied off but that arterial anastomoses are allowing blood to seep from the ethmoidal to the sphenopalatine area of the nose. If the anterior ethmoidal artery is to be ligated it is worth remembering that in 14·3 per cent of cases the anterior ethmoidal artery is absent unilaterally and in 2·4 per cent of cases absent bilaterally (Shaheen, 1967). The presence of a foramen with a periosteal cuff going through it does not necessarily mean that there is an artery present (Fig. 23). Finally, the blood vessel changes which occur and which are responsible for the persistence of nose bleeds in the elderly are a collagenous change in the muscle coat of medium and small arteries and calcification (not atheroma) in the larger feeding vessels.

REFERENCES

AL-TABIRI, A. I. R. (850) *Paradise of Wisdom*, book 4, section e, chapter 9 (British Museum).

BURNHAM, H. H. (1935) *Journal of Laryngology and Otology*, **50**, 569.

CAHN, H. L. (1955) *Archives of Otolaryngology*, **61**, 641.

MCDONALD, D. A., and POTTER, J. M. (1951) *Journal of Physiology* (London), **114**, 356.

MITCHELL, J. R. (1959) *British Medical Journal*, **1**, 25.

MOOREHOUSE, E. (1933) *Bloodsth. Breed. Rev.*, **22**, 82.

SHAHEEN, O. H. (1967) 'Epistaxis in the Middle-Aged and Elderly'. Thesis for the degree of Master of Surgery in the University of London.

WEDDELL, G. *et al.* (1946) *British Journal of Surgery*, **33**, 387.

ZUCKERKANDL, E. (1892) *Normale und Pathologishe Anatomie der Nasenhöle*, **1**, 128. W. Braumuller, Wien und Leipzig.

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