EPISTAXIS MORTALITY

THE PLACE OF BLOOD TRANSFUSION IN ITS PREVENTION

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NOSE bleeding ceases spontaneously in most cases and is usually a trivial complaint (Hallberg, 1952). It is however a potentially fatal condition and death probably occurs more often than is generally supposed. Although there has from time to time been considerable discussion about the management of epistaxis, this has mainly dealt with the surgical measures necessary to stop the bleeding. Less attention has been paid to the management of the systemic complications of severe epistaxis, i.e. hypotension and anæmia, and to the place of transfusion in their correction. In this paper seven fatal cases are described, with note of the post-mortem findings in five of them. Cardiovascular factors were found to be of considerable importance in all of them.

Case Reports

CASE I.

Mrs. E.H., aged 75 years. This patient was first seen by Mr. J. W. Cope at the Royal National Throat, Nose and Ear Hospital in November 1955. She gave a history of recurrent epistaxis of recent onset and was admitted to hospital for one day. The exact site of bleeding could not be seen but after the nose was packed it ceased. Subsequent epistaxis required frequent readmission. Her diastolic blood pressure was always less than 100 mm. Hg, and there were no signs of systemic disease. In February 1956, her hæmoglobin was found to be 48 per cent. (Haldane) and she was given packed red cells from one pint of blood. Her hæmoglobin rose to 64 per cent. (Haldane) and epistaxis did not recur. A fortnight later she suddenly became stuporous and showed signs of acute heart failure. She died on the following day.

Post-mortem findings: There was extensive atheroma of the coronary arteries and the anterior descending branch of the left coronary artery was blocked by thrombus which had caused a large myocardial infarct. Microscopy showed this to be recent in origin. No local nasal lesion found.

CASE II.

Miss M.G. aged 63 years was a patient of Dr. J. H. Sheldon at the Royal Hospital, Wolverhampton, admitted in March 1956, having been

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bleeding severely from the left nostril for an hour. There was no relevant past history. B.P. 130/90. A transfusion on one pint of blood was given and the nose bleed stopped. Eleven days later the nose bleeding recurred and the blood pressure fell to 90/60. Hæmoglobin at this time was 75 per cent. (Haldane). The patient appeared to recover from this episode, but two days later she suddenly collapsed and died three minutes later.

Post-mortem findings: In the heart there was marked left ventricular hypertrophy. The aortic valve was bicuspid and completely calcified with fusion of the cusps at either end. The resultant stenosis must have been of long standing. The other valves were normal and the coronary arteries healthy. No local lesion found in the nose.

CASE III.

Mr. H.G. aged 22 years was admitted to New Cross Hospital, Wolverhampton under the care of Dr. E. J. Blair in July 1956 with a severe epistaxis. He also complained of headache and dizziness for the two previous days. His blood pressure was 210/110. He had albuminuria and a blood urea of 460 mg. per cent. After admission he became comatose and died on the following day.

Post-mortem findings: The brain showed petechial pontine hæmorrhages. The lungs were the site of an acute purulent bronchitis. Both kidneys showed an extreme degree of hydronephrosis. The surfaces were grossly scarred and little renal parenchyma remained. The ureters were both greatly dilated and their openings into the bladder were large and incompetent. No organic obstruction present. It was considered that these lesions were congenital. No local lesion found in the nose.

CASE IV.

Mr. E.P. aged 65 years was admitted to the Royal Hospital, Wolverhampton in October 1956, under the care of Mr. W. L. Thomas following a severe nose bleed. There was no relevant previous history. Blood pressure 180/120. The nose was packed and morphine given but the bleeding could not be stopped despite repacking. A transfusion of ¾ pint of blood was given three days later but on the following day the patient became comatose and died.

Post-mortem findings: There was some coronary atheroma but no other significant lesions. Death was considered to be due to hæmorrhage. No lesion was found in the nose.

CASE V.

Miss A.P., aged 87 was first seen by Dr. C. Evans at St. Charles' Hospital, London in July 1958 when her nose had been bleeding for two days. Her blood pressure was 150/80 and hæmoglobin 58 per cent. (Haldane). She was given morphine and both sides of her nose were packed.

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The bleeding stopped and she was sent home three weeks later taking iron tablets and with a hæmoglobin of 63 per cent. (Haldane). Two months later she was again admitted with epistaxis. Her blood pressure was 115/60 and hæmoglobin 53 per cent. (Haldane). Her nose was packed and one pint of blood given. Bleeding stopped and she was discharged after a week in bed. Two weeks later she returned with a further hæmorrhage which did not cease after three days in hospital. Her hæmoglobin was at this time 46 per cent. (Haldane). A bleeding point on the left side was found and cauterized and one pint of blood was transfused and her hæmoglobin rose to 67 per cent. (Haldane). On the next day, however, another hæmorrhage occurred and the blood pressure fell to 90/50. The patient showed marked mental deterioration and died three days later.

Post-mortem findings: There was an acute bronchitis but no other significant lesion. It was considered that death was due to hæmorrhage combined with the effect of a pre-existent post-hæmorrhagic anæmia.

Case VI.

Mr. S.R. aged 60 years was admitted to St. Charles' Hospital, London under the care of Dr. Hinds-Howell in December 1958. His nose had been bleeding for most of the day and had not been staunched by packing. Blood pressure 146/90. An oozing area on the septum was cauterized and a paraffin pack was inserted. Two days later the nose was still bleeding and further cauterization was carried out. The bleeding stopped and the hæmoglobin at this time was 67 per cent. (Haldane). Four days later the patient complained of a severe pain in the chest radiating down both arms. He was very breathless and his blood pressure fell to 110/60. A diagnosis of coronary thrombosis was made. The patient died two days later. A post-mortem examination was not performed.

Case VII.

Mrs. E.A. aged 88 years was admitted to St. Charles' Hospital under the care of Dr. C. Evans having had intermittent epistaxis for some weeks. Plugging of the nose had not stopped the bleeding. On admission the patient was pale and confused. There was a little ankle œdema. Blood pressure 160/90. Hæmoglobin 36 per cent (Haldane). A pack was inserted into the nose and bleeding stopped. Two days after admission a transfusion of one pint of blood and one pint of packed red cells was given. The patient remained rather confused and later became semi-conscious and incontinent of urine. She died five days after admission. Death was considered to be due to anæmia following hæmorrhage. A post-mortem examination was not performed.

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Epistaxis Mortality

Epistaxis Admissions to Hospital

An attempt has been made to assess the potentially dangerous factors in epistaxis by reviewing one hundred consecutive cases admitted specifically for this cause to the Royal National Throat, Nose and Ear Hospital. Only cases considered severe enough to require admission have been included. Patients in whom the bleeding followed some surgical manœuvre have not been included, nor have those admitted for some reason other than the severity of the bleeding, e.g. children requiring general anæsthesia to allow cautery of the nose.

The age distribution of the group is shown in Table I. The majority of cases occur in middle life or later. There were 53 males and 47 females. There was less than one week's history of nose bleeding in most cases, but eight cases had been bleeding recurrently for a year or more (Table II). One patient in the group died in hospital (Case I).

TABLE I.

AGE DISTRIBUTION OF EPISTAXIS ADMISSIONS TO HOSPITAL

Age (years)	under 21	21-30	31-40	41-50	51-60	61-70	over 70	TOTAL
No. of cases	7	4	7	8	24	24	26	100

TABLE II.

DURATION OF SYMPTOMS IN EPISTAXIS ADMISSIONS.

Duration	1 day or less	1 day-1 week	1 week-1 month	1 month-1 year	1 year-or more	No record	TOTAL
No. of cases	26	39	16	10	8	1	100

A hæmoglobin estimation was performed on admission in 68 cases and a variable degree of anæmia was found in most of them (Fig. 1). In 17 of the 68 cases in which the hæmoglobin was measured it was less than 60 per cent. (Haldane). The presence of anæmia was not related to the duration of symptoms, nor to the age of the patient. The blood pressure was measured on admission in 84 cases and of these 30 had a diastolic pressure greater than 90 mm. Hg. In 8 cases the diastolic blood pressure was greater than 110 mm. Hg.

Five patients gave a history of cardiac disability other than coronary artery disease, and two patients had suffered from myocardial ischæmia. Two patients had acute attacks of coronary thrombosis in hospital following their epistaxis. In one case this was the cause of death. In both these cases bleeding necessitated blood transfusion.

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Blood transfusion was given as part of the treatment in 9 cases. All these patients had a hæmoglobin of less than 60 per cent.; 5 were hypertensive. They were all over 50 years old. Two or three pints of blood were given in all cases except the one which later proved fatal.

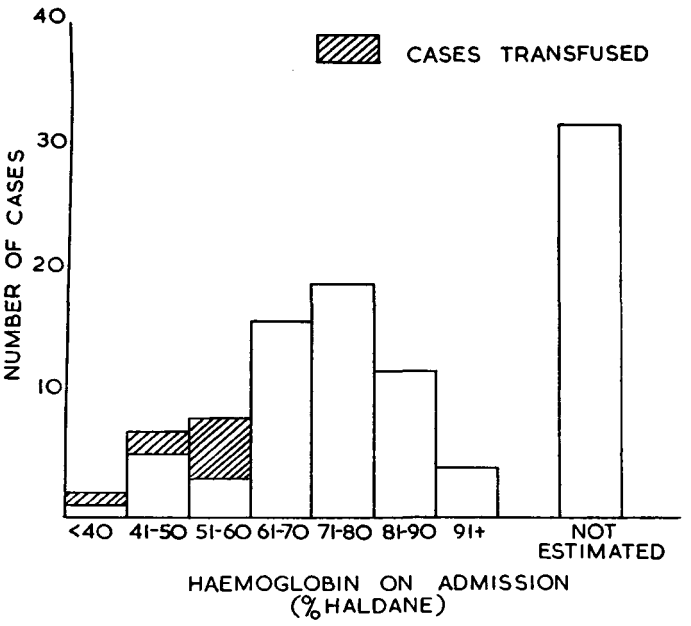


FIG. 1.

Discussion

There is little doubt that the mortality from epistaxis has been seriously underestimated. In the nine years from 1950 to 1958 the Registrar General's records show only 8 deaths from this condition in England and Wales. During a much shorter period, however, we have known of seven fatal cases occurring in only 4 hospitals. A similar impression of the frequency of fatal epistaxis was gained by Collier (1948) who states she had known of 5 such cases.

The reason for this discrepancy is probably that in many cases the immediate cause of death is a complication of the epistaxis, e.g. coronary thrombosis; or there may be a pre-existing disease, most commonly hypertension. These are certified as the cause of death and the epistaxis is not recorded.

An examination of the literature suggests that the majority of these cases are not published. It has been possible to trace only 5 case reports of fatal epistaxis. Of these, the bleeding was secondary to leukæmia (Bourdon, 1952), rupture of a carotid aneurysm (Rasquin, 1949) and

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Osler's disease (Siguier, 1954). One developed myocardial infarction due to post-hæmorrhagic hypotension (Bean, 1937) and the fifth patient died of multiple cerebral hæmorrhages following carotid ligation for otherwise uncontrollable bleeding (Hallberg, 1952).

In a condition which is as common as epistaxis, and is so often trivial and self limiting it is difficult for the clinician to pick out those few cases which may develop serious complications, and treat them appropriately. Three factors seems to us important in determining a serious outcome. The first is the degree and duration of post-hæmorrhagic hypotension; the second is the presence of pre-existing heart disease, especially coronary insufficiency; and the third is the presence of anæmia.

Sudden reduction of the blood pressure is well known to be dangerous. Where the blood pressure is high to begin with, even reduction to normal levels can be damaging. Master, *et al.* (1950) analysed 103 cases of acute hæmorrhage and found 59 to show clinical or E.C.G. evidence of coronary insufficiency. Among 13 of their cases which were examined post-mortem 4 showed myocardial necrosis without coronary occlusion. Actual thrombosis is also a recognized complication of profound hypotension. Blumgart *et al.* (1941) found that coronary occlusion was due to hypotension in 11 out of 450 cases, and they emphasize the importance of preventing under any circumstances a decrease of blood pressure in cases of coronary sclerosis. Two out of the 100 Royal National Throat, Nose and Ear Hospital patients developed a coronary thrombosis while in hospital, and one of these died (Case I).

The presence of heart disease in a patient who has a severe epistaxis might be expected to increase the risk, and in fact all the fatal cases but one were suffering from severe cardiovascular disease.

Anæmia of some degree is common in patients with epistaxis (Fig. 1) and may be profound. In anæmia the oxygen carrying capacity of the blood is reduced, with a consequent reduction in the repiratory reserves of the tissue. This is especially important in the myocardium, where signs of disturbed function due to anoxia are often seen. Pickering and Wayne (1934) found anginal symptoms in 8 out of 25 anæmic patients, and Wood (1956) observed E.C.G. changes of an ischæmic character in a similar proportion of cases. Friedberg (1956) is of the opinion that even myocardial infarction can occur due to anæmia alone. It is clear that, if hæmorrhage with consequent hypotension occurs in the presence of anæmia, the oxygen supply to the myocardium becomes critical and infarction is likely to occur.

When more than one of these three factors is present in the same patient the risks will be greater. When all three are present there should be considerable cause for alarm.

The systemic effects of hæmorrhage are in no way related to the site of the bleeding, and the results of a severe epistaxis may be as dramatic

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as those following a hæmatemesis. The dangers of hypotension and anæmia in the latter condition are well recognized and blood transfusion is the "sheet anchor of treatment" (Hunt, 1957). This should be equally true in the management of severe epistaxis.

In recent years several authors have emphasized the importance of prompt replacement of blood lost by severe nose bleeding, particularly in patients already suffering from hypertension or coronary disease (Collier, 1948), (Keogh, 1952). In spite of this, current teaching in Otolaryngology does not appear to accept this view. The myth that epistaxis is in some way beneficial to the hypertensive patient ("Nature's safety valve"), still survives in the most recent British textbooks. Negus (1955) says: ". . . In conditions of high arterial tension bleeding from the nose is looked upon as salutary, and not be checked too hastily", and Milling (1953) teaches that: ". . . Epistaxis, particularly in old people with degenerate vessels also requires a guarded prognosis, because if the flow of blood be too promptly arrested, one of the cerebral vessels may be the next to give way." The concern is with the danger of stopping the bleeding too quickly. Neither author mentions the possibility of complications due to hypotension, and only a brief phrase refers to the remote necessity of blood transfusion. Most of the other standard British textbooks present similar views.

In any patient suffering from severe epistaxis it is important to know if heart disease is present. A careful history, paying special attention to such symptoms as angina or dyspnœa on exertion, and past history of coronary disease as well as a full examination of the cardiovascular system should be routine. If there are any positive findings the clinician can then be on his guard.

The other two danger factors, anæmia and hypotension, are both readily treated by blood transfusion. Where fall in blood pressure is the main indication whole blood should be used; if anæmia is being treated packed cells are more appropriate, particularly if heart disease is also present. If these factors are treated vigorously whenever they occur it should be possible to save patients who would otherwise have died from the effects of their epistaxis. It may be noted that, in most of the seven fatal cases, both anæmia and hypotension were left untreated, and even when blood transfusions were given never more than one pint was used. Of the nine cases admitted to the Royal National Throat, Nose and Ear Hospital considered to need transfusion, only one received less than two pints, and this was the only patient in the group who died.

Measures directed toward arresting the hæmorrhage are of prime importance. They have been fully described (Negus, 1955), Hallberg (1952), Collier (1948), Macbeth (1948) and are not the subject of this paper. It is our belief, however, that current teaching in Otolaryngology lays insufficient stress on the importance of blood transfusion in certain

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cases of epistaxis. This is reflected by the finding that, in three of our fatal cases, and 32 per cent. of the Royal National Throat, Nose and Ear Hospital series no hæmoglobin estimations were made on admission. Eight out of 17 patients whose hæmoglobin was less than 60 per cent. on admission were given no transfusion; while one patient admitted with a hæmoglobin of 51 per cent. was treated without transfusion for 10 days, until he developed a coronary thrombosis. A transfusion was then given and he recovered.

We conclude that in the treatment of epistaxis the presence of cardiac disease, anæmia or hypotension should be treated as serious prognostic signs. Prompt treatment of the last two by blood tansfusion may do much to reduce the mortality from this condition.

Summary

A review of seven fatal cases of epistaxis, and of one hundred consecutive hospital admissions for this condition indicates that the cardiovascular effects of the hæmorrhage are of crucial importance. Fatal epistaxis is probably not rare, and active treatment of hypertension and anæmia by blood transfusion may be life-saving.

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