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On the occurrence of necrotising lesions in arteritis temporalis: review of the literature with a note on the potential risk of a biopsy

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Summary—Against the background of a steadily growing proportion of elderly individuals within the populations in the Western countries, arteritis temporalis with its wide diversity of presenting symptoms in the elderly patient, including purely psychiatric ones, has become a disease of increasing interest to the medical profession. Increasing demands may be made on the surgeon, often a plastic surgeon, responsible for carrying out the biopsy that is necessary in arteritis temporalis for both diagnostic and therapeutic reasons. Attention is called to certain elements of risk inherent in taking a biopsy of an artery under local anaesthesia, and to the fact that the administration of ergotamine tartrate may provoke serious complications in this particular disease.

Arteritis temporalis (AT) was first described as a clinical entity by Hutchinson in 1890. The designation "arteritis temporalis" was first applied by Horton *et al.* (1932) based on hitherto undescribed histopathological findings within the superficial temporal artery. Synonyms are: arteritis cranialis, giant-cell arteritis, granulomatous arteritis, Horton's disease or, as suggested by Paulley and Hughes (1960), "arteritis of the aged".

The full-blown histopathology in AT shows panarteritic inflammation, probably spreading along the vasa vasorum (Cooke *et al.*, 1946), as evidenced by granulomatous infiltration and destruction of the artery wall architecture with oedematous swelling of the intima, a pronounced reduction of the lumen of the vessel, and a tendency towards the formation of thrombi. A typical, but not obligatory, finding is the collection of multinucleated giant-cells around necrotic foci between the media and intima with disruption of the internal elastic lamina (Cohen and Smith, 1974; Klein *et al.*, 1975; Editorial, *Lancet*, 1983), (Figs 1 and 2).

Fig. 1

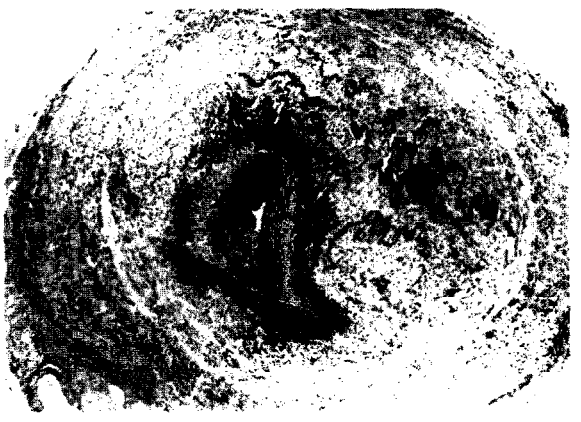

Fig. 2

Figure 1—Normal superficial temporal artery. Figure 2—Arteritis temporalis. Panarteritic granulomatous infiltration and oedema with destruction of the normal artery wall architecture together with disruption of the internal elastic membrane and pronounced diminution of the lumen of the vessel. Note: the presence of multi-nucleated giant cells is not obligatory to make the diagnosis.

In the process of spontaneous healing, parallel to the clinically ascertained tendency, before the advent of steroids, of the vasculitis to run a self-limiting course (Cooke *et al.*, 1946), AT may present histological findings deviating from the above, that may result in fibrosis of the arterial wall and obliteration of the lumen (Östberg, 1971), or such changes may be therapeutically induced. Histopathological findings are by no means pathognomonic of AT but overlap with a number of vascular diseases among the so-called primary vasculitides, viz. diseases where the vascular lesions are the dominating pathogenic factor in the development of clinical manifestations. Vasculitides within this category are, among others, polyarteritis nodosa, Wegener's granulomatosis and Takayasu's arteritis. Besides the clinical manifestations, additional information concerning age, sex, ethnic factors and response to therapy are as a rule diagnostically distinctive. For example, Takayasu's arteritis prevails among young Orientals and American Indians (Alarcón-Segovia, 1980); it is a disease mainly affecting young females in the age group 10 to 24 years with a sex ratio 8:1 (Fraga and Lavalle, 1980). By contrast, AT primarily occurs in elderly Caucasians, 90% over the age of 60 years with a preponderance for women 2:1 (Calamia and Hunder, 1980).

The aetiology of AT is unknown, a fact that together with the non-specific histological findings hampers any attempt at classification. The modern concept of AT is that of a subacute to subchronic-chronic generalised vascular disease with points of resemblance to autoimmune and chronic inflammatory conditions (Gilmour, 1941; Chassagnon *et al.*, 1967; Alarcón-Segovia, 1980) with the site of predilection within the cranial vascular territory, the pathogenesis probably being mediated through a delayed hypersensitivity mechanism (Bentata-Pessayre and Delzant, 1981).

Although AT in terms of pathology is conceived as a generalised vascular disease (Cooke *et al.*, 1946), arteries are mainly affected, especially the larger and medium-sized arteries of the head, neck and upper torso (Calamia and Hunder, 1980). A close correlation has been demonstrated between the susceptibility to AT and the amount of elastic tissue in the media and adventitia of the individual arteries of the head and neck (Wilkinson and Russell, 1972). Based on post-mortem findings, the authors rate the superficial temporal, vertebral, ophthalmic and posterior ciliary arteries (nutritive arteries of the optic disc) as the most constantly and severely involved vessels in AT (Fig. 3). Occlusion of the nutritive arteries of the optic disc produces irreversible blindness of sudden onset with the pathognomonic ophthalmoscopic finding of ischaemic optic neuropathy with a pale oedema of the disc (Hayreh, 1969). Furthermore, the retinal artery (after entering the optic nerve), the common carotid and the cervical segment of the internal carotid are found to be less frequently, and the intracranial arteries only rarely, involved. The reduction, or loss, of the elastic tissue component of the intracranial arterial walls can easily be demonstrated, according to the authors, by the reduction in size of the vertebral arteries of about 0.5 cm after perforating the dura.

The question of "patchy" arteritic involvement or "skip lesions" in AT, implying the possibility of false negative findings of intervening areas of normal histology, is a matter of some controversy in the literature. "Skip lesions" were found by Klein *et al.* (1976), among others, in 17 out of 60 patients with AT, while Cohen and Smith (1974) maintain that areas totally devoid of pathological changes

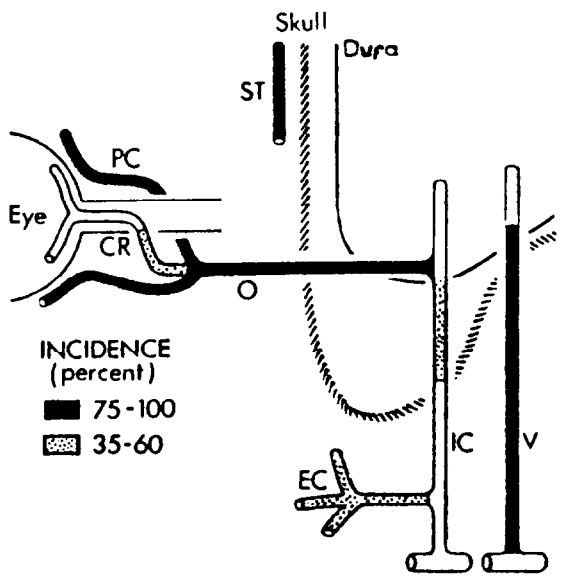

Fig. 3

Figure 3—Schematic representation of the relative distribution of arteritis temporalis within the vascular territory of the head and neck. V = vertebral, IC = internal carotid, ST = superficial temporal, O = opthalmic, CR = central retinal, PC = posterior ciliary arteries.

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are extremely rare, having failed to demonstrate such findings in 42 patients over a 12-year period. The question of comparability between the two groups of patients is debatable. It seems reasonable to assume that "patchy" or "skip lesions" antecede the full-blown panarteritic pathology of AT, and the differing findings in the two groups may be related to an increased alertness to AT on the part of the medical profession in charge of the former group, resulting in earlier diagnosis and start of therapy. In any event, the findings cited stress the need for a sufficient length of artery to be removed at biopsy, to provide representative material for histological examination. Cohen and Smith (1974) demand at least 2.5 cm of an artery, to be followed by serial sections of fixed preparations. They consider frozen sections unreliable. Klein *et al.* (1976) have demonstrated unilateral involvement of temporal arteries by AT in three of 60 patients. In their series of 76 patients with "arteritis of the aged", Paulley and Hughes (1960) found random scalp and muscle biopsy unhelpful.

Epidemiology

In a prospective study of AT and polymyalgia rheumatica, taken by the authors to be clinical manifestations of the same disease (giant-cell arteritis) and following the exclusion of other causes of the polymyalgic syndrome on clinical, laboratory and therapeutic criteria, Boesen and Sørensen (1984) found an overall incidence of $21.5/10^5$ or $76.6/10^5$ in age groups over 50 years in a Danish county with approximately 200 000 inhabitants, an incidence about twice as high as that found in previous retrospective studies (Bengtsson and Malmvall, 1981; Chuang *et al.*, 1982). The maximum incidence was found in the age group 70 to 79 years, well over 10 times that of the age group below 60. In all age groups the incidence in women was four to five times higher than in men. Obviously, AT in its different clinical manifestations, and its association with ethnic factors, is increasingly common in women as they grow older.

By comparison, Östberg (1971) has reported histopathological changes, interpreted as sequelae to AT, in 1% of large necropsy series, namely, 1097 adults who died over one year in Malmö, a Swedish town with about 250 000 inhabitants. The necropsy material represented 65% of all deaths in the period. The samples of the temporal arteries examined were only about 1 cm long, and no serial sections were made. The prevalence of 1% of AT in this necropsy series as compared to an incidence of about two cases per 100,000 population per year, estimated over a period of 10 years, seems to indicate that AT is grossly underdiagnosed and that it is often a concomitant disease in deaths from other causes.

Symptomatology

To cite Goodman (1979): "AT like tuberculosis and syphilis is a disease of protean manifestations. No single clinical symptom, sign or laboratory test can be relied upon for establishing the diagnosis, —as might be expected with a disease the substrate of which is ischaemia due to progressively obstructive vascular pathology". This statement by Goodman merely emphasises the need for a clinical approach including a careful history to be evaluated in connection with laboratory findings.

The symptomatology of AT may include several general prodromic features liable to defer the diagnosis such as malaise, anorexia, loss of weight, fever and sweats together with pain and stiffness in the joints and muscles of the upper extremities and upper torso, usually worse in the morning, the so-called "rheumatoid syndrome" or, "anarthritic rheumatoid disease" (Bagratuni, 1953, 1956) or, more recently, polymyalgia rheumatica, or, as suggested by Hamrin (1972), polymyalgia arteritica. The syndrome is associated with AT in about 50% of the cases (Hamilton *et al.*, 1971) otherwise with malignancy, infections or connective tissue diseases (Hunder *et al.*, 1969). Among 46 patients with AT collected over 1 year in a prospective study by Boesen and Sørensen (1984), almost 90% of the presenting symptoms were related to the polymyalgic syndrome.

Apart from the classical finding of a tender swollen non-pulsatile temporal artery covered by reddened skin, AT displays a diversity of focal clinical manifestations either alone or in addition to an already established polymyalgic syndrome. Focal symptoms in question are: headache, usually severe and typically a constant pain with exacerbations of a throbbing character in temporal, frontal or occipital region. The headache may occur with brain stem strokes and occipital blindness, with vertebral arteries heavily affected by AT (Wilkinson and Russell, 1972). Hemispherical strokes occur in AT but the pathogenesis is not clear: it may be related to arteritic changes in the wall of the internal carotid artery (Cardell and Hanley, 1951) or to coincidental thromboembolism (Mulley, 1982). It

Table 1 Tongue and scalp necrosis in arteritis temporalis

					Necrosis of			
		Age	*Sex*	*Biopsy*	*Tongue*	*Scalp*	*Loss of sight*	*Cortisone treatment*
Cooke	(1946)	66	F	–		+	Total	–
—		73	F	+		+	Total	–
Riis	(1947)	65	M	–		+	Left eye	–
Schweitzer	(1949)	80	M	+	+		Right eye	–
Girard	(1952)	78	F	+		+		+
Kendall	(1953)	78	F	–		+	Total	–
Whitfield	(1953)	66	F	–		+	Reduced	+
Siguier	(1957)	?	M	+		+		+
Tirschek	(1957)	74	M	–		+	Reduced	–
Poppy	(1959)	77	F	+		+		–
Howard	(1959)	74	F	–	+			–
Bergan	(1959)	80	F	–	+			–
Russell	(1959)	?	?	–		+		–
Boroffka	(1960)	67	M	–		+		–
Fleischl	(1960)	85	M	+		+	Right eye	+
Clark	(1960)	75	M	–		+	Left eye	+
Brearley	(1961)	82	F	+	+	+		+
Pitt	(1961)	78	F	+	+		Reduced left eye	+
McGill	(1961)	77	F	–	+			+
Kinmont	(1964)	79	F	–	+		Reduced	+
—		75	F	–		+	Total	+
—		79	M	–		+		+
Wittels	(1964)	75	M	+		+	Total	+
—		75	M	+		+		+
Reed	(1965)	72	F	–	+			–
Novotny	(1966)	72	M	+		+		+
Davis	(1966)	74	F	+	+			+
Kaul	(1966)	69	M	+		+		+
Barefoot	(1966)	80	M	+		+	Reduced	+
Louyot	(1966)	74	M	–		+	Reduced	+
Bonnet	(1967)	70	M	+		+	Total	+
Freedman	(1967)	81	M	–	+			–
Haye	(1967)	76	M	+	+	+	Reduced	+
Schrecke	(1967)	82	F	+		+		+
Chassagnon	(1967)	76	M	+		+		+
—		81	M	+		+		+
Harris	(1968)	81	F	–	+	+		+
Labouche	(1968)	72	M	+		+		+

naturally follows that in the age group at risk, atheromatosis remains an important concurrent pathogenic factor.

Focal symptoms may precede more generalised ones and should be evaluated with a high degree of suspicion of an underlying AT. This applies particularly to symptoms such as claudication of the jaw (pathognomonic of AT according to Horton *et al.* (1932). and hypersensibility of the facial skin and scalp. Hutchinson (1890) in the first report on AT tells of a gentleman "upwards of 80 and almost in his dotage" with "red streaks" in his scalp. The streaks were painful, and prevented him from wearing his hat. On palpation the streaks turned out to be tender swollen arteries covered by reddened skin extending their dividing branches on both sides almost to the middle of the scalp. Other focal symptoms are burning sensation or numbness of the tongue, pain in and around the temporomandibular joint masquerading as a dysfunction syndrome (Siemssen, 1952), pain in the ear, vertigo and deafness of sudden onset, evidently of vascular origin (Paulley and Hughes, 1960), amaurosis fugax, and transient ocular symptoms of external ophthalmoplegic origin such as diplopia and ptosis, altogether symptoms reflecting an intermittently ischaemic focus somewhere within the area of blood supply via branches of the external carotid and ophthalmic arteries. Of 248 patients with AT studied by Klein *et al.* (1975), 34 showed clinical evidence of involvement of the aorta and its major branches. Typical presenting symptoms were claudication of an extremity following repetitive muscle contractions, together with paraesthesias and Raynaud's phenomenon. Three patients died with rupture of the aorta. Apart from biopsy of apparently

Table 1 (*contd.*)

Bour	(1969)	76	M	+	+	+	Total	+
Folan	(1969)	78	F	–		+		–
Bourgeois	(1970)	76	F	+		+	Reduced	+
Hitch	(1970)	76	M	+		+	Reduced	+
Schulz	(1971)	54	F	+	+			+
Feraut	(1971)	77	M	+	+	+		+
Reuther	(1972)	69	M	+		+	Right eye	+
—		54	F	+	+	+		+
Wolpaw	(1973)	*68	F	+	+			+
Higgins	(1973)	90	F	–	+			–
Samson	(1974)	75	M	+	+	+	Total	+
Reboul	(1975)	†78	F	+	+	+	Temporary	+
Beylot	(1975)	77	M	+	+	+	Total	+
Vrébos	(1976)	74	F	+		+	Reduced	+
Dufetelle	(1976)	†72	M	+	+			+
Soderstrom	(1976)	81	M	+		+		+
Rosenman	(1978)	84	M	+		+	Reduced	+
Arnung	(1979)	†69	F	+	+			+
Rousset	(1979)	68	M	+		+		+
Allen	(1980)	66	F	+	+			+
Hicks	(1980)	73	F	–	+			+
....		80	F	–	+			+
Quintanilla	(1980)	68	M	+		+	Reduced right eye	+
Sofferman	(1980)	74	M	+	+			+
-		68	F	+	+			+
Dare	(1981)	82	M	+	+			+
Doumith	(1981)	80	F	+	+			+
Maradona	(1981)	?	?	+		+		+
Remky	(1981)	‡70	F	+	+		Total	+
Renard	(1982)	84	F	+	+			+
Browne	(1982)	89	F	–	+			+
—		69	M	+	+			+
Hujic	(1982)	70	M	+	+			+
Storm	(1983)	72	F	+	+		Left eye	+
Pedersen	(1983)	77	F	+	+			+
Symanzik	(1984)	79	F	+	+			+
Roseman	(1984)	58	M	+	+			+
Christensen	(1985)	*74	F	+	+			+

* Necrosis after injection of ergotamine tartrate.
† Necrosis after biopsy.
‡ Necrosis after injection of Ultracain".

normal temporal arteries, angiography was considered helpful in establishing the diagnosis, an assumption disputed by Cohen and Smith (1974).

To add to the diversity of symptoms in AT it is just briefly mentioned that purely psychiatric signs such as depression, confusion and dementia may herald the onset of AT in the elderly, a possibility that ought to be taken into consideration in such findings (Cooke *et al.*, 1946; Paulley and Hughes, 1960).

As a rule a raised erythrocyte sedimentation rate (ESR) helps to substantiate the clinical diagnosis of AT. In estimating an ESR, allowance must be made for the fact that the upper limit for ESR should be raised to 40 mm/h as normal reading in patients over 50 years, and to 60 mm/h in older anaemic patients (Kulvin, 1972). Once the diagnosis of AT has been established, Cohen and Smith (1974) consider the ESR a sensitive monitor of disease suppression following specific corticosteroid therapy. Likewise, Wadman and Werner (1972) conclude from a comprehensive set of laboratory tests in 53 consecutive patients with AT (48 verified at biopsy) that the findings are non-specific and of low predictive value, and that no biochemical factor gives better information for long term control than the ESR.

Most patients display a slight normocytic anaemia, and an abnormal electrophoresis picture. A slight elevation of the white cell count may be seen together with an elevation of the platelet count, as evidenced in 84 out of 100 patients (Calamia and Hunder, 1980), the latter finding possibly indicating the presence of compensated intravascular coagulation (Grau *et al.*, 1979).

In addition to the above symptomatology, cases

with or without histologically proven AT have been published with necrotising lesions of the scalp, facial skin and tongue, usually as short casuistic communications scattered throughout the literature (see Table 1).

In one personal case (to be published elsewhere) a 69-year-old woman with AT developed paraesthesia and reduced skin temperature of the lower face and immobility of the tongue within hours of a biopsy of a temporal artery under local anaesthesia followed by extensive necrotising lesions of the scalp, lower lip, floor of the mouth with exposure of the alveolus, loss of teeth and the entire mobile part of the tongue. This astonishing list of complications aroused my interest in the reported occurrence of such complications, and particularly the circumstances under which they occurred.

Discussion

A total of 76 reports of necrotising lesions of the scalp facial skin and tongue have been collected from the literature with or without histological proof of AT (Table 1). As regards the latter group (23), it should be recalled that no such lesions have been described in the clinical course of any disease other than AT. A negative temporal artery biopsy may result from insufficient length of the sample owing to patchy or segmental pathology, or the surgeon may have omitted to do a biopsy of the contralateral temporal artery or some other branch of the external carotid more relevant to presenting focal symptoms, often the facial artery (Paulley and Hughes 1960). The final issue rests with the care taken by the pathologist in the examination of serial sections of fixed preparations. Presumably electron microscopy may reveal early changes in AT not detectable by light microscopy.

It seems reasonable to assume that tissues critically dependent on an unstable blood supply via stenosed and partially thrombosed arteries may undergo necrosis triggered by a spasm, whether this occurs spontaneously, or is being induced by a vasoactive drug, or brought about by manipulative trauma.

There is ample evidence of spontaneous arterial spasms occurring in AT as exemplified by focal symptoms such as claudication of the jaw, tongue, pharynx, and intermittent facial pain, and, especially, by intermittent partial blindness, sometimes coinciding with ophthalmoscopical findings of severely retarded retinal circulation and contraction of visual fields (Riis, 1947; Calamia and Hunder, 1980).

Epinephrine and noradrenaline are routinely added to a local anaesthetic to obtain vasconstriction and delay absorption within the operative field. An ascending and/or descending arterial spasm may be elicited by the vasoactive drug, and provoke complications even in patients without signs of peripheral vascular disease. Extremely unhappy instances of permanent blindness (partial or total) following infiltration anaesthesia of the nasal septum prior to surgery have been reported by Plate and Asboe (1981). The pathogenic mechanism is believed to be a spasm of the ophthalmic artery elicited by an intra-arterial injection.

Of the total number of necrotising lesions, two followed the administration of ergotamine tartrate. The ability of this drug to provoke necrotising lesions in a patient already suffering from AT is well brought out in a report by Wolpaw *et al.* (1973). A severe headache was misdiagnosed as migraine in a 68-year-old woman who on two occasions developed numbness and cyanosis of the tongue following administration of ergotamine tartrate, to end up with partial necrosis of the tongue. The authors stress the caution required in the use of ergotamine tartrate in the elderly patient whose headache, as in the reported case, might very well have been the presenting symptom of AT, quite apart from the fact that migrainous headache is a rare occurrence in the elderly.

Roseman and Granite (1984) reported a massive necrosis of the tongue in a male, 58 years of age, who since childhood had been treated intermittently for cluster headaches with Cafergot®. Presumably this patient had been rendered susceptible to a necrotising lesion by a chronic ergotism as a complication of an underlying AT.

Conclusions

Until the advent of steroids, after 1949, with the sensational discovery of the ability of corticosteroids to inhibit inflammatory processes, the treatment of AT was entirely symptomatic. Some beneficial effects have been ascribed to nicotinic acid (Cooke *et al.*, 1946), probably owing to dilatation of the smaller cutaneous vessels. Reduction or elimination of pain has repeatedly been observed following biopsy of cranial arteries (Horton and Magath, 1937; Cooke *et al.*, 1946), and has been carried out for the relief of headache, the

effect presumably being due to the exclusion of pain receptors within the arterial wall.

However appealing the administration of specific anti-inflammatory therapy based on histologically proven disease may seem, the diagnosis of AT should be a tentative one, based on clinical examination including a careful history, concentrating on possibly previous episodes of blurred vision and/or severe headache, symptoms that may be forgotten by the patient when first seen. The patient who is suspected of having AT should be recognised and treated as an emergency, the primary concern being the prevention of blindness that is usually irreversible (Hayreh, 1969; Cullen, 1982). An ESR should be obtained immediately, to help support the diagnosis and to provide a base-line or reference value, before corticosteroid therapy is started. The recommended dosage (Drug and Therapeutic Bulletin, 1984) is Prednisolone 60 mg initially, to be continued for a week, and then gradually reduced with abating symptoms and decreasing ESR. Clinical improvement is often dramatic, and discernible to the patient within hours of starting corticosteroid therapy. This dramatic response helps to substantiate the clinical diagnosis.

Although AT may behave, and has been described, as a self-limiting disease, there seem to be many exceptions to this. The minimum recommended length of treatment is two years in a study by Fauchald *et al.* (1972) who observed a relapse rate of 26% in a group of 20 patients with histologically proven AT. By repeated biopsies during and after steroid therapy, the authors have shown that arteritic changes may persist for a long time. Thus, histologically typical AT has been reported in a patient, clinically asymptomatic, 10 years after steroids were discontinued, and recurrence of a polymyalgic syndrome with intense inflammatory changes at biopsy of the temporal artery nine years after the initial disease, has been observed by Blumberg *et al.* (1980).

Grave risks may be incurred by prolonged steroid treatment (for example a 25% incidence of symptomatic vertebral fractures following steroid-induced osteoporosis (Huston *et al.*, 1978)), and an arterial biopsy is a must, whenever feasible, to confirm or exclude the diagnosis of AT (Hall *et al.*, 1983). However even if treatment is urgent in suspected AT there is clearly no reason to regard a biopsy as being equally urgent. Indeed the latter procedure is probably best postponed a day or two to be planned and performed as an elective operation. The surgeon must be prepared to do biopsies not only on both temporal arteries but to modify his search for representative material according to presenting focal symptoms in the individual patient. The facial and occipital arteries may be biopsied and as the search should be guided by frozen sections, it is best managed under general anaesthesia.

This study has emphasised, once again, the need for caution in the use of vasoactive drugs, both systemically and locally, in peripheral vascular disease. In order not to add to the risk inherent in an arterial biopsy of provoking ischaemia, the admixture of a vasoconstrictor agent such as epinephrine or noradrenaline to the local anaesthetic is considered inadvisable on an empirical basis. It naturally follows that an arterial biopsy on a patient with peripheral vascular disease, *e.g.* AT, must carry a certain risk (Kirsch, 1983). It therefore seems prudent to limit the length of the arterial biopsy specimen to three centimetres or less, and to handle the artery at biopsy with the utmost care.

With the growing proportion of the elderly within the population of the Western countries, AT, by its diversity of presenting symptoms, has become a disease of increasing interest to the medical profession. To cite Paulley and Hughes (1960): "When elderly people begin to fail mentally and physically this (AT) should be one of the first disorders to be considered, and not one of the last", and one might feel tempted to add: "and before ergotamine tartrate is administered in suspected migraine in the elderly". This clinical approach concerns among others the surgeon, often the plastic surgeon, who is responsible for carrying out the appropriate diagnostic biopsies.

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