

A simple parameter for accurate detection of severe carotid disease

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The value of the ratio of the peak systolic velocity in the internal carotid artery to the end diastolic velocity in the common carotid artery in quantifying the severity of carotid arteriosclerosis has been investigated in 86 diseased vessels and 30 normal vessels. The ratio permitted gradation of disease into 60 per cent stenosis and greater with 95 per cent accuracy, 65 per cent stenosis and greater with 97 per cent accuracy and greater than 90 per cent stenosis with 100 per cent accuracy. The values for 30 normal sides studied fell below those of the 60 per cent stenosis group. The ratio has a value in predicting the severity of disease in the internal carotid artery in those with a 60 per cent stenosis and greater.

In recent years, a great deal of effort has gone into the development of simple non-invasive diagnostic measurements in the assessment of arterial disease. In our laboratory, an ultrasonic Duplex scanner which provides simultaneous real-time B-mode arterial images and a single gate pulsed Doppler are used to detect velocity changes in the extracranial carotid system. This velocity information is analysed by an on-line spectrum analyser which permits an evaluation of the frequency content of the back-scattered Doppler signal to be made. This has enabled us to estimate the degree of stenosis of the extracranial internal carotid artery (1,2). We have previously reported on the use of a velocity ratio derived from the common and internal carotid arteries for the assessment of the severity of disease (3). Another velocity ratio has been assessed in an effort to define its role as a predictor of severe disease of the internal carotid artery.

Materials and methods

A retrospective analysis of 86 common and internal carotid arteries which had been studied with the Duplex scanner was performed. All of these vessels had been assessed with biplanar contrast arteriography. Those sides which had had an arteriogram performed more than 2 months after the Duplex scan were excluded from the analysis as the disease might have progressed in these patients rendering any comparison of Doppler-derived parameters with arteriography inaccurate. The degree of stenosis was calculated from the arteriogram by measuring the diameter of the diseased vessel and using the equation below to calculate the percentage of stenosis:

$$\% \text{ stenosis} = 100 \times \left[1 - \frac{\text{diseased vessel diameter}}{\text{diameter of this vessel if normal}} \right]$$

The Duplex scanner combines B-mode imaging with a 5 MHz single-gated pulsed Doppler. The B-mode image was used to place the sample volume of the pulsed Doppler in the centrestream of the desired vessel. The pulsed Doppler signals were taken from the low common carotid artery and the internal carotid artery at the site of maximal disturbance of flow. The back-reflected Doppler signal is subjected to a fast Fourier spectral analysis and a hardcopy output obtained (4).

A typical spectrum obtained from a normal common carotid artery is shown in Fig. 1 and the spectrum from a common carotid artery feeding a diseased internal carotid artery in Fig. 2. This shows that in disease, the characteristic outline of the waveform is lost and the diastolic flow is tending to fall to zero levels. The spectrum from a normal internal carotid artery is shown in Fig. 3 and that from a diseased internal carotid artery in Fig. 4. The frequencies at peak systole are increased in the diseased vessel as are the frequencies in late diastole.

The frequency of the back-scattered Doppler signal (F_d) is related to the velocity of flow within the vessel (V) as given by the equation:

$$V = \frac{F_d C}{2F_0 \cos \theta}$$

where C = a constant related to the speed of sound in tissue (15.4×10^4 cm/s), F_0 = frequency of the incident Doppler beam

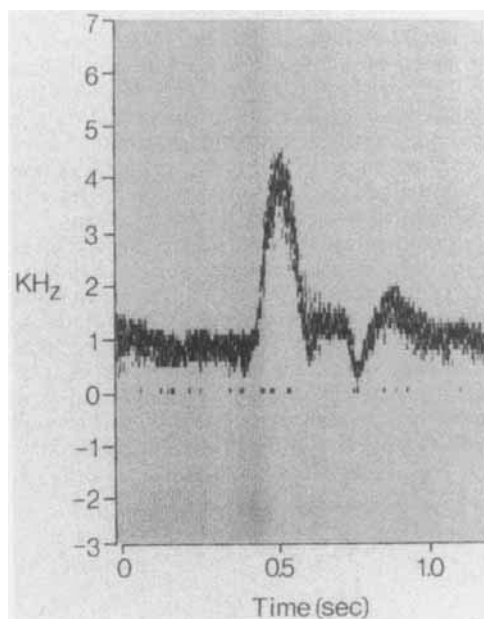


Fig. 1. Typical spectrum from a normal common carotid artery feeding a normal internal carotid artery.

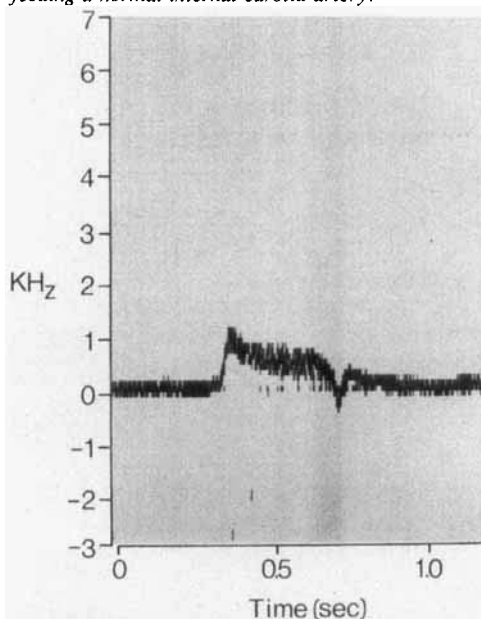


Fig. 2. Typical spectrum from a normal common carotid artery feeding a diseased internal carotid artery.

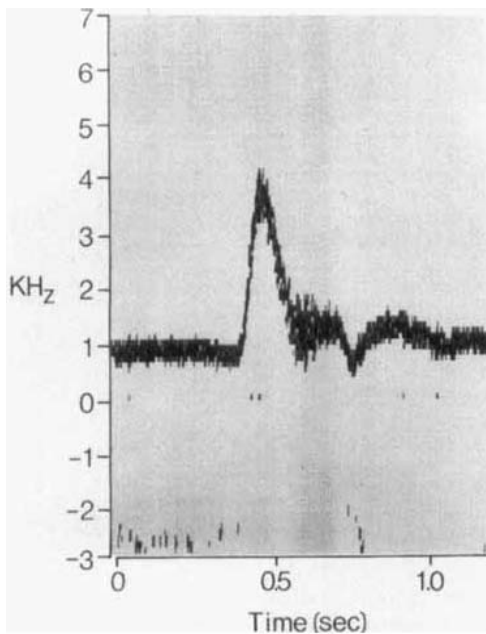


Fig. 3. Typical spectrum from a normal internal carotid artery.

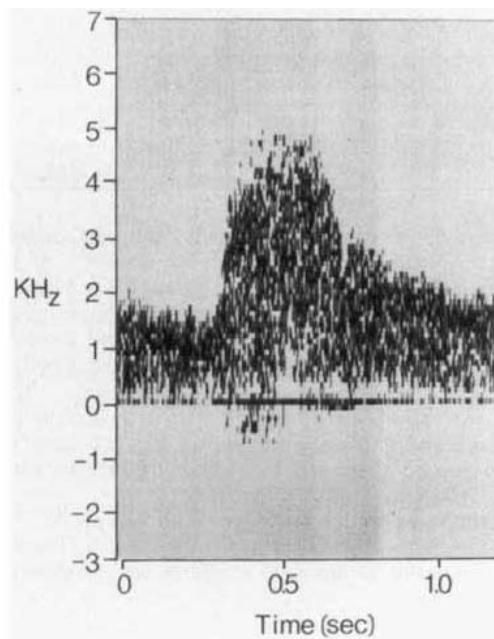


Fig. 4. Typical spectrum from a diseased internal carotid artery.

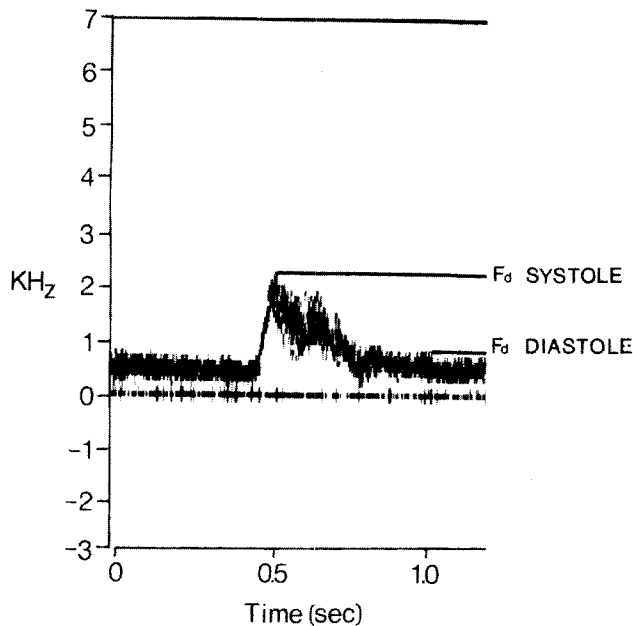


Fig. 5. The measurement of the frequency shift (F_d) from the spectrum at peak systole and late diastole.

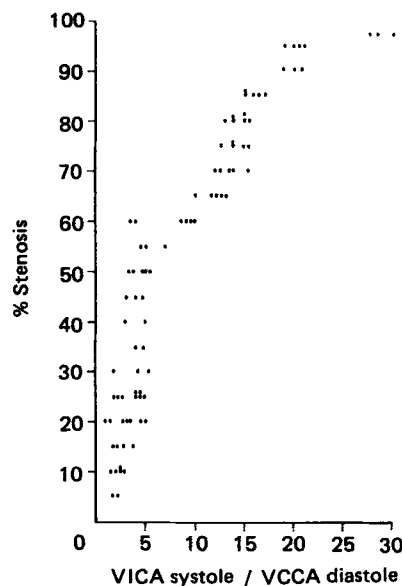


Fig. 6. VICA systole/VCCA diastole in the 86 vessels plotted against the percentage stenosis.

(5×10^6 Hz) and θ = the Doppler angle between the incident beam and the longitudinal axis of the vessel. As θ may be measured from the B-mode image at the time of the study, calculation of the velocity of flow within the vessel is simple. F_d was measured from the output of the spectrum analyser as shown in Fig. 5.

A group of 30 normal subjects was studied using the Duplex scanner in an identical fashion to the diseased subjects. These subjects were 'presumed' normals in that they had not undergone confirmatory arteriography. Their ages ranged from 29 to 32 years. The ratio of the peak systolic velocity in the internal carotid artery to the late diastolic velocity as measured 500 ms after peak systole in the common carotid artery was calculated for all vessels studied.

Results

The range of velocities found in each group is shown in Table I. The velocity ranges found in each group are wide but the diastolic velocity in the common carotid artery (VCCA) tends to fall with increasing disease, whereas the peak systolic velocities in the internal carotid artery (VICA) increase.

The variation of the ratio VICA (systole) to VCCA (diastole) with severity of disease is shown in Fig. 6. The ratio increases exponentially as the severity of disease increases. It is impossible to distinguish stenoses less than 60 per cent using the ratio but for stenoses greater than 60 per cent it is possible by selecting particular values of the ratio to grade the severity of disease (Table II). If a value of 7.5 is selected as defining

Table I: LATE DIASTOLIC VELOCITY AND PEAK SYSTOLIC VELOCITY RANGES IN THE COMMON CAROTID AND INTERNAL CAROTID ARTERY (cm/s)

Carotid lesion	Common carotid (diastole)	Internal carotid (systole)
Normal	48-53	30-114
10-60% stenosis	22.4-53.9	39-220
65-90% stenosis	7.9-23.1	123-282
Greater than 90% stenosis	7.2-15.4	154-325

Table II: THE ACCURACY OF THE RATIO IN PREDICTING DISEASE

Category	Number of sides	Accuracy	Ratio
≥60% stenosis	42	95	7.5
≥65% stenosis	36	97	11.0
≥90% stenosis	10	100	18.0

disease greater than 60 per cent stenosis, then 2 out of 42 sides (4.8 per cent) are wrongly classified. Similarly, selection of a value of 11.0 as defining disease greater than 65 per cent stenosis results in 1 out of 36 sides (2.7 per cent) being wrongly classified. Selection of a value of 18 allows all diseased vessels with stenoses equal to or greater than 90 per cent to be correctly classified (Table II).

The values of the ratio for each category of disease have a small range which for the disease categories selected do not overlap. There is considerable overlap in values of the ratio for all states of disease less than 60 per cent stenosis.

The values of the ratio for the normals are shown in Fig. 7. All were equal to or below 3 with a range from 1.3 to 3. These did not overlap with the values seen in those with stenoses greater than 60 per cent.

Discussion

If constant flow rates are to be maintained through a progressively narrowing stenosis, the velocity of flow through the stenosis must increase. The velocity of flow is proportional to the cross-sectional area of the arterial lumen at the site of measurement. Studies of steady flow in bifurcating tubes have revealed that the mean flow velocity in the branch bears a constant relationship to the velocity in the parent tube. While the flow velocity is influenced by the cross-sectional area of the vessel, the compliance of the vessel wall and the peripheral resistance also have an effect (5). The ratio of velocity in the internal carotid artery to the velocity in the common carotid artery will cancel the effects of the compliance of the vessel wall. However, the problem associated with the differing peripheral resistance perfused by the internal and external carotid arteries remains. In a previous report, we implied that the velocity of flow in the external carotid artery should be taken into account in calculating velocities in the common carotid artery (3). Experience has shown that it is difficult to image the external carotid artery reliably and to place the sample volume of the pulsed Doppler accurately in the centrestream of this vessel as it is frequently quite small. Therefore, in this study we have not measured the velocity of flow in this vessel.

Previous reports from this laboratory have documented the increased velocities seen in a stenosed vessel at peak systole (1, 3). We have also noted that in more severe disease of the internal carotid artery, the end diastolic velocity in the common carotid artery tends to fall to zero levels (6). The velocity pattern of the common carotid artery is dominated by the low resistance of the ipsilateral hemisphere resulting in a waveform which is similar to that observed in the internal carotid artery. Occlusive disease of the internal carotid artery which increases the resistance to flow may be detected by changes in the common carotid velocity waveform (7). A total occlusion of the internal carotid artery will produce a flow pattern in the common carotid artery which resembles that in the external carotid artery, i.e. the flow tends to zero during diastole and may even reverse in some instances. Ideally, any velocity ratio, in order to be sensitive to increasing disease should consist of a numerator which increases with severity of disease and a denominator which decreases with increasing disease. Such a ratio will tend towards infinity with increasing severity of disease. The ratio of peak systolic velocity in the internal carotid artery to the diastolic flow velocity in the common carotid artery appears to fit this definition tending to infinity with increasing severity of disease.

This retrospective study has demonstrated the feasibility of using this ratio to grade disease states greater than 60 per cent diameter reduction. It was not entirely unexpected to find that we were unable to distinguish normals from diseased subjects with a 60 per cent or lesser diameter reduction using a velocity

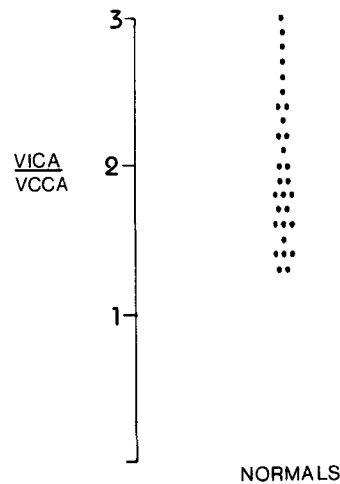


Fig. 7. VICA systole/VCCA diastole in the 30 normal vessels.

ratio. Markedly decreased end diastolic flow in the common carotid artery is not seen until more severe disease states are reached. The ratio is, therefore, reliant on increasing velocities at peak systole in the internal carotid artery to produce discriminatory changes in the normal to 60 per cent stenosis group. These changes in peak systolic velocity are not seen until stenoses of 50 per cent and greater are present and are not large enough to permit a gradation of disease state below this. There is a need to grade further the severity of disease in those with a greater than 50 per cent diameter reducing lesion. As yet little work has been done in this direction, most reports classifying disease as greater or less than 50 per cent diameter reduction with no further gradations in the greater than 50 per cent group. This ratio provides a useful method of further grading this group and as such may be used for follow-up studies of asymptomatic stenoses.

When correlating the ratio of velocities with arteriography, it must be accepted that there are errors in the classification of disease based on arteriography. Recent work has shown that the intra-observer agreement for the classification of carotid disease into the categories selected was 83 per cent. The second reading disagreed with the first reading 17 per cent of the time, despite the fact that both readings were made by the same radiologist. The inter-observer agreement for the same categories was 75 per cent. However, for disease categories greater than 50 per cent diameter reduction, the agreement is 85 per cent (8). It is important to bear this variation in mind when interpreting any comparative study. The arteriograms in this series were read by one radiologist; therefore, we do not have to consider the inter-observer agreement which is lower than that for a single observer.

The method chosen for the calculation of the percentage stenosis does not involve a measurement of the diameter of the distal internal carotid artery. The measurements are made from unsubtracted films which allow the identification of calcium within the vessel wall. This permits an estimation of the site of the vessel wall at this point and allows the measurement of the diameter of the vessel to be made. This technique enables the identification of minimal lesions encroaching on the lumen of the carotid bulb.

While we have used a Duplex scanning device to obtain our data, it is possible to use other pulsed Doppler devices or combined B-mode and continuous wave devices, providing that these incorporate a method of measuring the angle of the incident Doppler beam to the long axis of the vessel. A potential problem may arise in cases where the internal carotid artery is kinked and the incident Doppler beam approaches the longitudinal axis of the vessel. In this case, the shifted frequencies will be high resulting in a false impression of severity of disease.

In conclusion, therefore, this ratio seems to be useful in detecting and grading severe disease in the internal carotid artery. Spectral broadening is a sensitive indicator of lesser

disease states (less than 50 per cent stenosis), and it would appear that further research is necessary in order to define more accurately this parameter to render accurate gradation of minimal disease states possible.

Acknowledgements

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Recto-urethral fistula in Crohn's disease

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Enteric fistulas commonly complicate Crohn's disease (1, 2). Perineal fistulas are frequent and may demand surgical treatment. A fistula between the rectum and the prostatic urethra is rare and, in the patient described here, closed spontaneously.

Case report

An 18-year-old man presented in 1977 with a year's history of increased stool frequency, a painful perineal swelling and intermittent mucopurulent perianal discharge. His sister had Crohn's disease of the colon. Examination under anaesthesia showed an indurated perineum with a sinus track to the left of the anal margin, passing proximally alongside the rectum for a distance of 5 cm. On sigmoidoscopy, the rectum was inflamed and biopsy showed an ulcerated mucosa with chronic inflammatory cells extending into the submucosa. Granulomas provided histological confirmation of the clinical diagnosis of Crohn's disease. Treatment with hydrocortisone enemas (Colifoam) and oral prednisone was associated with a resumption of normal bowel habit and a reduction in the perianal discharge.

In early 1979 the perianal discharge increased and he was treated with azathioprine. Examination under anaesthesia after 6 months showed a complex anorectal fistula with multiple tracks and narrowing of the rectum. A barium enema showed shallow ulceration throughout the colon but the caecum was spared. A barium follow-through was normal. The haemoglobin was 11.3 g/dl, ESR 84 mm/h and albumin 39 g/l. He continued on azathioprine and Colifoam enemas.

One month later urine began to leak from the perianal opening during micturition and a fistulous communication was shown between the rectum and the prostatic urethra (Fig. 1). The urine was sterile. Over the next 6 weeks the perianal urinary leakage disappeared and, following metronidazole therapy, his perineal symptoms have also subsided.

Discussion

Fistulas connecting the large bowel with the bladder (2), the vagina (1, 2) and the uterus (3) are well-recognized complications of Crohn's disease. A fistula between the rectum and the prostatic urethra has not been described; there is only one patient reported with a recto-urethral fistula complicating Crohn's disease (4).

Urinary infection, pneumaturia and faecaluria did not occur in this patient, presumably because his fistula connected with the prostatic urethra. He complained of discharge of urine from the perianal opening but not from the anus; it is likely that any urine passing into the rectum during micturition was absorbed.

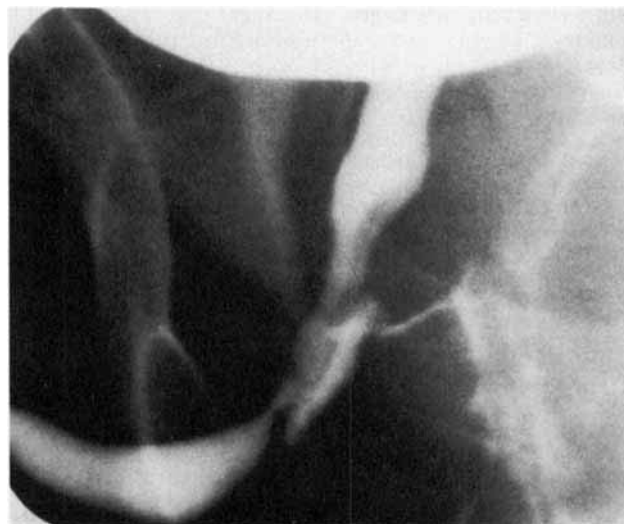


Fig. 1. Fistulous communication between the rectum and the prostatic urethra.

It is possible that this patient's unusual recto-urethral fistula resulted from trauma during the examination under anaesthesia, barium enema or self-administration of Colifoam. The complexities of treating such a fistula surgically were thought to outweigh the patient's disability and, to date, our conservative approach seems to have been vindicated.

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