Discussion

After a series of careful studies of the results of surgery of intracranial aneurysms in 1960-5 McKissock *et al*[3-5] concluded that surgery was of little overall benefit compared with conservative management. They also stated that patients who survived six months after an initial subarachnoid haemorrhage had little risk of recurrence. Both these points are arguments in favour of conservative management of these patients. Pakarinen,[1] however, in his total population study of the citizens of Helsinki could not agree with their finding of a low recurrence rate six months after subarachnoid haemorrhage: Pakarinen showed that patients treated conservatively, even if they survived one year after subarachnoid haemorrhage, still had a mortality from recurrent haemorrhage of 25% when assessed at five years. He also found recurrences occurring up to 21 years after the initial subarachnoid haemorrhage.

Conservative management becomes less attractive in the light of this information, particularly when obliterating an intracranial aneurysm by direct surgery will prevent a recurrent haemorrhage from that aneurysm. The long-term advantages of direct surgery of intracranial aneurysms are clear, but the effectiveness of surgery must be compared with that of conservative management in terms of mortality within the first six months.

The series of McKissock *et al* has the unique virtue of providing a control series of patients treated conservatively but who were also suitable for surgery. When assessed at six months they had a mortality of 37%.

Surgical results depend especially on how long after the bleed the operation is performed. Unless this information is provided with the results then assessment of the effect of surgery may be difficult. For example, the apparent mortality in our series is 15%. If the four patients who bled waiting for surgery are added, then the "true mortality" is 19%. Alternatively, knowing from Pakarinen's series that 5% of patients die each week in the first five weeks and that 22% of the patients in our series were operated on in the second week and 20% after that, then from theoretical considerations about four patients should be expected to die before surgery. Therefore the "true mortality" in our series is about 19%. In the only other large published series of microneurosurgery for intracranial aneurysms Krayenbuhl *et al*[7] reported a mortality of only 5·6%. But 48% of their patients were operated on after the 14th day and only 24% in the first week after the bleed. Assessing their "true mortality" by referring to Pakarinen's figures brings their mortality to a similar level to that of our series, which was about half that of the control series of McKissock *et al.*

Morbidity is more difficult to assess, there being no well defined end-point. McKissock *et al* classified their patients as being at full work, partially disabled, and totally disabled, whereas Krayenbuhl *et al*[7] had good and poor groups. When doubt existed in our assessment of patients the poorer category was chosen. The McKissock *et al* control series when assessed at six months showed that 10% were partially disabled and 5% totally disabled. Comparison is difficult; allowing for variations in assessment there would seem to be little difference in outcome between the control series, our series, and the series of Krayenbuhl *et al* (12% poor results).

Hypertensive patients pose a particular problem. If untreated at the time of surgery they seem to do badly: 11 out of 20 untreated hypertensive patients had a poor result or died. Although the numbers are small, our results suggest that hypertensive patients might do better if treated before surgery. Experience of further cases (unpublished data) reinforces the view that bringing the blood pressure under control in the interval between subarachnoid haemorrhage and surgery improves the results of surgery.

Cerebral vasospasm remains a major problem for which no treatment is effective once it occurs. It is common but affects the outcome of surgery little unless it is generalised, when it often results in death or disability.

Further improvement in the results will depend on better control of the blood pressure, careful timing of surgery, prevention of rebleeding, and control of vasospasm.

We thank Mr R S Gye for permission to include patients who were under his care.

References

1 Pakarinen, S, *Acta Neurologica Scandinavica*, 1967, **43**, suppl No 29.
2 Crawford, M D, and Sarner, M, *Lancet*, 1965, **2**, 1254.
3 McKissock, W, Richardson, A, and Walsh, L, *Lancet*, 1965, **1**, 873.
4 McKissock, W, Richardson, A, and Walsh, L, *Lancet*, 1960, **1**, 1203.
5 McKissock, W, Richardson, A, and Walsh, L, *Lancet*, 1962, **2**, 417.
6 Botterell, E H, *et al*, *Journal of Neurosurgery*, 1956, **13**, 1.
7 Krayenbuhl, H A, *et al*, *Journal of Neurosurgery*, 1972, **37**, 678.

Diagnostic and therapeutic assessment by telephone electrocardiographic monitoring of ambulatory patients

Y HASIN, D DAVID, S ROGEL

British Medical Journal, 1976, **2**, 609-612

Summary

The electrocardiograms of ambulatory patients have been monitored over the telephone by staff of the intensive cardiac care unit using equipment in the unit. Telephone monitoring is a useful way of diagnosing transient symptomatic arrhythmias and a reliable aid in supervising the patient's rhythm at the beginning or end of treatment. The doctor has direct contact with the patient at the time of his symptoms so that he can reassure or instruct him. This system costs relatively little in manpower and equipment and permits relatively long periods of follow-up. It is effective, however, only in symptomatic cases in which the rate or rhythm disturbances last long enough to be transmitted. Also important are the negative findings when the patient complains of symptoms and abnormal findings during routine telephone transmissions. Accurate detection of transient ischaemic changes seems to be less reliable, and further technical improvements are required.

Intensive Cardiac Care Unit, Hadassah Medical Centre, Jerusalem, Israel

Y HASIN, MD, resident
D DAVID, MD, senior resident
S ROGEL, MD, professor of medicine

Introduction

The introduction of the Holter monitoring system has greatly advanced the diagnosis and treatment of transient or phantom arrhythmias.[1-7] This recording system can usually be applied only for a day or two, however, the recorded electrocardiogram cannot be read immediately, and analysis of the tracings takes time and requires expensive equipment. In contrast, telephone transmission of the electrocardiogram demands simple equipment, permits immediate analysis of the tracings and prompt instruction of the patient, and enables a longer follow-up. We report here the results of monitoring ambulatory patients by telephone from an arrhythmia-detecting centre located in an intensive cardiac care unit.

Patients and methods

A small, portable, battery-powered, transistorised electrocardiogram pre-amplifier is connected to a modulation system. The device is connected to the patient's chest by three electrodes located so that distinct P waves, QRS complexes, and ST-T segments are obtained. The modulator converts the electrocardiographic signals into sound with a frequency range of 1600-2100 Hz. The conventional telephone line permits transmission to a demodulator with a frequency cut-off of 1500-2200 Hz and a frequency response DC to 100 Hz. The telephone calls are received through an unregistered telephone line, the number of which is known only to the patients. The decoded tracings are viewed on an oscilloscope and simultaneously recorded on an electrocardiograph and a magnetic tape. This receiving station serves as an arrhythmia detecting centre. It forms an integral part of the intensive cardiac care unit. The transmitted electrocardiograms are received by a nurse, who identifies the patient, takes note of the relevant clinical information, and by analysing the incoming tracing decides whether the cardiologist should be consulted at once for instruction to the patient or whether the recording can be filed for later analysis.

Patients were included in the study if they had suspected transient arrhythmias or ischaemic events, or if drug treatment of known arrhythmias was being evaluated.

The patients referred to the arrhythmia detecting centre were told how to use the transmitter by the nurse on duty. Every patient was advised to transmit his electrocardiogram several times a day at predetermined times ("routine transmissions") in order to ascertain that satisfactory recordings could be obtained and also to evaluate treatment or diagnose symptomless phenomena. The patients were also urged to contact the centre as soon as their symptoms appeared.

On completion of follow-up, which lasted from one day to several weeks, depending on the nature of the clinical problem, the patients were given representive tracings with suggestions about treatment and told to report to their doctor.

Results

Two hundred patients were studied by the arrhythmia-detecting centre. One hundred and nineteen (59·5%) were referred for diagnosis of probable arrhythmias, 49 (24·5%) for evaluation of antiarrhythmic treatment, and 32 (16%) for objective diagnosis of suspected ischaemic episodes.

TABLE I—*Relation between indication for referral and results of survey*

Problem	No of patients	Conclusion after telephone monitoring		
		Definite	Probable	None
Arrhythmias ..	119	105 (88%)	2 (2%)	12 (10%)*
Treatment ..	49	45 (92%)		4 (8%)
Ischaemia ..	32	9 (28%)	20 (63%)	3 (9%)
Total	200	159 (79·5%)	22 (11%)	19 (9·5%)

*Follow-up was incomplete in three uncooperative patients

A definite conclusion was reached in most patients (table I). A probable diagnosis was made in 22 patients, most of whom suffered from ischaemic heart disease but had equivocal ST-T changes with their symptoms. During follow-up only 19 patients did not experience the symptoms that required clarification, so no conclusions could be reached. In three cases follow-up had to be interrupted because the patient would not co-operate.

DYSRHYTHMIAS AND ISCHAEMIC EVENTS

Table II summarises the correlation between the clinical diagnosis and the results of monitoring. In most patients referred for arrhythmias well-defined rhythm disturbances were detected, explaining the clinical manifestations for which the patients were referred. Twenty-four patients suspected of having paroxysmal arrhythmias had regular sinus rhythm with a normal heart rate during episodes of palpitations. On the other hand, disturbances were detected during routine transmissions in seven other patients not suspected of having arrhythmias. In 15 patients the minor rhythm abnormalities detected on routine transmission could not account for the patient's complaints. Possibly, however, these were induced by a prolonged arrhythmia of the same type.

TABLE II—*Relation between symptoms and findings on telephone electrocardiograms*

Problem	No of patients	Symptoms and findings	Symptoms and no findings	Findings but no symptoms	Findings unrelated to symptoms
Arrhythmias	119	73 (61%)	24 (20%)	7 (6%)	15 (13%)
Ischaemia	32	11 (34%)	19 (59%)	2 (6%)	0

In 32 patients with normal resting electrocardiograms the clinical manifestations suggested ischaemic events. In 11 diagnostic ST-T changes were detected during chest pain, whereas in 19 other patients no significant ischaemic changes were observed during pain. Typical transient electrocardiographic changes due to myocardial ischaemia were also observed during routine transmission in two patients.

TABLE III—*Classification of telephone-transmitted dysrhythmias*

Type of arrhythmia	No of patients	Type of arrhythmia	No of patients
Sinus tachycardia	20	Paroxysmal atrial fibrillation	21
Sinus bradycardia and arrest	8	Junctional extrasystoles ..	18
Supraventricular extrasystoles	23	Ventricular extrasystoles ..	47
Paroxysmal supraventricular tachycardia	9	Ventricular tachycardia ..	2
Paroxysmal atrial flutter ..	14	Pacemaker function ..	21

The incidence of the various arrhythmias detected is summarised in table III. Supraventricular arrhythmias formed 65% of all the arrhythmias detected. They included many long sustained or short recurrent bouts of atrial tachyarrhythmias. On the other hand, rhythm disturbances of ventricular origin were mostly in the form of single ventricular premature beats except for two episodes of ventricular tachycardia.

TABLE IV—*Telephone monitoring during adjustment or cessation of drug treatment*

Monitoring	No of patients	Conclusions	No of patients
During treatment	38 (77·5%)	Dysrhythmia suppressed	34 (89%)
		Ineffective treatment	4 (11%)
After discontinuation of treatment	11 (22·5%)	Treatment may be discontinued	6 (54·5%)
		Treatment should be resumed	5 (45·5%)

TREATMENT

In 49 patients in whom the nature of the transient arrhythmia was known and who received antiarrhythmic drugs telephone monitoring was used to evaluate the efficacy of treatment (table IV). In 34 of these patients medication could be so adjusted until the patients were free of symptoms and the transmitted tracings were free of rhythm dis-

turbances. Only four patients had to be admitted to hospital for further treatment. The surveillance period in this group varied from seven to 27 days. Repeated transmissions four times a day for three weeks while gradually reducing the dose of antiarrhythmic drugs proved that treatment could be discontinued safely in six patients. Reappearance of the arrhythmia warranted reinstitution of treatment in five patients.

Case 1—A 23-year-old man who underwent double valve replacement was referred to the arrhythmia detecting centre because of recurrent episodes of palpitations. Only after 10 days of follow-up showing normal sinus rhythm was paroxysmal supraventricular tachycardia recorded (fig 1). Quinidine treatment was started, and he recovered completely.

Case 2—A 26-year-old woman complained of palpitations and light-headedness. The first routine transmission disclosed a short episode of ventricular tachycardia. Other characteristics of unstable ventricular rhythm were also recorded (fig 2). Consequently verapamil treatment was started, and the arrhythmia and symptoms disappeared.

Case 3—A 72-year-old woman was suffering from short episodes of extreme weakness and perspiration without loss of consciousness. These episodes recurred for several months, but repeated ambulatory examinations showed no disease. After two weeks of follow-up by telephone-transmitted electrocardiogram a short episode of sinus arrest with slow junctional escape rhythm was recorded (fig 3). The patient was immediately admitted to the hospital and implanted with a demand pacemaker.

Case 4—A 79-year-old man received long-term procainamide treatment after an episode of ventricular tachycardia following myocardial infarction. An attempt to discontinue treatment was supervised by telephone control, which disclosed frequent premature ventricular beats requiring immediate reinstitution of the antiarrhythmic treatment (fig 4).

Case 5—A 54-year-old woman suffering from cardiomyopathy was known to have intermittent complete atrioventricular block, which required implantation of a demand pacemaker. During a long control period of telephone surveillance bouts of paroxysmal ventricular tachycardia were repeatedly observed. Despite increasing the doses of the various antiarrhythmic drugs the dysrhythmia still recurred. Increasing the pacing rate to 100/min controlled the dysrhythmia (fig 5).

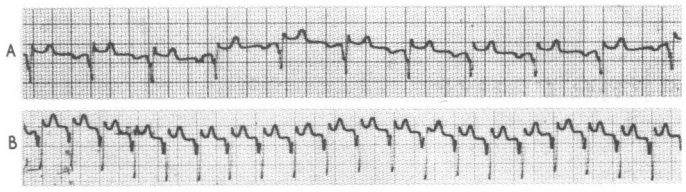

FIG 1—Case 1. Normal sinus rhythm, which lasted for 10 days (A). Paroxysmal supraventricular tachycardia (B).

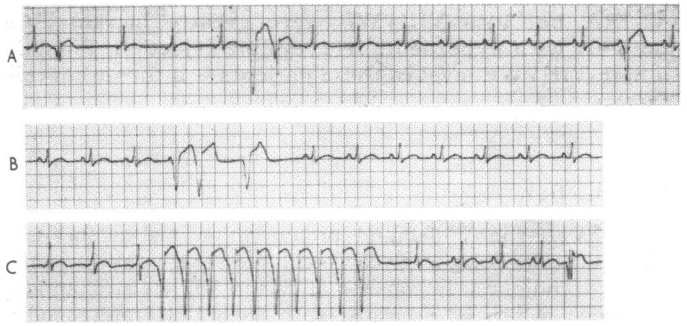

FIG 2—Case 2. Junctional rhythm interchanging with sinus rhythm (A). Multiple ventricular premature beats with changing coupling interval and interectopic intervals (A and B). Paroxysmal ventricular tachycardia (C).

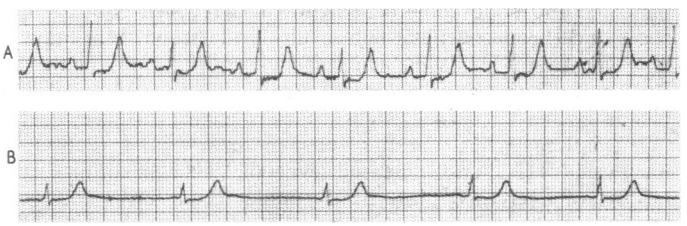

FIG 3—Case 3. Normal sinus rhythm (A). Sinus arrest with slow junctional escape rhythm (B).

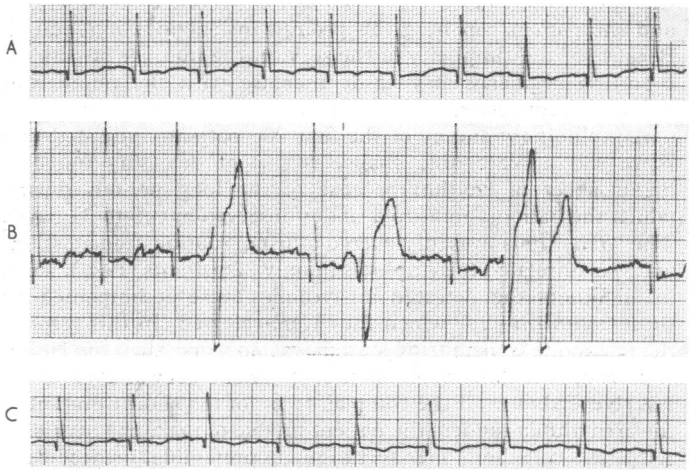

FIG 4—Case 4. Regular sinus rhythm before stopping procainamide treatment (A). Multiple ventricular premature beats (B). Undisturbed sinus rhythm after reinstitution of drug treatment (C).

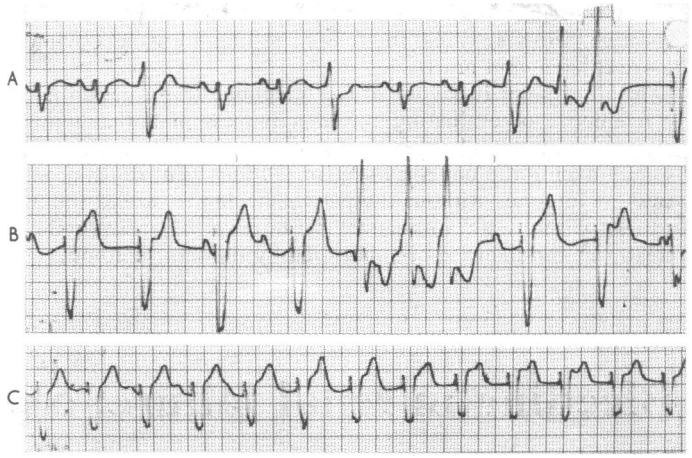

FIG 5—Case 5. Sinus rhythm with right bundle-branch block (A) or demand pacemaker function (B) interrupted by multiple ventricular ectopic beats. Overdrive pacing at rate of 100/min suppressed dysrhythmia (C).

Discussion

Monitoring the electrocardiograms of ambulatory patients is becoming increasingly popular in the search for transient phenomena such as paroxysmal arrhythmias and ischaemic changes. It also enables coronary artery disease to be detected and various therapeutic regimens to be evaluated.[1–11]

The most widely used ambulatory monitoring system is the Holter device, which provides a continuous tape recording of the electrocardiogram. The advantage of this method is that the heart's electrical activity is recorded during the patient's daily activities, irrespective of symptoms and the duration of the abnormality. The follow-up period is usually limited to 24-48 hours, however, since the equipment for recording and rapid scanning is rather expensive. Transient dysrhythmias do not necessarily occur every day or two, which reduces the likelihood of their detection. Another disadvantage of this method is that the information obtained is retrospective and no immediate medical advice can be given even if the recorded electrical phenomena would fully justify it.

Another method of ambulatory monitoring uses the conventional telephone line to transmit the patient's electrocardiogram by converting the electrical signals into voice frequency.[7 8] We have used such a method, which uses relatively inexpensive equipment and a simple mode of transmission that permits prolonged observation. The trained personnel and standard equipment of an intensive cardiac care unit are also used. With

this round-the-clock service data can be recorded and analysed and the patient can be given prompt instructions if required. Because of the simplicity of the service, the number of patients reporting regularly may easily increase with the number of available transmitters.

A preliminary survey of the first 200 patients indicated that there was a reasonable chance (88%) of detecting transient disturbances of rhythm because of the long periods during which the patients remained under observation (up to 31 days). The arrhythmia was discovered during routine transmissions and was unrelated to symptoms in only 22 patients (19%), while in all others the detected arrhythmias were accompanied by those complaints for which they were referred. In this respect, the telephone transmitting system was no worse than the Holter technique, which detects arrhythmias even if they are symptomless. Nevertheless, if the complaints are of too short duration or the arrhythmia renders the patient unable to make the necessary telephone call the Holter technique is preferable, provided the dysrhythmia occurs on the day of monitoring.

The immediate analysis and corresponding therapeutic measures that were taken in the six patients in whom multifocal ventricular ectopic beats or short runs of ventricular tachycardia were detected could not have been carried out with the Holter technique since the scanning in this method is always retrospective. In these cases, the telephone monitoring is obviously more efficient and practical.

Another point in favour of the telephone transmission is the convenience of evaluating the effectiveness of antiarrhythmic treatment given. The repeated contact with the medical personnel during the day allows optimal adjustment of drug doses at short intervals without the patient having to attend the outpatient clinic. Safe ambulatory withdrawal of antiarrhythmic treatment can likewise be supervised by the same method.

The results in patients in whom chest pain was considered ischaemic were rather disappointing. A definite electrocardiographic change during pain was discovered in less than a third of the patients, while in the other patients the ST-T changes were not of diagnostic value. In eight of the 32 patients suspected of having angina pectoris the symptoms were accompanied by rhythm disturbances but not by diagnostic ST-T changes. The complaints disappeared on successful treatment of the dysrhythmia.

Another important aspect of this type of patient surveillance is that it helps to exclude the existence of significant cardiac rate or rhythm disturbances in patients suffering from symptoms resembling cardiac arrhythmia. If recordings are normal when symptoms are present unnecessary medication can be prevented and the patient can be reassured. In many such patients symptoms disappeared after they were reassured.

Requests for reprints should be addressed to Dr S Rogel, Intensive Cardiac Care Unit, Hadassah Medical Centre, Jerusalem, Israel.

References

1 Holter, N J, *Science*, 1961, **134**, 1214.
2 Gilson, J S, Holter, N J, and Glasscock, N R, *American Journal of Cardiology*, 1964, **14**, 204.
3 Corday, E, *et al*, *Journal of the American Medical Association*, 1965, **193**, 417.
4 Stern, S, *et al*, *Israel Journal of Medical Sciences*, 1970, **6**, 103.
5 Bleifer, S B, *et al*, *Progress in Cardiovascular Disease*, 1974, **15**, 569.
6 Cannom, D S, and Harrison, D C, *American Journal of Cardiology*, 1974, **33**, 399.
7 Lipski, J, *et al*, *American Journal of Cardiology*, 1976, **37**, 102.
8 Peter, T, *et al*, *Lancet*, 1973, **17**, 1110.
9 Danilevicius, Z, *Journal of the American Medical Association*, 1974, **229**, 1975.
10 O'Rourke, R A, and Ross, J, jun, *Annals of Internal Medicine*, 1974, **81**, 695.
11 Stern, S, and Tzivoni, D, *British Heart Journal*, 1974, **36**, 481.
12 Stern, S, Tzivoni, D, and Stern, Z, *Circulation*, 1975, **52**, 1045.

Bleeding in renal failure: a possible cause

M KAZATCHKINE, Y SULTAN, J P CAEN, J BARIETY

British Medical Journal, 1976, **2**, 612-615

Summary

Increased concentrations of factor VIII-related antigen (VIIIRA), factor VIII-procoagulant activity (VIIIC), and decreased factor VIII-von Willebrand activity (VIIIVWF) were found in the plasma of patients with chronic renal failure (CRF). This functional abnormality of the factor VIII protein may partly explain the prolonged bleeding time commonly found in CRF. It was not improved by dialysis, but it was no longer found in patients with normally functioning grafted kidneys after the sixth month after transplantation. VIIIVWF levels remained decreased when compared with VIIIRA or VIIIC in transplanted patients undergoing acute reversible rejection soon after transplantation. Yet, not only VIIIC and VIIIRA but also VIIIVWF were greatly increased in patients with hyperacute irreversible rejection. Possibly a high VIIIVWF level in these patients is a thrombogenic factor.

Laboratoire d'Hémostase et de Thrombose Expérimentale, INSERM U 150 et CNRS ERA 335, Hôpital Saint-Louis, et Groupe de Recherches sur la Pathologie Rénale et Vasculaire, INSERM U 28 et CNRS ERA 48, Hôpital Broussais, Paris, France

M KAZATCHKINE, MD, physician
Y SULTAN, MD, physician
J P CAEN, MD, FRCPATH, pathologist
J BARIETY, professor of nephrology

Introduction

Factor VIII-von Willebrand protein in normal plasma is a high-molecular weight glycoprotein measurable by immunological techniques. It consists of factor VIII-related antigen (VIIIRA), which is associated with factor VIII-procoagulant activity (VIIIC or antihaemophilic activity) and with von Willebrand factor activity (VIIIVWF). VIIIVWF is detected in vitro by platelet retention on glass bead columns[1] and measured by the ristocetin-induced platelet aggregation assay.[2] The relation between factor VIII-von Willebrand protein, platelets, and endothelial cells in the early processes of haemostasis and thrombogenesis is not fully understood. It is known, however, that VIIIVWF, which is lacking in patients with von Willebrand's disease, is necessary, in vivo, for the control of skin bleeding time.

In patients with chronic renal failure (CRF) reports of increased VIIIC[3] and increased levels of VIIIRA[4] conflict with reports of decreased glass bead platelet retention[5 6] and prolonged bleeding time.[7] In the hope of clarifying this apparent discrepancy we investigated the factor VIII complex components