Interference effect of myopotentials on function of unipolar demand pacemakers

O.-J. Ohm, H. Bruland, O. M. Pedersen, and E. Waerness

From Medical Department A and Neurological Department, University of Bergen, School of Medicine, Bergen, Norway

Of 44 patients with demand pacemakers, 33 had pacemaker rhythm and were examined for failure in pacemaker function produced by myopotentials. Most patients were subjected to a standard experimental programme, with simultaneous recording of electrocardiogram and electromyogram.

In 28 of the 33 patients the pacemaker was inhibited by muscle potentials. Three patients had suffered symptoms which can be ascribed to this effect. Only one patient had symptoms in connexion with the examination. In 12 patients, lifting from 2 to 4 kg was sufficient to produce pacemaker failure.

In one patient asystole occurred during the experiment, and lasted for almost 7 seconds before idioventricular rhythm took over. Failure in pacemaker function was produced in 5 patients pressing a hand against a firm horizontal surface, and in 7 patients rising from an armchair.

The highest myopotential recorded was 3.2 mV. In some the deflection was off the scale.

The cause of inhibition is the difference in voltage between the two electrodes of the pacemaker.

In patients requiring the implantation of a demand pacemaker, greater use should be made of bipolar or ventricular-triggered pacemakers, or of fixed rate pacemakers.

In the course of the past few years there have been several publications concerning undesirable inhibition of demand pacemakers (Furman *et al.*, 1968; Sowton, Gray, and Preston, 1970; Barold *et al.*, 1971; Blaser *et al.*, 1972; Schulten *et al.*, 1972). It has recently been shown that potentials from skeletal muscle are capable of inhibiting the demand mechanism and causing temporary pacemaker failure (Wirtzfeld, Lampadius, and Ruprecht, 1972; Mymin *et al.*, 1973).

Little is known concerning the frequency and symptomatology of this complication. The object of our study was to investigate the frequency of this form of pacemaker failure, and to describe the symptomatology more closely.

Subjects and methods

During the period 1 January 1970 to 1 January 1973, demand pacemakers have been implanted in a total of 58 patients in Medical Department A. Nine patients are dead, and 5 could not be examined because of other serious illnesses. Forty-four patients were examined for failure in pacemaker function caused by myopotentials (Table 1). Of these patients, 43 had had pulse gener-

ators implanted subcutaneously in the pectoral region, 39 on the right and 4 on the left side. In 1 patient the pacemaker was implanted subcutaneously in the abdominal wall. All except 2 patients had unipolar pacemakers (Medtronic 5843 and 5943). The sex, age, and diagnosis for each patient is shown in Table 2, as well as the type of pacemaker and the smallest load which gave inhibition and escape rhythm.

The electromyogram was recorded by 2 different electromyographs (DISA, type 13A69 and type 14A21) and by concentric needle electrodes (DISA 9013K0032). Recordings were made with 3 to 5 electrodes simultaneously, one being placed praecordially on the skin (DISA 13K60). The others were placed in the lateral part of the pectoralis major muscle, approximately 10 cm from the acromion, immediately medial to, or beneath, the pulse generator; in the fifth left intercostal space in the midclavicular line; and in Case 8 in the rectus abdominis muscle. Standard lead III was simultaneously recorded by a separate electrocardiograph (Cardiomat, Type F).

Forty-three patients were examined in the supine position with the arm abducted 90° at the shoulder joint without underlying support, on the side in which the pulse generator was implanted. By putting weights in the patient's hand, and encouraging him to hold the arm in the same position, an isometric contraction of the pectoral muscle on the same side was achieved. The

Received 3 August 1973.

TABLE 1 Pacemaker type and occurrence of pacemaker failure and natural rhythm in all patients

	Men	Pacemaker failure	Not inhibited	Heart rate > 60/min	Women	Pacemaker failure	Not inhibited	Heart rate > 60/min
Medtronic 5843	6	4	0	2	3	3	0	0
Medtronic 5943	26	17	2	7	7	4	1	2
Medtronic 5942	1	0	1	0	0	0	0	0
Stanicor	1	0	1	0	0	0	0	0
	34	21	4	9	10	7	1	2

TABLE 2 Clinical details in 33 patients with pacemaker rhythm

Case No.	Sex	Age	Diagnosis	Indication for treatment	Pacemaker type	Duration of treatment (mth)	Load (kg)	Inhibition (sec)	Escape rhythm	Effect of armchair/pressure
1	M	50	AV block III	Syncope	Ventricor Stanicor	38	Ungraded	0		
2	M	75	AV block III	Syncope	5841	2				
3	F	64	AV block II, III, RBBB	Syncope	5942	5	Ungraded	0	24	
4	F	70	AV block II, III, Mobitz II, LBBB	Syncope	5843	22				
5	M	61	Atrial fibrillation, AV block III, LBBB	Syncope	5843	21	2	1.9	32	Armchair + pressure +
6	F	69	AV block I, SA block	Bradycardia	5843	20	4	1.3	48	Armchair - pressure +
7	M	49	AV block III	Bradycardia	5843	20	2	1.9	31	Armchair + pressure +
8	M	76	AV block I, II, LAH, RBBB	Syncope	5843	20	Abdominal implant	1.4	43	
9	M	56	AV block I, III, RBBB	Bradycardia	5943	16	10	1.5	38	
10	M	78	AV block I, III, RBBB	Syncope	5943	15	Ungraded	0		
11	M	69	AV block I, Mobitz I	Syncope	5843	15	4	1.4	42	Armchair + pressure +
12	M	73	AV block II, III, alternating RBBB + LAH and LBBB	Vertigo	5943	12	4	1.2	50	
13	M	68	AV block I, III, Mobitz I	Heart failure	5943	11	Ungraded	0		
14	M	60	AV block I, LBBB	Syncope	5943	10	4	3.8	Pacemaker escape	Armchair +
15	F	63	SA block, atrial fibrillation	Syncope	5943	10	2	1.1	Pacemaker escape	
16	M	52	Coronary sinus rhythm	Vertigo	5943	10	8	1.3	46	Armchair -
17	M	83	AV block I, III, LBBB	Heart failure	5943	9	Ungraded	2.0	30	
18	M	63	SA block	Vertigo	5943	8	4	1.2	50	
19	M	72	Coronary sinus rhythm	Vertigo	5943	7	6	1.3	48	Armchair +

TABLE 2 (Cont'd)

Case No.	Sex	Age	Diagnosis	Indication for treatment	Pacemaker type	Duration of treatment (mth)	Load (kg)	Inhibition (sec)	Escape rhythm	Effect of armchair/pressure
20	M	61	AV block I, III, RBBB, LAH	Syncope	5943	6	Ungraded*	5.0	Pacemaker escape	
21	M	84	AV block I, III, RBBB	Syncope	5943	6	4	3.5	17	
22	F	68	Tachybradyarrhythmia		5943	6	Ungraded	1.2	52	
23	M	74	Hypersensitive carotid sinus	Vertigo	5943	6	10	1.0	Drop in pacemaker frequency	
24	M	80	AV junctional rhythm, RBBB	Syncope	5943	5	8	3.3	Pacemaker escape	
25	M	50	AV block I, Mobitz II, LBBB	Vertigo	5943	5	Ungraded	1.2	50	
26	M	71	AV block I, III, alternating RBBB+LAH and LBBB	Syncope	5943	5	Ungraded	1.4	40	
27	F	83	AV block II, III, alternating RBBB, and LBBB	Syncope	5943	4	Ungraded	0		
28	F	69	AV block II, III, LBBB	Vertigo	5943	4	6	1.9	Pacemaker escape	
29	F	77	AV block III	Syncope	5943	4	2	1.4	Pacemaker escape	
30	M	72	Sinus-bradycardia	Syncope	5943	4	4	1.8	33	Armchair +
31	M	64	AV block I, III, LAH	Syncope	5943	3	8	1.4	41	
32	M	47	AV block III, Mobitz II	Syncope	5943	2	Ungraded	2.0	30	
33	M	65	Tachybradyarrhythmia		5943	2	6	1.2	52	

AV block I, II, III: Alternation between atrioventricular block grade I, grade II of high degree, and grade III. RBBB and LBBB: Right bundle-branch block and left bundle-branch block, respectively. LAH: Left anterior hemiblock. Load in kg refers to least weight causing inhibition. 5841, 5843, 5942, 5943 are all Medtronic pacemakers. Ungraded*: Inhibition when the patient placed his right arm across the chest.

Armchair: Pressure: See text.

least weight resulting in inhibition of the pacemaker was noted. In some patients the load had to be altered, and other arm positions, and magnitude and direction of load, had to be tried before inhibition of the pacemaker was achieved.

In addition to examination in the supine position, 9 patients were examined while rising from a chair, assisting themselves by pressing their arms against the armrests.

Five patients were also examined in a standing position, while pressing a hand lightly against a firm horizontal surface.

The patient with the pulse generator implanted in the abdomen was examined while sitting up from a supine position without using his arms.

Case reports

Three patients had symptoms that could be ascribed to inhibition by myopotentials.

Case 20 suffered his first syncope in the beginning of October 1960. After numerous syncopes he was admitted to the department in June 1972. A week later a demand pacemaker was implanted in the right pectoral region, with a unipolar endocardial electrode.

In the middle of September 1972, he again suffered attacks of vertigo and syncope. Failure in pacemaker function was seen, and was ascribed to unstable electrode position, which was corrected. In the following months the tendency towards vertigo continued, especially if he put his right arm across his chest, or if he used his arms

FIG. 1 Case 20. Cardiac arrest of almost 4 seconds' duration occurred when the patient laid his right arm across his chest. The patient then complained of vertigo and the experiment was broken off. No idioventricular rhythm occurred. Electromyogram was registered from a needle placed laterally in the right pectoralis major muscle. Lowest strip in all figures is standard lead III in electrocardiogram, recorded simultaneously.

to assist in rising from a chair. Fig. 1 shows simultaneous registrations of electrocardiogram and electromyogram from the patient while he is putting his right hand against his left shoulder. The myopotentials of this patient measured 2.0 to 2.1 mV during inhibition.

A fixed rate pacemaker was later implanted in this patient, and has functioned satisfactorily.

Case 4 had, for 2 years, suffered a tendency towards attacks of vertigo, with syncope of short duration. On 20 April 1971, a demand pacemaker was implanted with an endocardial electrode. The patient has subsequently on several occasions noticed a tendency towards vertigo while working with her arms above her head.

In this patient failure in pacemaker function was produced by weight load in supine position, on the patient rising from an armchair, and on exercising a slight pressure with her right hand against a firm surface at chest height.

Case 32 had a demand pacemaker and an endocardial electrode implanted in October 1972, because of multiple syncopal attacks and complete atrioventricular block. He has subsequently suffered attacks of vertigo on several occasions, with pulse frequencies between 50 and 60. Here again inhibition of pacemaker function was produced by muscle contraction. On the other hand, it was found that certain QRS complexes did not inhibit pacemaker function. Some premature beats, however, recognized by the pacemaker, did not result in pulse waves

and pulse rate falling below 50 because of the hysteresis mode of action of the pacemaker.

Results

At the time of examination 33 of the patients had pacemaker rhythm, and it was only in these that the effect of myopotentials on pacemaker function could be assessed. In 28 patients contraction of the skeletal muscle in the immediate vicinity of the pulse generator led to inhibition of the demand function.

In 12 of the patients, a load of 2 to 4 kg on the outstretched arm was sufficient to cause inhibition. In some patients the load direction was altered to give a contraction in the part of the pectoral muscle that lay nearest the pulse generator.

The duration of cardiac arrest varied with the idioventricular rhythm. In Case 14 cardiac arrest of almost 7 seconds' duration was registered while lifting 6 kg (Fig. 2).

In some patients asystole of varying duration was interrupted by 1 to 2 pacemaker escape beats (Fig. 3).

In some patients, there was only a short prolongation of the RR interval between the last pacemaker

FIG. 2 Case 14. Lifting 6 kg and simultaneously raising right arm resulted in an arrest of ventricular activity for 6.5 sec, before idioventricular rhythm ensued. Electromyogram from right pectoralis major muscle near pacemaker (top) and deep in intercostal muscle over apex (middle).

FIG. 3 Case 7. Partial inhibition of pacemaker with pacemaker escape corresponding to a rate of 33 per minute on lifting 4 kg. Electromyogram from right pectoralis major muscle, medial to pacemaker (top) and lateral to pacemaker (middle).

impulse and the natural escape, corresponding to a heart rate between 30 and 50 a minute.

Inhibition during constant muscle contraction could be terminated by placing a ring magnet over the pulse generator, but was still present when the magnet was removed (Fig. 4).

Our patient (Case 8), in whom epicardial leads were used, had the pulse generator implanted in his

abdominal wall. Inhibition of the pacemaker was produced when he rose from a supine to a sitting position.

Discussion and conclusions

This study has shown that myopotentials are capable of producing inhibition of the demand function in 28 of 33 patients (85%).

FIG. 4 Same patient and electromyogram recordings as in Fig. 2 during contraction of right pectoralis major muscle on lifting 4 kg. Only P waves could be seen in the electrocardiogram until a magnet was placed over the pulse generator. On removal of magnet circulatory arrest returned, and remained until patient terminated muscle contraction.

The lowest myopotentials registered simultaneously with inhibition had in some cases an amplitude of <1.5 mV. The apparent effect on pacemaker function of myopotentials of such low voltage may be partly due to the recording technique. The concentric electrode registers motor-unit potentials from a very limited area of the muscle. It is probable that potentials with higher voltages and different frequencies are present in other areas, and that these affect the pacemaker without being registered by the EMG electrode.

In one of the patients (Case 27) the force exerted was slight. The myopotentials registered were weak, and the frequency far less than in a normal interference pattern, which may explain why the pacemaker was not affected in this case.

Only contraction of the muscle in the immediate vicinity of the pulse generator produced inhibition. Contraction of the contralateral pectoral muscle produced no inhibition, though motor-unit potentials with high voltage were recorded.

It is reasonable to assume that the cause of inhibition is the net difference in voltage measured between the pulse generator and the electrode tip, caused by the subcutaneous position of the pulse generator. Inhibition is most likely to occur if the anode and cathode are situated on opposite sides of the contracting muscle. In one patient, however, we produced inhibition though the pulse generator was implanted under the pectoral muscle.

One way of reducing the frequency of this complication might be to place the pulse generator on the side opposite to the dominant hand.

The area of the indifferent electrode does not seem to be of major importance as the frequency of inhibition was even higher in Medtronic 5843 (diameter 2.5 cm) than 5943 (diameter 6.4 cm).

We have not had the opportunity of examining ventricular-triggered pacemakers. Other workers have found that these pacemakers react to myopotentials by an increase in frequency up to the listed maximal frequency (Wirtzfeld *et al.*, 1972). The effect on ventricular-triggered pacemakers does not, in most cases, appear to be as serious as in those with a ventricular-inhibited mechanism, as a high heart rate for the duration of a few seconds is tolerated better than a corresponding asystolic period. On the other hand, the long refractory period of this type of pacemaker prevents inhibition by a QRS complex in this phase, and the succeeding pacemaker impulse will then coincide with the T wave in the vulnerable phase (Castellanos *et al.*, 1969).

Inhibition of demand pacemakers produced by myopotentials from skeletal muscle or electric interference of the pulsed-energy type is a more serious complication than inhibition by external electrical

apparatus of the continuous wave type, because the pacemaker will, in the latter case, function asynchronously with the interference rate.

Patients with slow or non-existing idioventricular rhythm may suffer a circulatory arrest of such long duration that life-threatening tachyarrhythmias, or myocardial or cerebral damage, may ensue. Only one patient (Case 20) suffered symptoms in the form of vertigo in this study. It is possible that sudden death during permanent treatment with demand pacemaker is due to complications of this type.

In the experimental programme we attempted to reproduce some of the muscular movements to which patients are often exposed in daily life. Inhibition of pacemaker function was produced in 7 of 9 patients during the act of rising from an armchair, and in all 5 patients during pressure of the hand against a firm horizontal surface.

Bipolar demand pacemakers will probably not be affected by myopotentials, as the relative position of the electrodes gives a lower difference in voltage between the poles.

The use of a metal capsule around the pulse generator (Medtronic 5942 and 5943) reduces the risk of an electromagnetic field directly affecting the electronics of the pacemaker, with the result that the pacemaker will operate at the interference frequency. This form of screening of the pulse generator is not capable of preventing undesirable inhibition from myopotentials. Patients requiring demand pacemakers should therefore to a greater extent be fitted with bipolar systems or ventricular-triggered pacemakers, unless fixed rate pacemakers are used. The manufacturers give the sensitivity to R-wave potentials of the different pacemakers as 1.5–2.5 mV (Medtronic 5843–5943 and Stanicor). As of 23 June 1971, the R wave sensitivity of Medtronic 5943 was altered to 2.3–3.7 mV. The highest potential measured from the pectoral muscle in this study was 3.2 mV, though in 13 patients the maximal voltage could not be measured because the deflection was off the scale. If the use of unipolar demand pacemakers is to continue, the sensitivity to incoming wave forms as well as amplitude should be altered. The present study indicates that this would reduce inhibition.

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Requests for reprints to Dr. O.-J. Ohm, University of Bergen, School of Medicine, Medical Department A, Haukeland Hospital, 5000 Bergen, Norway.