Safety of Asynchronous Ventricular Pacemaker Stimulation

Asynchronous ventricular pacemaker stimulation occurs during magnet application, sensing of electromagnetic interference, undersensing of spontaneous rhythms and resulting from technical defects of pacemakers. In the case of interference, this is intended to prevent pacemaker inhibition over a longer period of time, thus causing asystole in pacemaker-dependent patients.

The safety of this asynchronous ventricular stimulation in patients with intrinsic rhythm is the subject of discussion in Germany, as there are no controlled studies available about this topic. This has, among other things, led to controversial discussion in technically oriented standards committees. This statement brings together, therefore, the current state of knowledge about the safety of asynchronous ventricular stimulation in order to enable an adequate assessment.

In the beginning of pacemaker therapy in the 1960s, asynchronous ventricular stimulation in the VOO mode was the only pacing mode available. Here, the competition between ventricular stimulation and the heart’s own rhythm was called parasystole (10). Due to stimulation in the vulnerable phase of the T-wave, parasystole was linked to cases of sudden death caused by induction of ventricular fibrillation (2, 4, 17, 21).

There are case reports available in which, during magnet application with resulting asynchronous stimulation, ventricular fibrillation was induced (5, 6, 9, 11, 18, 23). These cases are discussed further below. In contrast, an asynchronous stimulation through magnet
application is regularly performed during pacemaker follow-up as a part of an everyday routine, without problems being reported. According to the current guidelines for pacemaker therapy, the magnet application and with it asynchronous stimulation, are a regular part of pacemaker follow-up (13,14). Similarly, in the current American guidelines for telephone monitoring – that is, without a doctor being present – a magnet application for 6 to 9 seconds by the patient is recommended (8). The following statement on the safety of asynchronous ventricular pacemaker stimulation is based on the literature available and, further, on an international survey about the safety of asynchronous stimulation during pacemaker follow-ups.

Pathophysiology

A stimulus will only be relevant, if it induces an action potential during the vulnerable phase of the T-wave, that is, during the relative refractory period. This requires clearly above threshold stimuli. A current review (1) addresses the pathophysiology of the risks of an asynchronous ventricular stimulation. Through this, a stimulated extrasystole can be induced, which in some circumstances can result in circus movement excitations. Pre-conditions are, however, the occurrence of unidirectional pathway blocks and a sufficient conduction delay. In particular, patients with prior myocardial infarction, reduced left ventricular function and frequent spontaneous arrhythmias appear to be at risk. In normal hearts, ventricular fibrillation can be induced by above threshold stimuli during the vulnerable phase within the relative refractory period, of the heart muscle cells. Therefore are mostly higher voltages and currents necessary, than normally programmed in pacemakers.

It has been proven experimentally that, in order to induce ventricular fibrillation with a pacing impulse, there must be 12 to 15 times the power usually required for the depolarization of cells which, in the normal case of implanted pacemakers, is not generated. A drop in the threshold is conceivable under certain conditions, for example, electrolyte disorders. The authors conclude that above threshold stimulation in the vulnerable phase, as pathomechanics of proarrhythmia through asynchronous ventricular stimulation, is not very likely. Zehender writes 1999 in a review about sudden cardiac death in pacemaker patients: "Without doubt, however, it can be assumed thereof, that in the vast majority of cases the sudden cardiac death is a consequence of pre-existing underlying disease in patients and thereby fully independent from pacemaker therapy. The risk of inducing malignant arrhythmias increases the more the underlying disease predisposes for spontaneous arrhythmias (24).

Literature Review

Whereas some authors link an stimulation into the vulnerable phase of the T-wave with cases of death in pacemaker patients (2, 4, 17, 21), have other authors, in contrast, observed no induction of malignant arrhythmias through a parasystolie (11, 19, 25). In a review regarding parasystolie, von Knorre (1970) comes to the following conclusion: "Under only rare adverse conditions pacemaker parasystolie may result in life-threatening tachyarrhythmias. The typically applied impulses do not have the intensity to trigger
ventricular fibrillation" (10). Caused by myocardial diseases, cardiac decompensations, metabolic and electrolyte disorders, digitalis overdose, oxygen deficiency, operations and anesthesia, the fibrillation threshold may nevertheless be lowered (10).

This is supported by case reports about the induction of malignant arrhythmias through asynchronous stimulation. Here, the following patient groups were nearly always affected: patients with acute myocardial infarction (5, 9, 11, 18, 23) and patients with severe electrolyte disorders (6). These patients are acutely ill and require hospital treatment. They are, regardless of their pacemakers, at risk of malignant arrhythmias.

Malignant ventricular arrhythmias were also triggered through asynchronous stimulation in patients with severe left ventricular function dysfunction and spontaneous ventricular tachycardias (3, 6, 7, 12, 22). These patients are also at risk of sudden cardiac death, independent of whether or not they have pacemakers implanted.

There is only one case report in which an asynchronous stimulation triggered ventricular fibrillation in a patient without myocardial infarction, electrolyte disorders or digitalis intoxication (20).

**Survey about the Safety of asynchronous Stimulation**

The survey was answered by 102 pacemaker specialists worldwide (15, 16). The survey participants were active in pacemaker therapy for between 3 and 40 years (mean 20 ± 8 yrs, median 20 yrs). The cumulative pacemaker experience of all survey participants was 1,974 years. The number of pacemaker follow-ups which were performed by the survey participants ranged from 50 to 12,000 per year (mean 2326 ± 2082, median 1550). A total of 230,305 pacemaker follow-ups were performed by the survey participants per year.

In total, 34 / 102 (33%) survey participants have experienced an arrhythmia induction in 48 cases. An arrhythmia induction by asynchronous pacemaker stimulation was reported by 14 survey participants in Germany (34%), by 11 in Europe (34%) and by 9 of the remaining survey participants worldwide (31%). Of these, 25 events were insignificant (single extrasystoles) or clearly caused secondarily by myocardial ischemia and/or electrolyte disorders.

23 cases were classified as severe. In 13 patients, ventricular fibrillation or sustained ventricular tachycardia were induced. Of these, one patient died. In 4 / 13 patients, the left ventricular function was severely reduced. In further 10 cases, there was no specific information available, hence these are counted with the severe cases, in order to avoid an underestimation.

A risk assessment on the basis of the collected data is, due to the limitations of a survey (see below), affected by inaccuracy and, therefore, cannot be compared with a controlled study. For such a risk assessment, the years of pacemaker experience are the likeliest reliable
reference parameter. If one were to refer the number of serious cases on this data, it would give the result that for the 102 surveyed physicians, a significant arrhythmia through asynchronous ventricular stimulation during pacemaker follow-up would be anticipated every 85.5 years. The survey participants conduct around 2.3 million pacemaker follow-ups in ten years. If one were to correlate the serious cases of arrhythmia induction only with this data, this would correspond to a incidence of 0.001%. As the pacemaker experience is, however, at a median of 20 years and here 10 cases were included in the calculation – in which there were no specific information available – the actual incidences may be rather lower.

The last arrhythmia induction by asynchronous stimulation dates back to 1996. Consequently, in the 7 years prior to the conducting of the survey in 2003, no induction of arrhythmias by asynchronous stimulation was reported. One possible explanation for this is, that through modern electrodes with lower pacing thresholds, ever lower impulse amplitudes can be programmed. Hence, the manufacturer programmed impulse amplitudes of the pacemaker are also lower than in former times. For an effective stimulation during the relative refractory period, however, significantly above threshold impulses are required, which possibly can no longer be achieved.

**Limitations:** The presented data is derived from a retrospective survey and reflects the memory of the survey participants. It is not derived from a controlled scientific study but, rather reflects many years of experience in pacemaker therapy. Cases could have been forgotten. In contrast, the double mention of cases also is possible.

Nowadays, an asynchronous stimulation is no longer induced during every pacemaker follow-up. Through magnet application, the stimulation rate is increased in some devices and thus spontaneous rhythm is potentially suppressed. For this reason, the behavior of asynchronous stimulation by external interference and during pacemaker follow-up is not always similar.

**Summary**

The induction of ventricular tachyarrhythmias by short term asynchronous ventricular stimulation is a rarity. The seldom events occur probably only in patients with an organic heart disease, especially acute myocardial ischemia, reduced left ventricular function and spontaneous ventricular tachyarrhythmias or with severe electrolyte disorders. Asynchronous ventricular stimulation, as it occurs during magnet application during pacemaker follow-up, is therefore only associated with a minimal risk.

This does not mean that pacemaker patients could or should be exposed unconsidered to electromagnetic interference, with consecutive device interactions. Basically, interference must be avoided as much as possible. Furthermore, other possibilities of interference between implants and electromagnetic fields are not assessed in this statement about asynchronous ventricular stimulation.
References


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