

Pacemaker dependency after pacemaker implantation

The issue of pacemaker dependency is a complex one as appropriately identified by Dr. Lelakowski and his colleagues (Cardiol J 2007; 14: 83–86). There have been a multiplicity of different definitions. This paper provides a valuable service to the medical community by trying to define the incidence of pacemaker dependency. One needs to keep in mind that the incidence of pacemaker dependency is actually based on the unique practices at a specific hospital or even country. In countries where the cost of a device is prohibitive or where the patients must pay for the device themselves, only the most severely symptomatic patients will receive a pacemaker. As such, in that environment, the incidence of pacemaker dependency is likely to be significantly higher than in an environment, such as the United States and Europe, where many patients have insurance and the use of pacing for improving quality of life rather than just sustaining life is far more prevalent.

For any individual practice, it is still essential to identify those patients within that practice who are pacemaker dependent as this will guide their management when a developing problem is identified with the system or an advisory notice is received from the manufacturer. Some physicians presume that if a patient is being paced most if not all the time, they must be “pacemaker dependent.” I would take issue with this definition as it is highly dependent on how the device is programmed: too short an AV delay that usurps control from the normal AV conduction system or too high a base rate will predispose to pacing the majority of the time when the patient may have a perfectly good intrinsic rhythm.

Dr. Lelakowski and his colleagues used a very common approach and one that I have also used in the implant suite at the time of a pacemaker implantation. It is not uncommon for a patient who presented with symptomatic complete AV block and was acutely managed with a temporary pacing system to overdrive suppress the escape focus such that, upon abrupt cessation of pacing, the patient would be virtually asystolic for a period of time. By gradually slowing the paced rate, the escape focus below the level of block is allowed to awaken and warm up such that it will take over. This

approach enables one to assess the signal amplitude (sensing threshold) as well as look for a current of injury pattern associated with the native complex after placement the permanent lead as part of the assessment for lead placement (see the article by Saxonhouse et al. in J Am Coll Cardiol 2005; 45: 412–417). While I have used this approach in the unique situation of permanent device implantation, I also think that it is less than optimal for assessing pacemaker dependency for the chronic device patient.

Another approach that is available in some devices or could be achieved years ago with chest wall stimulation was to abruptly inhibit the implanted pulse generator looking for an escape rhythm. I have always considered this to be potentially dangerous and have seen too many cardiology fellows effectively continue inhibition too long with profound asystole. This has the potential for adverse consequences in an elderly patient with significant cerebrovascular disease. If one uses this approach, it should **ONLY** be used with the patient lying supine on an exam table and not sitting partially upright in a geriatric chair.

My preferred approach, and I admit it is arbitrary, is to temporarily program the pacemaker to a very low rate in a non-tracking mode (VVI, AAI in the case of marked sinus node dysfunction with a single chamber pacemaker or DDI). My choice is 30 bpm. I know other physicians whose choice is 40 bpm. I go to this rate abruptly rather than slowly decreasing the rate as proposed by Dr. Lelakowski and colleagues. **IF** after 5–10 s, there is **NO** escape rhythm and the patient is totally paced at 30 bpm, I consider that patient to be pacemaker dependent and the patient’s chart is flagged as being “pacemaker dependent”. This is not to say that the patient will not have a potential escape rhythm if one is willing to wait long enough, either at a base rate of 30 bpm or with asystole but it is highly likely that if the pacing system were to fail abruptly as with a lead conductor fracture, component malfunction or transient inhibition from some external source such as electrocautery during surgery, the patient is likely to be very symptomatic and may even experience syncope or worse.

The above approach is further modified by the inclusion of symptoms since I treat patients, not just electrocardiograms or pacemakers. For the patient who promptly has an escape rhythm when the base rate is reduced to 30 bpm and is asymptomatic: I do not consider that patient to be pacemaker dependent. But if the patient starts having an increase in ectopy that may then trigger tachyarrhythmias or who expresses marked symptoms while lying supine on the exam table with their own intrinsic, albeit slow, rhythm is also labeled as being pacemaker dependent.

In the patient who is totally paced at the time of an office follow-up evaluation, part of that evaluation always includes an assessment of pacemaker dependency until I have identified the patient as being pacemaker dependent. Once identified as pacemaker dependent, the patient's chart is flagged and this diagnosis does not change even though the patient may have an escape rhythm on a future evaluations. Once I have identified the patient as being "pacemaker dependent", I do not have to repeat this evaluation at subsequent office or clinic evaluations. The reason is that, at least on an intermittent basis, the patient cannot be sure that an intrinsic rhythm will always be present should something happen to the pacing system. My patients whose indication for pacing is intermittent asystolic complete heart block (classic Stokes Adams Syncope) presenting with syncope but between their spells have an intact rhythm are also labeled as being pacemaker dependent because if something happened to their pacing system, e.g. lead fracture, they would do absolutely fine and be asymptomatic until another spell occurred and then they would not be protected.

Why is it so important to identify "pacemaker dependency"? The implanted devices are man-made devices. As good as they are, problems do develop that are then subject to a manufacturer's advisory. Depending on the identified failure mechanism, presuming that it may be abrupt loss of output without any means to monitor the patient (e.g. this would not be an accelerated battery depletion which could be monitored by more frequent office or transtelephonic evaluations), I would recommend a prophylactic replacement in the pacemaker dependent patient where as I would simply follow the patient who always had a stable escape rhythm, hence was not pacemaker dependent. By the same token, if I identified a significant trend showing a progressive rise or fall in the stimulation impedance indicative of a mechanical problem developing with the implanted lead, this would trigger a more intensive evaluation (e.g. chest x-ray to

look for a visible defect in the lead, program the output configuration from bipolar to unipolar) and possibly even the prophylactic replacement of that lead in a patient who I had identified as being pacemaker dependent where as the non-dependent patient would continue to be followed in a routine manner. Similarly, if a referring physician or the patient called reporting symptoms suggestive of a potential problem: I would be more concerned with the patient who was pacemaker dependent than one who always had had a stable slow intrinsic rhythm.

Dr. Lelakowski identified a 2.6% incidence of pacemaker dependent patients using his approach in his patient population. My approach with abruptly decreasing the paced rate to 30 bpm rather than slowly decreasing it would probably identify a higher incidence of pacemaker dependent patient (hence, this is highly definition dependent) in that same population. I also suspect that the incidence of "dependency" will vary based on the implant selection criteria. In a country such as the United States where a majority of patients have insurance, we tend to implant a higher number of devices for quality of life rather than life itself. In China, India and other countries where the patient's must pay for their devices, the indication for an implant is usually complete heart block and the patient must be very symptomatic prior to implant — as such, the incidence of "pacemaker dependency" is likely to be higher in those countries than in the United States.

The Association for the Advancement of Medical Instrumentation (AAMI) developed a glossary some years ago under the guidance of Dr. Doris Escher. It has only been available on the AAMI website (www.aami.org/glossary/index.htm) and a definition for Pacemaker Dependent was included in this glossary. The limitation of this glossary is that so few people knew about it. It is also now outdated and I am chairing a task force for AAMI to update the Glossary. The glossary in the future will also be named in honor of Dr. Doris J. W. Escher who was passionate about having a glossary which engineers could use to understand some medical terms and clinicians could use to understand engineering terms. I am currently chairing a subgroup of physicians and representatives from industry to update the glossary. The Definition of pacemaker dependency as provided in the AAMI Pacemaker Glossary is: "PACEMAKER-DEPENDENT. There is no definition of pacemaker dependency that is generally agreed upon. This confounds the clinician's efforts to categorize those patients at greatest risk in the event of a sudden pacing-system

failure. Pacemaker dependency could be defined as being present when abrupt cessation of pacing results in bradycardia-related symptoms or signs that create an emergent or urgent clinical situation. Similarly, a history of symptoms or signs of an emergent or urgent nature prior to pacemaker implantation may constitute pacemaker dependency. From a practical standpoint, pacemaker dependency may be categorized as follows. Class 1: Patients in whom abrupt cessation of pacing results in bradycardia-related symptoms or signs that create an emergent or urgent clinical situation, or in whom there is a history of symptoms or signs of emergent or ur-

gent nature in the absence of pacing. Class 2: Patients who are asymptomatic even when the intrinsic ventricular rate is less than 30 bpm. Class 3: Patients whose intrinsic ventricular rate exceeds 30 bpm but who have never experienced an emergent or urgent clinical situation related to bradycardia. Patients in Class 3 are not pacemaker dependent.”

*Paul A. Levine, MD, FHRS, FACC
15900 Valley View Ct., Sylmar, CA 91342
Tel: 1 818 493 2342, fax: 1 818 362 2242
e-mail: plevine@sjm.com*

From the Authors

We were delighted to read prof. Paul Levine’s comment about our paper “Pacemaker dependency after pacemaker implantation”. The issue of pacemaker dependency (PD) is very important from clinical point of view since the proper identification of pacemaker dependent patients may influence therapeutic decisions, e.g. prophylactic replacement of the lead. However the definition of PD is a source of controversy. The original method we used in our study to evaluate PD is a combination of gradual slowing of pacing rate and cessation of pacing. We believe this method is more sensitive to detect intrinsic escape rhythm as compared to abrupt slowing of pacing rate. It has been well established that pacing may inhibit automaticity of a subsidiary pacemaker due to rate-dependent and pacing duration-dependent changes in the activity of the cellular membrane ion channels [1]. Therefore some patients without escape rhythm following abrupt cessation of pacing may present one during gradual decrease in the pacing rate. On the other hand an abrupt cessation of pacing better resembles sudden pacemaker malfunction and may better identify patients at higher risk in the event of sudden pacing malfunction. We found the AAMI definition of PD provided by prof. Levine very interesting and clinically useful. Certainly the recognition of PD requires simultaneous evaluation of escape rhythm

and clinical symptoms of the patient. The presence of escape rhythm does not preclude PD. The good example are patients after radiofrequency ablation of atrioventricular junction who are PD despite having escape rhythm. It is also worth emphasizing that escape rhythm as evaluated in pacemaker clinic is not always reliable in long-term follow-up. We found that in patients after radiofrequency ablation of atrioventricular node the pattern of escape rhythm behaviour was variable during the long term follow-up [2].

References

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*Jacek Lelakowski, Jacek Majewski,
Jacek Bednarek, Barbara Matecka
and Andrzej Zabek
Department of Electrophysiology,
Institute of Cardiology, Collegium Medicum
Jagiellonian University, John Paul II Hospital,
Cracow, Poland*