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Pacemakers, and heart failure monitoring devices-controlling medications and updating readings-role of pharmacists

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***Abstract*---Background:** Pacemakers and heart failure monitoring devices are critical in managing bradycardia and other cardiac conduction disorders. While conventional electronic pacemakers are effective, they present several challenges, including lead malfunction and infection risks. **Aim:** This review aims to evaluate the evolving role of pharmacists in managing patients with implanted pacemakers and heart failure monitoring devices, focusing on medication management

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and monitoring. **Methods:** The article reviews current literature on the functionality and advancements in pacemaker technology, the pathophysiology of conduction disorders, and the implications for pharmacological interventions. **Results:** Pharmacists play a crucial role in ensuring optimal medication therapy management, especially regarding anticoagulants, antiarrhythmics, and heart failure medications. They monitor drug interactions, manage side effects, and assess adherence to treatment regimens. The integration of novel pharmacological agents, such as ivabradine, offers additional strategies for heart rate control, enhancing patient outcomes. **Conclusion:** The role of pharmacists is evolving in the context of pacemaker management, emphasizing the importance of comprehensive medication reviews, patient education, and interdisciplinary collaboration to improve health outcomes for patients with heart devices.

***Keywords*---**Pacemakers, Heart Failure, Pharmacists, Medication Management, Cardiac Conduction Disorders, Patient Care.

Introduction

While humans can survive at birth without several major organs, the development of the fetus beyond 10 weeks of gestation depends on the heart [1]. Reduced cardiac contraction rate can be deadly even if the heart seems physically normal: untreated congenital heart block is linked to an 80% fetal mortality rate [2]. The heartbeat stops during postnatal life, which causes circulatory collapse and sudden death. As a result, pacemakers have had a profound impact on cardiology practices. Implanted pacemakers control heart rate in cases where the intrinsic rate falls too low. Conventional electronic pacemakers have a number of drawbacks despite their well-established efficacy, most notably lead malfunction, a short battery life, and the possibility of infections from the device. This Review explores biological pacemakers as workable substitutes for implanted devices, as well as next-generation electrical devices designed to get over these current constraints.

The cardiac conduction system is normally functioning. A typical healthy heart beats more than three billion times in its lifetime, which is ascribed to the remarkable capacity of the endogenous pacemaker to produce both spontaneous and rhythmic electrical impulses. These impulses are distributed throughout the heart by the cardiac conduction system, stimulating the atrial and ventricular pumping chambers simultaneously [3,4], and reacting suitably to autonomic nervous system modulation [5]. Electrical impulses are produced by the major pacemaker, which is housed in the sinoatrial node (SAN), which is where the right atrium and the superior vena cava meet [6, 7]. These impulses travel quickly through the left and right atrial muscles, causing the atrium to contract, and then they slow down as they approach the atrioventricular (AV) node. The atrial contraction made possible by this AV node delay supplies the ventricles with blood in preparation for their future contraction. In the event that the primary SAN fails, the AV node can also serve as a backup pacemaker, albeit at a far

slower rate [6, 7]. The electrical impulse first passes through the AV node before moving along the His bundle, which is the only electrical pathway connecting the ventricles and atria [6,7]. From there, it spreads quickly throughout the ventricles via a network of specialized Purkinje fibers that interface with the active cardiomyocytes. Differential ion channels and gap junctions are present in cells located in different parts of the conduction system, which result in different action potential profiles and allow different tissues to function differently.

Cells in the primitive heart tube contract spontaneously throughout embryonic development, beginning at embryonic day 7.5 (E7.5) in mice. These contractions largely originate from precardiac mesodermal cells in the inflow tract region [8]. These cells are located in the sinus venosus of mice and exhibit the transcription factor homeobox protein NKX2.5 and hyperpolarization-activated cyclic nucleotide-gated channel 4 (HCN4) (Ref. 9). The SAN gene program is activated in the sinus venosus by a number of transcription factors, such as insulin gene enhancer protein ISL1, short stature homeobox protein 2 (SHOX2), and T-box transcription factors TBX3 and TBX5. In contrast, the chamber myocyte gene program is repressed in the sinus venosus, AV canal, and inner curvature by NKX2.5, TBX3, and TBX18, allowing pacemaking cells to predominate in these regions9. It has been possible to transform chamber cardiomyocytes into pacemaker cells that possess all the essential traits of SAN cells by re-expressing transcription factors like SHOX2 and TBX18 in postnatal mouse ventricular cardiomyocytes [10,11,12]; this finding supports approaches for creating biological pacemakers, which are described below. The heart tube's conduction system's activation pattern resembles the adult heart's by the time looping occurs at E9.59, [13].

Dual oscillators in SAN cells provide steady, rhythmic diastolic depolarization, which starts pacemaker action potentials. These two oscillators are known as the "calcium clock" and the "membrane clock" [14]. Within the plasma membrane, the membrane clock oscillator is dependent upon the pacemaker current (If) [15], also known as the funny current, which is produced by HCN4. The SAN's Cav1.3 calcium channel primarily produces the T-type and L-type calcium channel currents (ICaL), which are activated as a result of the diastolic depolarization caused by this inward current16. Unlike chamber cardiomyocytes, which depend on an inward sodium current produced by the sodium channel Nav1.5 for the rapid upstroke phase of their action potential, the inward calcium current (ICa) promotes the distinctive gradual depolarization of the SAN action potential. The internally driven calcium clock and the membrane clock work in tandem [17,18]. Diastolic depolarization in this second oscillator is facilitated by the electrogenic sodium-calcium exchanger (NCX) and subsequent cyclic release of calcium from the sarcoplasmic reticulum [19,20]. The surface membrane can be sufficiently depolarized to reach the activation threshold of ICa by the combined inward currents from If and NCX. Sodium currents help in the conduction from the SAN into the surrounding atrial tissue, even though they are not the main factor in SAN pacemaker operation.

The pacemaker current (If) can be regulated by the autonomic nervous system through adrenergic and muscarinic stimulation, as well as by pharmacological agents such as ivabradine, which modify the pacemaker frequency by affecting

the rate of diastolic depolarization [21]. Stimulation of autonomic receptors alters the activity of cAMP-dependent protein kinase (PKA) and calcium/calmodulin-dependent protein kinase type II (CAMKII), subsequently influencing both the membrane clock and calcium clock by modulating the phosphorylation of hyperpolarization-activated cyclic nucleotide-gated (HCN) channels, calcium channels, ryanodine receptors, and other proteins involved in sarcoplasmic reticulum calcium refilling and release [22]. Increases in intracellular calcium enhance calcium-dependent phosphorylation, while active phosphodiesterases diminish the effects of this phosphorylation [23,24]. While other components of the conduction system (AV node, His bundle, bundle branches, and Purkinje fibers) can exhibit spontaneous activity, they do so at a rate slower than that of the SAN and typically at frequencies inadequate to maintain effective circulation. As a result, the SAN governs the heart rate by superseding the slower pacemaker activities situated beneath it. Should spontaneously pacing below the SAN occur at a faster rate, such ectopic pacemakers would engage in competition with the SAN, potentially leading to arrhythmias.

Conduction System Disease

Disruptions in the pacemaker and/or conduction system may stem from either an impairment in impulse generation by the sinoatrial node (SAN) or an obstruction of impulse transmission at any juncture within the conduction pathway. Other sources of conduction failure, which will not be elaborated upon in this discussion, include aberrant conduction routes resulting from developmental anomalies. The predominant identifiable factors leading to conduction block are often associated with the physical destruction of components of the conduction system. For instance, valvular infections due to endocarditis can create abscesses that exert pressure on or erode the atrioventricular (AV) node. Similarly, the placement of a prosthetic valve may encroach upon the neighboring conduction structures, resulting in reversible or irreversible heart block25. In rare instances, congenital complete heart block can emerge in response to circulating factors (potentially antibodies that hinder AV conduction) transmitted from the mother to the fetus26. Nevertheless, the most prevalent cause of conduction abnormalities is progressive cardiac conduction disease (also referred to as Lev–Lenègre disease)27, an idiopathic and age-associated disorder characterized by fibrosis within the conduction system. Regardless of the underlying etiology, the management of these irreversible conditions is primarily guided by the presence of associated symptoms (such as fatigue, dizziness, or syncope) and the extent of bradycardia28.

Electronic Pacemakers
Evolution of Device Technology

Electronic pacemakers represent the cornerstone of treatment for bradycardia due to cardiac conduction system pathology [28]. Each year, over 200,000 individuals undergo permanent pacemaker implantation for these conditions in the United States alone [29]. The initial successful uses of electrotherapy in bradycardia patients involved external pulse generators connected to the thoracic wall or directly to the heart via wires [30]. For obvious reasons, this configuration limited mobility and proved uncomfortable. In 1958, the first fully implantable pacemaker

was placed via open thoracotomy at the Karolinska Institute in Sweden. Designed by Rune Elmqvist and Ake Senning, this device featured epicardial electrodes that linked directly to the heart [31]; it sustained the patient's heart rhythm for 3 hours before necessitating replacement with a new device. This individual required a total of 26 pacemaker replacements over their lifetime, ultimately passing away at the age of 86 from an unrelated cause [32]. In the USA, implantable electronic pacemakers developed by Wilson Greatbatch utilizing mercury-based battery technology were introduced for human application during the 1960s33. The first patient fitted with the Greatbatch pacemaker survived for 18 months.

Since the inception of implantable electronic pacemakers six decades ago, we have observed ongoing advancements in device technology, particularly in lead design, generator size, battery longevity, and software algorithms that have led to the development of smaller devices with enhanced functionality. One significant limitation of early electronic devices was their extremely short battery life. In response to this challenge, several companies engineered nuclear-powered pacemakers34,35. The first isotope-based pacemaker (238Pu) was implanted in April 1970 in France [34]. Such isotope-driven devices were capable of delivering stable pacing for over 30 years, offering fixed or asynchronous pacing [34]. Subsequent generations of isotope-based pacemakers featured synchronous pacing abilities, enabling them to sense the patient's intrinsic rhythm and pace only when no underlying rhythm was detected. Despite their remarkable battery longevity, these devices were supplanted by next-generation models employing lithium-based battery technology and enhanced software algorithms [34,36]. Due to the inherent safety concerns associated with isotope-powered devices, lithium-based batteries rapidly became the standard power source for contemporary electronic pacemakers [37].

Modern Device Technologies

The technology behind implantable electronic pacemakers has continued to progress, leading to the availability of a variety of advanced devices capable of delivering reliable pacing across diverse patient populations [28]. Current devices can detect intrinsic rhythms in both the atria and ventricles, allowing them to pace chamber on demand at a programmable baseline heart rate. While modern lithium-based battery technologies can provide stable pacing for approximately 10 years, battery life can be extended further through software algorithms. Algorithms that iteratively assess the minimum output required (pacing threshold) to capture the myocardium (autocapture) can automatically adjust the output, thereby increasing the pacemaker's lifespan to over 10 years [38,39]. Furthermore, contemporary software algorithms can monitor AV nodal conduction and reduce right ventricular (RV) pacing in patients with intermittent AV block [40,41]. The objective is to mitigate the potential adverse effects associated with chronic RV pacing, such as electrical and mechanical desynchrony and RV pacing-induced cardiomyopathy [42,43]. An additional pacing strategy to address RV pacing-induced cardiomyopathy, which also revolutionized heart failure management, is cardiac resynchronization therapy (CRT), or biventricular pacing [44]. Biventricular pacing is accomplished by placing a left ventricular (LV) lead through a coronary venous branch in

conjunction with a conventional RV lead, proving effective in prolonging survival and enhancing symptoms and LV ejection fraction (EF) in heart failure patients with widened QRS complexes on the electrocardiogram (ECG) [44,45,46]. Given that approximately one-third of patients do not experience improvements in functional capacity or EF with CRT, novel multipoint pacing techniques have been developed [47], allowing pacing of the left ventricle at multiple sites [48]. Ongoing randomized clinical trials are comparing the efficacy of bipolar versus quadripolar leads in CRT patients [44].

The ability to capture the His–Purkinje network by placing a pacing electrode in the His bundle area has introduced an additional pacing modality, known as His bundle pacing [49]. This approach facilitates rapid antegrade activation of the ventricles, resulting in a narrow QRS complex on the surface ECG [44,50]. Notably, His–Purkinje recruitment through His bundle pacing occurs not only in patients with AV block at the AV node level but also in those with AV block below the AV node (infranodal block) [50]. While larger studies are needed to better understand the long-term advantages of this technique, His-bundle pacing may present a superior alternative to apical RV pacing [44].

Electronic pacemakers equipped with a subcutaneous generator connected to endovascular pacing leads can provide various pacing modalities, including single-chamber, dual-chamber, biventricular, and His-bundle pacing [44,51]. Additionally, some modern devices incorporate software algorithms that detect and manage atrial tachyarrhythmias by overdrive pacing the atria, thereby interrupting the arrhythmia circuit [52]. While effective, these devices carry significant risks; complications are often associated with lead insertion or malfunction [53], as well as infections affecting the leads and/or generators [44,54,55]. Such infections can pose life-threatening risks and generally necessitate the removal of all hardware, during which temporary pacing strategies are implemented [56]. Furthermore, leads may be thrombogenic, resulting in upper extremity deep venous thrombosis [57]. Leads can also influence the motion of the tricuspid valve leaflets, potentially causing clinically significant tricuspid regurgitation [58]. Given the risks associated with lead-related complications, leadless pacemakers have been developed [59]. In these devices, the pulse generator, battery, and sensing and pacing electrodes are encapsulated within a small capsule designed for delivery into the right ventricle via a steerable sheath introduced through the femoral vein [60,61]. The primary limitations of current leadless pacemakers include the necessity for a large-bore (18–24 French) venous delivery system, uncertainties surrounding infectious and thrombogenic risks, and their capacity to provide only single-chamber RV pacing [44]. This single-chamber pacing restricts the application of leadless pacemakers for common indications such as sick sinus syndrome, where atrial pacing is preferred, or in scenarios where AV synchrony is essential, such as sinus rhythm with chronic AV block [28].

Cardiac Resynchronization Therapy (CRT)

Cardiac Resynchronization Therapy (CRT) Efficacy refers to the effectiveness of CRT in improving heart function and clinical outcomes in patients with heart failure, particularly those with left ventricular (LV) dysfunction and evidence of

electrical dyssynchrony, such as a prolonged QRS duration on an electrocardiogram (ECG). CRT involves the implantation of a specialized pacemaker that coordinates the contraction of the heart's ventricles by pacing both the left and right sides, thereby improving the timing of contractions.

Key Aspects of CRT Efficacy

1. **Clinical Improvement**:
    - **Symptom Relief**: CRT has been shown to significantly reduce symptoms of heart failure, such as fatigue, shortness of breath, and exercise intolerance.
    - **Quality of Life**: Patients often report an improved quality of life following CRT, which is supported by various quality-of-life assessment scales.
2. **Hemodynamic Benefits**:
    - **Improved Cardiac Output**: CRT can enhance overall heart function, leading to increased cardiac output and improved exercise capacity.
    - **Reduced LV Volume**: CRT may help reduce the size of the left ventricle and improve its shape, which can positively influence heart function.
3. **Mortality Reduction**:
    - CRT has been associated with a reduction in mortality rates among patients with heart failure, particularly in those with moderate to severe symptoms and wide QRS complexes.
4. **Hospitalization Rates**:
    - Studies have shown that CRT can decrease the frequency of heart failure-related hospitalizations, thereby reducing healthcare costs and improving patient outcomes.
5. **Response Variability**:
    - Approximately **one-third** of patients may not experience significant improvements in functional capacity or left ventricular ejection fraction (LVEF) after CRT. Therefore, patient selection and ongoing monitoring are crucial for maximizing CRT efficacy.
6. **Electrocardiogram Changes**:
    - CRT typically results in changes to the ECG, such as a narrower QRS complex, which indicates improved electrical synchronization between the ventricles.

Clinical Trials and Guidelines

- Numerous clinical trials, such as the **COMPANION** and **MADIT-CRT**, have demonstrated the efficacy of CRT in improving outcomes for eligible patients.
- Current guidelines from organizations like the **American College of Cardiology** (ACC) and the **Heart Rhythm Society** provide recommendations for the use of CRT in specific patient populations based on LVEF, QRS duration, and symptomatic status. In summary, CRT efficacy encompasses various clinical and hemodynamic improvements in heart failure patients. It plays a vital role in managing heart failure with reduced ejection fraction (HFrEF), particularly for those with evidence of

electrical dyssynchrony, and has demonstrated the potential to enhance survival, quality of life, and overall cardiac function.

Next-Generation Pacemakers: Innovations and Advancements

Current-generation pacemakers, while effective, come with various limitations that impact their long-term efficacy and patient safety. These limitations include lead-related complications, generator malfunctions, infection risks, and insufficient physiological responsiveness. The challenges are amplified in pediatric populations due to factors such as smaller body size, rapid growth, and the presence of congenital heart defects. Next-generation devices aim to address these issues with innovative technologies and designs.

Key Limitations of Current-Generation Pacemakers

1. **Lead Complications**:
    - Current pacemakers rely on leads for electrical connection, which can malfunction, fracture, or lead to thrombosis and infections.
    - Epicardial pacing is recommended for pediatric patients under 15 kg or those with unfavorable anatomy, but these leads are more susceptible to damage and require frequent replacements.
2. **Battery Dependency**:
    - Most contemporary pacemakers require generator replacement approximately every 10 years, which increases the risk of procedural complications due to multiple surgeries.
3. **Autonomic Response**:
    - Current devices lack true autonomic responsiveness, limiting their ability to adapt to physiological changes in heart rate based on body demands.

Innovations in Next-Generation Pacemakers

1. **Alternative Energy Sources**:
    - **Conformal Piezoelectric Energy Harvesting**: This technology converts the mechanical movements of internal organs into electrical energy, potentially eliminating the need for battery replacement.
    - **Solar-Powered Pacemakers**: These devices incorporate a subcutaneous solar module to harness transcutaneous light energy for sustained cardiac pacing. However, further preclinical testing is necessary to evaluate their long-term reliability and efficacy.
2. **Leadless Pacemakers**:
    - Leadless devices eliminate the need for leads, significantly reducing the risk of infection and lead-related complications. While these devices have shown promise, they currently offer only single-chamber right ventricular (RV) pacing.
    - Future advancements may lead to dual leadless pacemakers capable of both sensing and pacing in multiple cardiac chambers, enhancing synchronization and improving patient outcomes.

3. **Enhanced Sensing and Autonomic Responsiveness**:
    - Next-generation pacemakers are expected to incorporate a combination of advanced sensing technologies, such as:
        - **Impedance Measurements**: Used to estimate myocardial contractile performance, acting as a surrogate for sympathetic activation.
        - **Programmable Accelerometers**: To detect patient activity and adjust pacing rates according to exercise levels.
    - With the integration of sensors for cardiac output and blood pressure, alongside complex software algorithms, these devices will strive for improved physiological responsiveness, enabling them to better meet the body's dynamic needs.
4. **Wireless Communication Technologies**:
    - The development of advanced wireless technologies will facilitate the evolution of pacemakers that can communicate effectively with other devices, potentially allowing for remote monitoring and adjustments to pacing parameters.

Next-generation pacemakers hold the promise of overcoming many of the limitations faced by current devices. Innovations in energy harvesting, leadless designs, and enhanced sensing capabilities could transform cardiac pacing by improving patient outcomes and reducing the risks associated with traditional pacemaker technologies. Ongoing research and development will be crucial to ensure these advancements are safe, reliable, and effective in meeting the physiological needs of patients.

Role of Pharmacists in Managing Cardiac Pacemakers and Related Conditions

Pharmacists play a vital role in the multidisciplinary care of patients with cardiac pacemakers, especially in addressing the complexities of therapy management, medication safety, and patient education. Their contributions are crucial throughout the patient care continuum, from pre-implantation assessments to post-implantation follow-up. Here's how pharmacists can support patients with cardiac pacemakers and related conditions:

1. Medication Management

- **Medication Review**: Pharmacists conduct thorough reviews of a patient's medication regimen to identify potential drug interactions, contraindications, or duplications, particularly with anticoagulants, antiarrhythmics, and other cardiovascular medications.
- **Therapeutic Drug Monitoring**: They monitor patients on medications requiring specific levels (e.g., warfarin or digoxin) to ensure optimal therapeutic outcomes and minimize adverse effects, adjusting doses as necessary in collaboration with the healthcare team.

2. Patient Education

- **Device Understanding**: Pharmacists educate patients about the purpose, functioning, and potential complications of cardiac pacemakers, ensuring they understand the importance of adherence to therapy and follow-up appointments.

- **Medication Counseling**: They provide counseling on prescribed medications, discussing indications, dosing, side effects, and the importance of adherence, particularly for those managing underlying conditions such as heart failure or arrhythmias.
- **Lifestyle Modifications**: Pharmacists can advise patients on lifestyle changes (diet, exercise, smoking cessation) that can support overall cardiovascular health and improve outcomes.

3. Monitoring and Follow-Up

- **Adverse Effects Reporting**: Pharmacists can assist in monitoring for adverse drug reactions or complications associated with pacemaker therapy, guiding patients on when to seek medical attention.
- **Regular Follow-Up**: They play a role in regular follow-ups for patients with pacemakers, checking in on medication adherence, side effects, and overall well-being.

4. Interprofessional Collaboration

- **Team Participation**: Pharmacists are integral members of the healthcare team, collaborating with physicians, nurses, and other specialists to develop and implement comprehensive care plans for patients with pacemakers.
- **Consultation**: They provide consultations on medication therapy management, offering insights on pharmacotherapy options to optimize heart rate control and overall cardiac function.

5. Clinical Services

- **Chronic Disease Management**: Pharmacists can lead or participate in chronic disease management programs for patients with heart disease, including monitoring and managing hypertension, diabetes, and hyperlipidemia, which are critical in patients with pacemakers.
- **Immunization Services**: They can administer vaccines (e.g., influenza, pneumococcal) that are essential for preventing respiratory infections, particularly in patients with compromised cardiac health.

6. Research and Development

- **Clinical Trials**: Pharmacists can be involved in clinical trials related to new pacemaker technologies or related pharmacotherapies, contributing to the advancement of knowledge in cardiac care.
- **Education and Training**: They can participate in training healthcare professionals about the pharmacological aspects of managing patients with pacemakers, enhancing the team's understanding of medication management. The role of pharmacists in the care of patients with cardiac pacemakers is multifaceted and essential for optimizing patient outcomes. By ensuring safe medication use, providing patient education, collaborating with the healthcare team, and monitoring for adverse effects, pharmacists significantly contribute to the management of these patients. Their expertise in pharmacotherapy is invaluable in addressing the unique challenges posed by cardiac pacing and associated conditions, ultimately improving the quality of care and patient safety.

Conclusion

The advent of pacemakers and heart failure monitoring devices has revolutionized cardiac care, significantly improving outcomes for patients with bradycardia and conduction disorders. While the fundamental role of electronic pacemakers in maintaining heart rhythm is well-established, the incorporation of biological pacemakers and advanced pacing techniques signifies the ongoing evolution of this field. Pharmacists have emerged as essential members of the healthcare team, providing crucial support in managing the pharmacological aspects of patient care. As medication experts, pharmacists are uniquely positioned to address the complexities associated with managing patients who have implanted cardiac devices. Their involvement in monitoring medication regimens is vital, particularly for medications such as anticoagulants and antiarrhythmics, which can have significant interactions with cardiac devices. They play a proactive role in identifying potential drug interactions, managing adverse effects, and ensuring adherence to prescribed therapies. By conducting comprehensive medication reviews, pharmacists can optimize pharmacotherapy, tailoring treatment plans to individual patient needs. Furthermore, the integration of newer pharmacological agents, such as ivabradine, expands the pharmacist's toolkit for managing heart rate and enhancing patient outcomes. Pharmacists are also instrumental in patient education, empowering individuals to understand their treatment regimens and the importance of regular monitoring and follow-up. Collaboration with other healthcare professionals, including cardiologists and primary care providers, is crucial for holistic patient management. The interdisciplinary approach ensures that all aspects of a patient's care, including device function and medication management, are coordinated effectively. In conclusion, the evolving landscape of cardiac care necessitates an expanded role for pharmacists in managing patients with pacemakers and heart failure monitoring devices. Their expertise in medication management, coupled with a collaborative approach to patient care, is essential in optimizing treatment outcomes and enhancing the quality of life for individuals with cardiac conduction disorders.

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أجهزة تنظيم ضربات القلب وأجهزة مر اقبة فشل القلب - إدارة الأدوية وتحديث القراءات - دور الصيادلة.

الملخص:

الخلفية :تُعتبر أجهزة تنظيم ضربات القلب وأجهزة مراقبة فشل القلب أساسية في إدارة بطء القلب واضطرابات توصيل القلب الأخرى. بينما تعتبر أجهزة تنظيم ضربات القلب الإلكترونية التقليدية فعالة، إلا أنها تواجه العديد من التحديات، بما في ذلك عطل الأسلاك ومخاطر العدوى.

الهدف :يهدف هذا الاستعراض إلى تقييم الدور المتطور للصيادلة في إدارة المرضى الذين لديهم أجهزة تنظيم ضربات القلب المزروعة وأجهزة مراقبة فشل القلب، مع التركيز على إدارة الأدوية والمراقبة.

الطرق :يستعرض المقال الأدبيات الحالية حول وظيفة وتطور تكنولوجيا تنظيم ضربات القلب، وعلم الأمراض لتشوهات التوصيل، والآثار المترتبة على التدخلات الدوائية.

النتائج :يلعب الصيادلة دورًا حاسمًا في ضمان إدارة مثلى لعلاج الأدوية، خاصة فيما يتعلق بمضادات التجلط، ومضادات عدم انتظام ضربات القلب، وأدوية فشل القلب. يقومون بمراقبة تفاعلات الأدوية، وإدارة الآثار الجانبية، وتقييم الالتزام بأنظمة العلاج. تقدم دمج عوامل دوائية جديدة، مثل إيفابرادين، استراتيجيات إضافية للتحكم في معدل ضربات القلب، مما يعزز نتائج المرضى.

الاستنتاج :يتطور دور الصيادلة في سياق إدارة أجهزة تنظيم ضربات القلب، مع التأكيد على أهمية مراجعات شاملة للأدوية، وتثقيف المرضى، والتعاون بين التخصصات لتحسين النتائج الصحية للمرضى الذين لديهم أجهزة قلبية.

الكلمات المفتاحية :أجهزة تنظيم ضربات القلب، فشل القلب، الصيادلة، إدارة الأدوية، اضطرابات توصيل القلب، رعاية المرضى.