

HEART FAILURE AND SUDDEN CARDIAC DEATH

Khakimov Zaynobiddin Kobiljonovich

Andijan State Medical Institute, Uzbekistan

Abstract:Heart failure (HF) is a clinical manifestation of many heart diseases. This is a condition when the compensatory mechanisms of the cardiovascular system are no longer able to maintain normal homeostasis. Currently, there are more than 22 million patients with HF in the world. According to various estimates, every year 200–450 thousand people in the United States experience sudden circulatory arrest, which in 95% of cases leads to sudden cardiac death (SCD).

Keywords:SCD, HF, treatment, method, diagnosis.

INTRODUCTION:In economically developed European countries, about 2,500 people die suddenly every day, and only 2–5% of cases occur in medical institutions. In the world, the annual predicted number of cases of SCD is about 3 million with a survival rate of no more than 1.0%. The probability of successful resuscitation in economically developed countries does not exceed 5%. Most such events occur without witnesses, therefore, without hope of providing timely medical assistance in the first 6–8 minutes, when brain survival is possible. Thus, in 40% of cases, SCD is not witnessed or occurs during sleep, and in 80% of cases – at home [1].

MATERIALS AND METHODS:The famous Fremingen study (1970) showed that the survival rate with a primary diagnosis of HF was 62% in men and 42% in women. The mortality rate was 6–7 times higher compared to previous indicators. Of the total mortality, SCD accounted for approximately 40–50% [2].

The problem of heart failure and sudden cardiac death is very broad. This article will address issues related to the incidence of death in patients with HF [1], the arrhythmogenic substrates and triggers of malignant arrhythmias that cause SCD [2], the stratification of patients at high risk of SCD, and the results of You have seen clinical studies conducted on the prevention of sudden cardiac death.

RESULTS AND DISCUSSION:The main nosological cause of the development of SCD is ischemic heart disease – 80–85% of cases, of which more than 65% of cases are associated with acute coronary circulatory disorders. Dilated cardiomyopathy (DCM) causes 5 to 10% of sudden cardiac deaths and other heart diseases cause 5 to 10%. The immediate mechanism of circulatory arrest is cardiac arrhythmia, of which 90% are ventricular tachyarrhythmias. Electromechanical dissociation and bradyarrhythmias cause circulatory arrest in 10% of cases of SCD. Another cause of sudden cardiac death in severe heart failure is thromboembolism, which develops mainly against the background of atrial fibrillation.

The Intervention Trial in Congestive Heart Failure (MERIT-HF) [33] included 3991 patients with chronic NYHA class II–IV HF and LVEF less than 40%. 1990 patients received metoprolol, and 2001 patients received placebo. According to the study, in the group receiving metoprolol, the rates of overall mortality ($p<0.001$), mortality from cardiovascular diseases ($p<0.001$), the incidence of sudden death ($p<0.001$) and mortality associated with the progression of congestive heart failure ($p<0.002$) were significantly lower compared to the placebo group. The incidence of sudden death in the group of patients receiving metoprolol was 3.9%, in the placebo group - 6.6%, overall mortality was 7.2 and 10.9%, respectively. The occurrence of SCD decreased with increasing degree of heart failure: from 60% in patients with class II or III to 30% in patients with class IV according to NYHA. The number of patients who died due to low contractile function of the left ventricle increased from 12% in patients with NYHA class II to 28% in patients with class III and 56% in patients with class IV [2].

Connexons are specific channels that exist in the area of intercellular contacts. They penetrate the outer membranes of contacting cells, through which molecules up to 1 kDa in size can penetrate. These channels transmit intercellular signals and ensure the coordinated functioning of tissue cells. Disruption of intercellular communications causes cells to become “deaf” to regulatory signals. The connexin subunit is a large protein (25–28 kDa) with four transmembrane segments. Six connexin subunits are grouped around a hydrophilic pore that spans the membrane. Two connexons (half-channels) of neighboring cells, located opposite each other, connect and thus form a continuous channel between two fibers. Inorganic ions and small molecules are able to pass through the channel, which ensures metabolic cooperation of neighboring cells. One of the connexons is connexin-43, which plays a decisive role in the propagation of the impulse and in its synchronization between myocytes.

It is believed that ventricular arrhythmias in acute myocardial ischemia arise by the reentry mechanism, which develops due to the presence of slow conduction areas and the difference in the duration of the monophasic action potential inside and outside the ischemic (border) zone [2]. The most studied experimentally is the modeling of the reentry mechanism during MI, where the VT substrate of this mechanism is the myocardial zone bordering necrotic tissue, formed from intertwined islands of viable myocardial fibers and connective tissue. Intraoperative transmural mapping showed that the possibility of triggering inducible ventricular tachycardia depends on the formation of this reentry loop, the beginning of which is formed by a zone of critical deceleration of the conduction of the excitation wave in the myocardium. Experimental studies have shown the occurrence of a blockade or a change in the speed of excitation in a number of areas of the myocardium before the development of ventricular fibrillation. Under these conditions, a premature impulse leads to a sudden disruption of the propagation of the excitation wave. Ventricular fibrillation is maintained by the microreentry mechanism.

CONCLUSION: There are complex interactions between cardiac dysfunction and the generation of malignant arrhythmias. Sudden death is often reported in patients with HF. It is very important to stratify the risk of SCD during the clinical assessment of a patient with HF. Once the underlying cardiac disease has been identified, appropriate shock therapy should be administered to modify the disease course, improve symptoms and prognosis. The use of antiarrhythmic drug therapy for the prevention of SCD in all patients with HF is not recommended. And when it needs to be used to relieve symptomatic arrhythmia in patients with heart failure, the drug of choice is amiodarone; in case of contraindications, dofetilide or dronedarone. Patients with ischemic or non-coronary heart disease with reduced LVEF (less than 35%) and with symptoms of HF (NYHA class II–III) are indicated for implantation of CRT and ICD. The use of CRT-P or CRT-D can improve the clinical condition and quality of life of patients at various stages of HF. The number of patients with HF will increase as life expectancy increases as a result of improved medical care. Many methods for SCD risk stratification and pharmacological and non-pharmacological therapies for its prevention will continue to be developed and improved.

REFERENCES:

1. Antipenok, A.V. Risk factors for sudden cardiac death and depressive disorders in patients with chronic heart failure: dis. ...cand. honey. Sciences / A. V. Antipenok. – Perm, 2015. – 99 p.
2. Arbolishvili, G. N. Sudden (arrhythmic) death during Holter ECG monitoring / G. N. Arbolishvili, S. N. Nasonova, A. G. Ovchinnikov // Heart failure. – 2012. – T. 3, No. 4. – P. 200.
3. Maksimovna, M. M., Daliyevich, A. Y., Zuxritdinovna, M. M., Mamadjanovna, B. A., & Nozimjon O’g’li, S. S. (2021). Allergy to the Production Dust at Workers of Integrated Cotton Mill. *JournalNX*, 7(07), 52-54.

4. Nozimjon o'g'li, S. S. (2022). INFORMATION ABOUT THE STRUCTURE OF THE MEMBRANE OF EPITHELIAL TISSUE AND GLANDS. *British Journal of Global Ecology and Sustainable Development*, 10, 65-69.'
5. Maxmudovich, A. X., Raximberdiyevich, R. R., & Nozimjon o'g'li, S. S. (2021). Oshqozon Ichak Traktidagi Immunitet Tizimi. *TA'LIM VA RIVOJLANISH TAHLILI ONLAYN ILMIY JURNALI*, 1(5), 83-92.
6. Shoxabbos, S., & Mahramovich, K. S. M. K. S. (2023). CAUSES OF THE ORIGIN OF CARDIOVASCULAR DISEASES AND THEIR PROTECTION. *IQRO JURNALI*, 1-6.
7. CHULIEVA, V. E. (2021). THE PRINCIPLES OF COMMONALITY AND SPECIFICITY IN THE PHILOSOPHICAL TEACHINGS OF BAHA UD-DIN WALAD AND JALAL AD-DIN RUMI. *THEORETICAL & APPLIED SCIENCE Учредители: Теоретическая и прикладная наука*, (9), 566-573.
8. Mavlonovna, R. D. Factors That Increase the Activity of Women and Girls in Socio-political Processes at a New Stage of Development of Uzbekistan. *JournalNX*, 7(07), 61-66.
9. Mavlonovna, R. D. Participation of Uzbek Women in Socio-economical and Spiritual Life of the Country (on the Examples of Bukhara and Navoi Regions). *International Journal on Integrated Education*, 4(6), 16-21.
10. Mavlonovna, R. D., & Akbarovna, M. V. (2021, July). PROVISION OF FAMILY STABILITY AS A PRIORITY OF STATE POLICY. In *Archive of Conferences* (pp. 34-39).
11. Khairullayevich, S. H. Development of gymnastics in Uzbekistan and attention to gymnastics. *International scientific-educational electronic magazine" OBRAZOVANIE I NAUKA*, 21.
12. Sayfiyev, H., & Saidova, M. (2023). EFFECTS OF GYMNASTICS ON FUNDAMENTAL MOTOR SKILLS (FMS), POSTURAL (BALANCE) CONTROL, AND SELF-PERCEPTION DURING GYMNASTICS TRAINING. *Modern Science and Research*, 2(9), 204-210.
13. Saidova, M., & Sayfiyev, H. (2023). CONTENT-IMPORTANCE AND PRINCIPLES OF PHYSICAL EDUCATION CLASSES. *Modern Science and Research*, 2(9), 192-199.
14. Ayubovna, S. M., & Komiljonova, K. I. (2022). Features of Application of Sports Games in Preschool Children. *International Journal of Culture and Modernity*, 16, 17-23.
15. Saidova, M. (2023). THE CONCEPT OF PHYSICAL QUALITIES. *Modern Science and Research*, 2(10), 251-254.