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First Case of Brain Protection in Out of Hospital Cardiac Arrest

My wife Ilfat, a 73 year-old psychiatrist, experienced OHCA on June 2011 in the living room where she was watching the BBC world channel on TV. She suddenly stopped talking. I waited a few moments before turning towards her to check whether she might have fallen asleep. To my horror I found that she was cyanotic, her head drooped on her chest. It was obvious that she had signs of possible sudden cardiac arrest. I immediately laid her down on the floor. There was no femoral pulse. I initiated standard CPR including chest compression (90 compressions/minute) for about 30 seconds followed by mouth to mouth breathing. I then resumed cardiac massage since she did not regain consciousness and there was no femoral pulse. I had a defibrillator in the basement of my house because of my interest in the Fulguration procedure. Although the defibrillator was more than 30 years old I knew that this machine would work by electrical current from the outlet. The defibrillator was charged to allow for a first shock and a second shock to be delivered through metallic disk-shaped electrodes that were on this old machine. Her body jumped but the shocks were ineffective. Since I had no gel to apply between the electrodes and the skin to decrease the impedance, I applied saliva to the electrodes and then gave a third shock. This last shock was successful, and the femoral pulse returned strong and regular. Recurrent chest compression produced the characteristic sound made by a broken chondro-costal joint. The femoral pulse remained stable, regular and strong. I estimated that about 5 to 6 minutes elapsed between her loss of consciousness and the return to a stable circulation. Since there was no sign of return to consciousness and she had bilateral pupillary dilation, I then addressed the issue of possible brain protection. Fortunately, I had a smaller portable bottle now called the "Fontaine bottle" (JACC 2016) that was designed with a gas regulator to be used with a nasal cannula inserted into the nose to provide protection of the brain by nasal cooling. Because of the invasive insertion in fossa nasalis, Ilfat exhibited a pain reaction which I interpreted as a positive sign for her chances of survival. When the injector was in position I opened the valve of the cylinder and induced nasal cooling by expansion of the CO₂. Her hospital course was notable for repeated episodes of ventricular fibrillation with the same ECG pattern of Torsade de Pointes-like tachycardia degenerating in Ventricular Fibrillation in a few seconds. The absence of Troponin release demonstrated that she had no acute myocardial infarction which was my main concern. When sedation was stopped, she was fully awake and the tracheal tube was removed. Ilfat first question was whether she was treated with nasal cooling! She refused amiodarone but was treated with bisoprolol which proved effective. After recovery from the implantation of a dual chamber defibrillator she had only thoracic pain which was a direct consequence of chest compression. This pain disappeared in two months. Psychological test showed that she had absolutely no neurologic deficit. In particular, she could remember 9 telephone numbers. During the follow-up, she experienced three more episodes of sudden death with drop (one with injury of the face) immediately converted by the defibrillator leading to an increase of Bisoprolol. However, she experienced a total obstruction of the trunk cerebral artery in November 2015 also treated in La Salpêtrière by thrombolysis and clot extraction 1:20 after the loss of consciousness. She again recovered with absolutely no neurological deficit except a slower rate of speaking. She is now treated by Eliquis and enjoys normal life and continues to see her patients. Thanks to advanced techniques of resuscitation techniques and clinical electrophysiology (my area of expertise) as well as interventional neurology

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