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Wellbeing and agency in parents of children with congenital heart disease

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Introduction: Parents of children suffering from congenital heart diseases develop symptoms of depression, distress, anxiety and hopelessness more frequently than parents of healthy children (1, 2). Associated with the described symptoms, parents may experience a lack of control and disempowerment, which decreases the parent’s agency, a construct from development studies, and which may have negative consequences on adherence to treatment (3). The primary aim of this study was to assess the effect of medical treatment on wellbeing and agency in parents of children suffering from congenital heart diseases in Chile and to compare it with Reference values.

Methods: 40 parents of children suffering from congenital heart diseases (before surgery and before hospital discharge) and 115 parents of healthy children were surveyed. To evaluate mental wellbeing the General Health Questionnaire was applied; to assess agency the Basic Psychological Needs Scale and the Self-Determination Scale were used; in addition the Beck Hopelessness Scale and a socioeconomic survey were included in this study.

Results: Parents of children suffering from congenital heart diseases scored significantly worse than parents of healthy children on the General Health Questionnaire (p = 0.001). This difference was not found using the others scales. Children’s surgery decreased parents’ hopelessness (p = 0.04), and no significant differences were found in the remaining scales.

Discussion: Children’s surgery has a positive effect on parent’s hopelessness, but it does not have any impact on their wellbeing nor agency. Parents of children suffering from congenital heart disease have a decreased wellbeing compared to parents of healthy children, but have a similar level of agency. Socioeconomic level and gender may influence this association.


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Impact of combined anaesthesia on cognitive functions of patients after cardiac surgery

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Background and Goal: The incidence of postoperative cognitive dysfunction (POCD) occurs in 30-65% of patients after cardiac surgery. The aim of our study was to investigate the impact of general anaesthesia with thoracic epidural anaesthesia on cognitive functions after cardiac anaesthesia with CPB.

Methods: 80 patients were enrolled into two groups: general anaesthesia (GA, n = 42) and general anaesthesia with thoracic epidural anaesthesia (TEA, n = 38). Neurocognitive tests were accomplished 1 day before surgery and 7 days postoperatively: MMSE, Six – item cognitive impairment test, WAIS Digit symbol substitution test.

Results: All preoperative (baseline) test results did not differ significantly among TEA and GA groups. 7th-day WAIS test results did not differ much comparing TEA (20.03; SD 5.97) and GA (17.14; SD 6.48) groups. Comparing preoperative and postoperative WAIS test results there was a relevant decline in GA (preoperatively: 19 (SD 7.11); postoperatively 17.14 (SD 5.97) group. WAIS score change was significantly higher in GA groups, compared to TEA. There was no significant difference in 7th-day MMSE test results comparing TEA (median 28, min 23,
max 30) and GA (median 28, min 18, max 30) groups. Patients in TEA group demonstrated better Six-item test results than in GA group at the 7th day after surgery ($p = 0.16$). Less points demonstrate better cognitive function, more points - cognitive dysfunction: GA median 2 (min 0, max 16) vs TEA median 2 (min 0, max 16) before surgery and GA median 4 (min 0, max 18) vs TEA median 2 (min 0, max 18) after surgery.

Conclusions: Combined anaesthesia is not associated with POCD, compared to general anaesthesia. Memory and processing speed capabilities decrease after general anaesthesia.

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Pericardial disease after cardiac surgery: postpericardiotomy syndrome, purulent and constrictive pericarditis

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Postpericardiotomy syndrome (PPS) after cardiac surgery is a common inflammatory (20-40%) process due to opening of the pericardium, pleural or both (1), can occur a few days to several weeks after surgery. The diagnosis is clinical. Anti-inflammatory non-steroids and colchicine are the first line therapy and corticoids in severe cases.

Purulent pericarditis (PP) is a localised infection. It's a rare and serious complication. Staphylococcus aureus is the most frequent agent and is usually contiguous contamination, although it can be present by blood dissemination from distant focus. 30-40% have negative microbiological studies. The overall prognosis of purulent pericarditis is dismal with mortality rates up to 40% (2).

Case report: We report on a 68 year old male who presented with a sub acute stroke. A cardiac myxoma with extension over both atria was diagnosed. Tumour resection was uneventful and the histopathology confirmed a myxoma. In the postoperative period a VVIR pacemaker was implanted due to slow atrial fibrillation. The patient was discharged on postoperative day 12. After one week he was readmitted with clinical criteria suggestive of PPS with a large pericardial and pleural effusion. A sterile, serohematic liquid was aspirated during pericardiocentesis. Ibuprofen and colchicine was started. Peripheral phlebitis by methicillin-sensitive Staphylococcus aureus occurred and we added cloxacillin (Flucloxacillin) therapy. He had a persistent pericardial effusion and clinical signs for pericarditis, so we added prednisolone, in spite this an emergency sternotomy was performed due to pericardial tamponade. At this time the effusion was clearly purulent. Pleuromediastinal drainage tubes were left and used for topic gentamicin washings accompanied by endovenous daptomycin/cloxacillin although so far all cultures stayed negative. The clinical progression led to severe constrictive pericarditis with refractory cardiogenic shock despite of antiphrenic pericardiectomy performed. The patient died on day 45.

Discussion: The clinical challenge of this case lies in the distinction between two differently defined pericardial pathologies PPS and PP.

While PPS is diagnosed (2) based on clinical criteria (fever without infection, pleuropericardial rubs, chest pain, pericardial effusion and pleural effusion with CPR elevated), for the diagnosis of PP microscopic purulence is needed with more than 20.000 leucocytes with or without growth.

We believe that the persistence of severe PPS despite treatment, favoured bacterial haematogenous dissemination after phlebitis (MSSA positive). Our treatment strategy was changed putting an infectious process into the focus although the cultured pericardial effusion stayed negative. Nevertheless we could not safely rule out pericardial infection by haematogenous spread.

Conclusion: PP is severe condition with high mortality rate (3). Patients under suspicion for a PPS must be intensively screened for possible infectious signs as the clinical manifestation of both entities are similar but treatment strategies are clearly different. Delayed drainage and antibiotic treatment of manifested purulent infectious pericarditis has a very poor outcome.