Compartment Syndromes in Children and Adolescents

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Compartment syndromes are characterized by a discrepancy between the size of a limitedly compressible mass and the amount of space into which it is to be integrated. When a level of critical compliance is not reached, compensation mechanisms fail, leading to a decline in local or systemic perfusion. This resulting deficiency in perfusion is accompanied by hypoxemia and usually causes a switch to anaerobic energy production. It is at this point (at the latest) that an additional inflammatory stimulus is potentially induced, possibly contributing to a further triggering of the respective pressure gradients via capillary leak syndrome and extravasation. The smaller or younger the patient, the greater the risk of a size and space discrepancy accompanied by—when compared to adults—significantly lower blood pressure and tissue perfusion pressure.

In clinical practice, four types of compartment syndrome play a relevant role in children and adolescents:

1. Fascial/muscle compartment syndrome
2. Cerebral compartment syndrome
3. Thoracic compartment syndrome
4. Abdominal compartment syndrome

Evidence-based data on the first three types is limited. They do not differ from adults with regard to pathogenesis, diagnosis, and therapy [1–4]. There is no reliable epidemiological information on how frequently they occur in children and adolescents.

The lack of data becomes clear simply in that the respective suggested pressure limits vary greatly; moreover, they were set at 20–25 mmHg regardless of patient

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age (i.e., for adults as well as children) until only a few years ago. From the pediatric perspective, such pressure levels are unreasonable when tissue perfusion pressure (TPP = MAP − Compartment pressure) is considered in relation to blood pressure adapted for age. In the case of a regular MAP level of 40 mmHg for an infant, a compartmental pressure of 20 mmHg would be an effective perfusion pressure of 20 mmHg, i.e., half the norm for blood and perfusion pressure in this age group. In recent years, the respective norms for upper limits in pediatric patients have been revised downwards incrementally. Currently, a tissue pressure below 13–16 mmHg is acceptable for cerebral and muscular compartments; up to 10 mmHg is a standard pressure value for intra-abdominal compartments. Knowledge about these new limits, which are adapted to pathophysiological conditions, can be considered neither widespread nor extensive.

Thus far, there has not been any useful data for thoracic compartment syndrome in children and adolescents. Elevated intrathoracic pressure in connection with cardiac surgery is clinically relevant. The decision to leave the thorax open perioperatively is made regardless of the definite pressure values and based on the surgeon's subjective impression as well as cardiorespiratory stability when the patient is taken off the heart-lung machine.

This chapter does not provide a detailed description of the first three types of compartment syndrome named and refers readers to the respective adult-focused chapter in this book.

Only in connection with abdominal compartment syndrome in children and adolescents is evidence continually growing. This is a result of increased attention and scientific research. In spite of this growth, a great lack of knowledge and considerable ambiguities remain.

Due to the significantly better evidence, the author limits himself to a more detailed description of abdominal compartment syndrome.

15.1 Abdominal Compartment Syndrome in Children and Adolescents

15.1.1 Background

Although intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) should be diagnosed especially often in neonatal (NICU) and pediatric intensive care units, both pathologies are still considered too seldom and barely actually diagnosed. This is astounding insofar as the so-called prototypes of high-risk illnesses, and procedures, potentially leading to IAH and/or ACS are to be found original in pediatrics [5]: In this case, there is the existence, and the closure, of a congenital abdominal wall defect (gastroschisis, omphalocele, congenital diaphragmatic hernia) and the transplantation of parenchymatous organs that can differ in size, making their volume a critical issue. Besides these prototypes, there are numerous other combinations of risks (in addition to those known in adult medicine) that can lead to an increase in intra-abdominal pressure (IAP). Through

inflammation and capillary leak syndrome, a critical illness per se preordains the development of increased abdominal pressure. This is reflected in the fact that the likelihood of developing ACS along its associated likelihood of morbidity and mortality increases by 22 times when the PRISM-III-Score (PRISM: Pediatric Risk of Mortality [6, 7]) is above 17 [8].

By definition one speaks of ACS when organ dysfunction occurs or is aggravated in addition to intra-abdominal hypertension (IAH; present when intra-abdominal pressure [IAP] is ≥10 mmHg) [9, 10]. In a healthy child, IAP is between 0 and 5 mmHg; in a mechanically ventilated child (without IAH), one usually finds an IAP of around 7 mmHg [11]. In cases of delayed diagnosis or inadequate therapy, an ACS regularly leads to multi-organ failure and death as a result of mutually triggering organ dysfunction and increasing inflammatory cascades.

15.1.2 Epidemiology

Despite an ever-increasing body of evidence from more and more published studies and profound reviews, only few reliable statements on the epidemiology of ACS in children can be made. Depending on the spectrum of the clinic and severity of the underlying diseases managed and treated in the respective neonatal (NICU) or pediatric intensive care unit (PICU), the ***prevalence*** of ACS ranges between 0.6 and 4.7% in PICUs [8, 12–15] and 7 and 18% in NICUs [16, 17]. When grouped according to risk, the prevalence figures were from 27% (gastroschisis [18]), 37% (burn [19], pancreatitis [20]), and up to 74% (after liver transplantation [21]). All of these figures could still be rather underestimated since they are partially based on the data available at the time of their publication, i.e., when IAP limits were still significantly higher than the standard pediatric maximum of 10 mmHg issued by the WSACS in 2013. For instance, some previously applied standard maximums were at 25–30 mmHg [9].

That premature and newborn babies tend to develop an ACS more often can on the one hand be explained by the miniaturized anatomical conditions and pathophysiological consequences as well as limited compliance regarding intracorporal increases in volume. On the other hand, the "prototype" [5] diseases named above are the ones primarily affecting premature and newborn babies. There are also typical abdominal complications related to premature and critical newborn births, for example necrotizing enterocolitis (NEC), ileus, volvulus, and intussusception. Due to their primary and secondary damage mechanisms, these are extremely often accompanied by IAH and ACS, which contributes significantly to the morbidity and mortality of abdominal complications in this age group [17].

The only figures estimating ***incidences*** of ACS in children come from a yet-to-be-published 2016–2018 surveillance study of all 530 children's clinics and departments in Germany [22, 23]. According to this study, ACS occurs in at least approximately 0.2% of children in intensive care. This figure may underestimate the

actual circumstances, as there are massive signs of extensive underreporting due to failure to diagnose and failure to report (caused by the increased workload in intensive care) [22, 24]. Difficulties performing diagnoses did not occur due to the still somewhat little known WSACS criteria and definitions (ACS = IAH plus new or aggravated organ dysfunction). Only every fifth NICU, and PICU, even reported measuring IAP (at least in individual cases) [25]. Thus, it can be assumed that at least 80% of NICUs and PICUs are not (cannot be) considering the diagnostic criteria in a way that is true to definition. In approximately 18–20% of cases, even organ dysfunctions were not recognized correctly and in time—regardless of the organ system that was concretely affected [22].

Although neonatal patients are affected more often by IAH and ACS than older children, almost all case reports were made by PICUs (and barely by NICUs) in the framework of the surveillance study mentioned above as well as in that of two prevalence studies from 2010 and 2016, respectively [25]. It is even more astounding that—in spite of this—a peak in cases could be found at a median age of 7 months [22]. If neonatal intensive care stations were more thorough in their diagnostics and reporting, this peak would probably shift further towards an even younger class of infants. In contrast to adults, girls and boys appear to be almost equally affected by the development of ACS (no predominance in boys) [22].

That cases of ACS are almost only observed and reported by large departments and university clinics can be due to the on average greater complexity of diseases often treated in large hospitals. However, the data gathered speaks quite clearly for the idea that knowledge and trust in the ability to apply definitions, recommendations, and therapy options associated with IAP can be described as proportional to the size of the clinics, and a great need for training in small clinics and nonuniversity departments can be recognized. Although familiarity with and knowledge of IAP, IAH, and ACS has spread in recent years, it is still far from being sufficient across the board [25–27].

Lethality varies as well depending on the patient clientele and experience of the intensive care unit. It lies between 21 and 80% [8, 13, 17, 21–23, 28, 29], with specific combinations of risks being associated with a significantly higher lethality (above all pancreatitis, burns, NEC) [19, 20, 29, 30].

Risk of death is nine times higher when IAH and/or ACS occur [8]. According to Ejike et al., a 30% and 50% higher mortality rate can be predicted simply when the dynamic of an increase in IAP and "reaching" of the peak IAP value are observed.

The prognosis for the patient appears to essentially depend on doctors' openness to courageous and if necessary invasive but above all timely therapeutic intervention: when a conservative (noninvasive) therapy approach was applied in German-speaking (D-A-CH) NICUs and PICUs, the average likelihood of ***survival*** was 40–60%; meanwhile, the likelihood of survival following decompressive laparotomy proved to be at least 83% [25]. In cases of a significant IAP increase and dynamic as well as ACS that is either impending or already occurring, a rash reduction in pressure and with this usually an invasive procedure can be decisive for

survival. In spite of this, there is often a fatal lapse in action before adequate therapy is introduced in daily clinical practice.

In this case there were also relevant differences depending on the academic background of care providers: whereas university pediatric clinics stated that they perform a yearly average of 2.4 laparotomies for decompression in cases of ACS, nonuniversity departments reported only 0.3 per year. Thus, outcome data differ depending on the size of the intensive care unit, and its academic nature/background [25].

15.1.3 Classification and Pathophysiology

Depending on the origin of the disease leading to an increase in IAP, there are primary, secondary, and recurring (previously tertiary) geneses [9].

Neonatal patients and infants tend to develop a ***primary ACS*** (from a disease of/originating from an organ/tissue in the abdominal cavity) that is often associated with necrotized enterocolitis, intestinal perforation, or (meconium) ileus as well as volvulus [25]. In contrast, older pediatric and adolescent patients tend to develop a ***secondary ACS*** (due to an extra-abdominal pathology). This is related to their larger personal sphere of activity and increasing independence, which exposes them to greater traumatic, thermal, as well as inflammatory influences.

Secondary forms are often unexpected in this context and appear in connection with the surface activation of immunocompetent cells and to a certain extent in connection with every form of extracorporeal circulation (following a heart-lung machine operation, in the context of extracorporeal membrane oxygenation (ECMO), dialysis, etc.). Recently several research papers have described a compression of venal ECMO cannulas that is associated with IAH and results in an ECMO dysfunction or even ECMO failure, especially in pediatric patients [31–34]. As early as 2001, Beck et al. emphasized that—in contrast to those diagnosed in adult patients—secondary forms of IAH and ACS are more prevalent in pediatric patients [35].

Aside from this etiologically/pathogenetic classification, acuity is used as a basis for differentiating among acute, subacute, and chronic processes.

In general, there are four levels of IAH that differ from those in adults in regard to the respective pressure levels:

- Grade I: IAP 10 up to 12 mmHg
- Grade II: IAP >12 up to 15 mmHg
- Grade III: IAP >15 up to 18 mmHg
- Grade IV: IAP >18 mmHg [25]

Contrary to still commonly held opinions among active pediatricians, an ACS is not the same as an elevated or highly elevated IAH (see WSACS definitions). Interestingly, the mortality rate is almost identical in all four levels and—despite widespread beliefs—does not increase with the level [22, 23]. One explanation for

this is that neonatal and infant patients often become severely ill and die at an IAH level of no more than one or two.

As known to animal experts and proven by yet-to-be-published in vivo study data taken from children and adolescents, there is an increased distribution of abdominal and mesenterial tissue perfusion that results from IAP-related mobilization of abdominal pooling reserves (so-called autotransfusion) in IAH grades I and II [21, 36]. In spite of a cardiac output that tends to increase under optimal intensive care treatment management with a sufficient increase in volume and individually adapted catecholamine therapy, the microcirculation in the liver, intestines, and kidneys can decrease to the benefit of the spleen and pancreas (redistribution of organ perfusion with "net winners" (spleen and pancreas) as well as "net losers" (liver, intestines, kidneys)) in this phase [21].

Starting at grade III (IAP >15 mmHg), the compromising pressure components are predominant, above all in diastolic, venal, and lymphatic flow. This is also in regard to spleen and pancreas perfusion. In spite of cardiac output being maintained where appropriate, microcirculation in all abdominal organs and tissue falls rapidly and massively (also because the abdominal pooling reserves are usually used up due to IAH). It is here at the latest that these pathophysiological changes are clinically observable via a decrease in spontaneous diuresis [37]. Due to the increasing liberal use of loop diuretics in pediatric intensive care medicine, oliguria and anuria are barely still detectable early cardinal symptoms of an ACS. This is fatal insofar as—contrary to the WSACS criteria and definitions—the traditional school of thought maintains that an ACS is a clinical diagnosis that can only be determined when there is a concurrence of the cardinal symptoms "abdominal distension," "oliguria/anuria," and "cardiorespiratory failure."

The changes in and redistribution of perfusion mentioned above are barely detectable when using traditional intensive care monitoring. This is where somatic (= abdominal) near-infrared spectroscopy (NIRS) could gain increased significance (Fig. 15.1). In contrast to conventional monitoring, it appears to be able to unmask these subclinical changes [38–41]. According to yet-to-be-published research results from a collection of 350 critically ill children, somatic tissue saturation (NIRS) decreases by about 10% points in cases of IAH (IAP ≥10 mmHg) [21]. If there is a new or aggravated organ dysfunction in the sense of a complete ACS, middle tissue saturation falls again by further 10% points in comparison to the non-IAH control group (composed of critically ill children in intensive care). The alarming extent of desaturation within parenchymatic tissue detected in this context points to the extent of IAH-associated cell and organ damage. This makes it no surprise that multi-organ failure, sepsis, and death can occur when therapy is delayed.

Similarly, there are promising study results on the use of micro-dialysis catheters, e.g., in the musculus rectus abdominis. With their help, an IAH-related hypoperfusion can be monitored in real time by measuring the increasing lactate concentrate associated with the resulting transition into an anaerobic metabolic state. Due to the invasiveness of the procedure, micro-dialysis has yet to enter clinical practice in adult medicine [42–45].

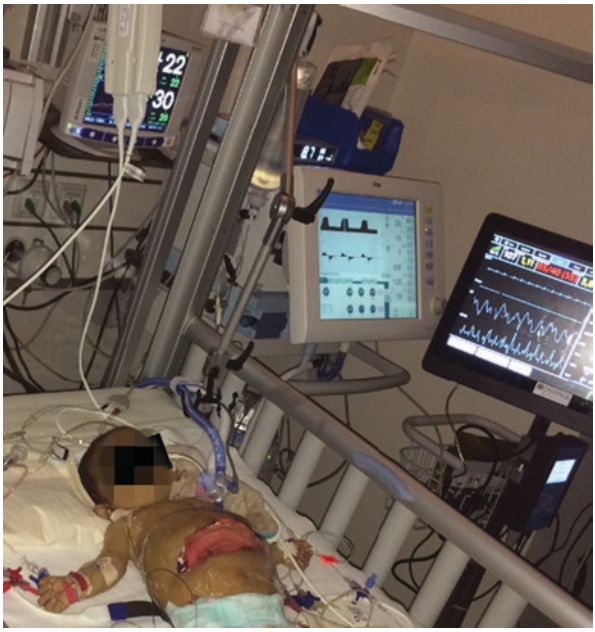

Fig. 15.1 Image of a 10-month-old infant with advanced hemodynamic monitoring following abdominal compartment syndrome with normalization of intra-abdominal pressure after a decompressive laparotomy and transient laparostomy. On the ventilator there is a gastric pressure monitor that indirectly measures an intra-abdominal pressure of 8.7 mmHg. On the right side of the screen, there is an impedance cardiography monitor for the noninvasive quantification of cardiac output, peripheral resistance, stroke volume, and intrathoracic fluid index. Above the head is a near-infrared spectroscopy (NIRS) monitor, which measures somatic tissue saturation right and left paravertebrally over the spleen-kidney or liver-kidney lodge. NIRS allows an indirect statement to be made on the histological restriction of perfusion as a function of intra-abdominal pressure via the course observation of tissue oxygenation

15.1.4 Measurement Methodology and Behavior

The measurement methodology in children does not differ from that in adults and is primarily based on the measurement of bladder pressure first described by Kron et al. [46, 47] and since then repeatedly modified. This is considered the gold standard for indirectly measuring IAP in children and adolescents. After one has carefully ensured that the bladder is completely empty, 1 mL/kg body weight of saline (warmed to body temperature) is inserted into the bladder under sterile conditions. It should be neither below 3 mL nor above 25 mL [11].

Semi-continual bladder pressure measurement via an AbViser®-Valve-System [11, 48, 49] as well as continual measurement of gastric pressure (Spiegelberg®-System, Fig. 15.1) [50–52] are establishing themselves as equally if not more valuable alternatives to measuring bladder pressure manually and have been validated for use in the field of pediatrics [21]. In addition to continual monitoring, the latter system stands out for its especially user-friendly, user-independent, and hygienic advantages.

In individual cases ventilator peak pressure is used to estimate IAP transmitted thoracically via the diaphragm. It is known from animal studies that around 30% of trans-diaphragmal IAP can be further transferred [53, 54]. The method appears to be less clinically feasible and is used for few indications (e.g., when "minimal handling" is necessary and/or in cases of injuries, and diseases, to the gastrointestinal and urogenital tract).

Measuring femoral vein pressure (FVP), and inferior vena cava pressure (IVCP), has proven to be not useful in children. For years this method of measurement was considered a reliable monitoring procedure. However, more and more publications in the field of adult medicine began to either dispute FVP's, and IVCP's, every ability to be used for the indirect quantification of pressure or only spoke for its at least justifiable tendency to estimate real IAP values once IAP has surpassed 18 mmHg [55, 56]. The data collected (but not yet published) recently in the framework of our work group was able to show that there is no justifiable correlation and that FVP, and IVCP, measurement must be rejected as a way of measuring IAP [21].

Direct methods of measurement only have an experimental character and—due to their invasive nature—no importance in the daily routine of pediatric clinics. In the mid to long term, a direct and continuous measurement system would, however, be desirable.

According to the surveillance study mentioned above, routine monitoring of IAP is usually part of the daily routine in pediatric clinics, above all in regard to operative closure of congenital abdominal wall and diaphragmatic hernias; liver failure and/or ascites; following parenchymatous organ transplantations; and following volume/transfusions as well as laparotomies in connection with polytraumatic events and/or larger pediatric abdominal surgical procedures [23].

Standardizing IAP monitoring in cases of specific combinations of risks and/or diseases is without a doubt correct and important [9]; however, this has been the absolute exception and only occurs in few clinics that generally have academic interests.

15.1.5 ACS Defining Organ Dysfunctions

Until the publication of the WSACS definitions in 2013, "new or aggravated" organ dysfunction was not necessarily a criterion considered for diagnosing ACS [9]. Published by Goldstein et al. in 2005, the criteria (depending on the standard values that sometimes vary remarkably among the different age groups within pediatrics) of the International Pediatric Sepsis Consensus Conference (IPSCC) consider static as well as dynamic criteria for assessing the function of every organ system and have proved to be helpful and sensible in standardizing the criteria used to define organ dysfunction [57] (Table 15.1):

Using these IPSCC criteria, a scientifically verifiable respiratory dysfunction in connection with the diagnosis of an ACS can be found in almost all pediatric patients (detectable in more than 90% of affected ACS patients). This dysfunction can be explained above all by the IAH-related elevation of the diaphragm with the

Table 15.1 Criteria for organ dysfunction, modified according to [57]

Cardiovascular	Despite intravenous application of ≥40 mL/kg isotonic volume in 60 min persisting: • Hypotension with BP <5th percentile for age or systolic BP <2 SD below normal for age OR • Vasoactive drug therapy to keep BP in normal range (dopamine >5 µg/kg/min or epinephrine, norepinephrine, or dobutamine at any dose) OR • Two of the following: – Arterial lactate >2 times upper limit of normal – Prolonged capillary refill >5 s – Oliguria: urine output <0.5 mL/kg/h – Metabolic acidosis (base deficit >5 mmol/L) – Core to peripheral body temperature difference >3 °C
Hematologic	• Thrombocyte count <80,000/mm^3 or decline of 50% in thrombocyte count from highest value recorded over the past 3 days (for chronic hematology/oncology patients) OR • International Normalized Ratio >2
Hepatic	• Total bilirubin ≥ 4mg/dL (not applicable for newborn) OR • ALT 2 times upper limit of normal age
Renal	• Serum creatinine ≥2 times upper limit of normal for age or twofold rise in baseline creatinine
Respiratory	• Oxygenation index <300 in the absence of cyanotic heart disease or preexisting lung disease OR • $PaCO_2$ >65 mmHg or increase of >20 mmHg over baseline OR • Proven need or FiO_2 >0.5 in order to maintain saturation ≥92% OR Need for nonelective mechanical ventilation (invasive or noninvasive)

Table displays diagnostic criteria for cardiovascular, hematologic, hepatic, renal, and respiratory dysfunction according to the International Pediatric Sepsis Consensus Conference
BP blood pressure, *ALT* alanine aminotransferase

successive development of dys- and atelectasis in the basal lobes of the lung. The second most widely made observation is that of cardiocirculatory impairment, and then kidney and liver dysfunction [23].

Neurological impairments are excluded as ACS defining organ dysfunctions, because the majority of ACS patients require an intubation and mechanical respiration with the corresponding analgosedation, resulting in the neurological criteria generally only being viewed and assessed with reservation. Regardless of this, it is debatable whether—depending on the amount of intra-abdominal pressure—this pressure is distributed intracranially after spreading to the thorax and whether—depending on the extent of the resulting stasis—there is also relevant impairment of and damage to intracranial structures in the course of the disease [58–63].

In accordance with IPSCC criteria, there are massive changes in the corresponding vital and laboratory parameters in the case of ACS. However, they do not

show any kind of specificity and cannot be interpreted as chemical biomarkers of an ACS [23]. Various work groups have been looking for such promising biomarkers for years now [64–66], because the transition from a "simple" IAH to ACS begins slowly and is usually recognized (too) late—but then with seriously deleterious results quoad vitam [64–66]. Just recently it was possible to identify and quantify a promising microRNA as well as diverse neuronal guidance proteins [21] that are detectable in significantly higher concentrations in patients' blood only after the transition from IAH to ACS [67, 68]. Furthermore, promising biomarkers include fatty acid-binding proteins [69–71], D-lactate [72], citrulline [73], and circulating tight-junction proteins of the enteral mucosa [74]. Considering the current state of research, it cannot be said how far these laboratory parameters can actually be used as biomarkers in daily clinical practice. Further studies are necessary.

15.1.6 Therapy Options and Goals

On average an ACS diagnosis is made too late. A retrospective investigation of adult patients found that the average diagnosis occurs with a latency of 18 h [75]. The goal of adequate therapy has to be to ensure sufficient perfusion of all tissue and organs, and reestablish it as quickly as possible—at the latest when ACS has been determined but more preferably once IAH has been recognized. Analogous to the surgical maxim with the ileus, the sun should not set and rise between when ACS is diagnosed and the therapeutic objective is reached (prose version of the max. 6-h ischemia rule). For estimation, determining abdominal perfusion pressure (APP) can be useful. Similar to cerebral perfusion pressure, APP = MAP − IAP (with MAP: mean arterial pressure; IAP: intra-abdominal pressure) [76, 77] [in the past by some authors (synonymous) also referred to as splanchnic perfusion pressure (SPP) [78, 79]]. Individual authors describe perfusion pressure instead as the difference in pressure between diastolic pressure and IAP. Considered in contrast to MAPs—but also considering the damaging components of stasis when diastolic pressure is exceeded—this form of calculation has yet to take hold. As long as the data available refer more to MAP, the methods first mentioned, and formulas, should be applied in an evidence-based way.

The goal of adequate therapy should be for MAP as well as APP to be oriented towards the standard blood pressure range that corresponds with the patient's age (see Table 15.2 [80]). An iatrogenic increase in MAP via forced catecholamine therapy with the goal of achieving an age-appropriate normalization of APP is neither useful nor sustainable and, thus, obsolete. In the neonatal age, for example, the goal MAP level is the number of weeks that have passed since conception (in mmHg). Accordingly, a newborn delivered in the 36th week of pregnancy should have an average MAP of 36 mmHg. Considering their cardiovascular condition in cases of even moderate increases in IAP as well as the impairment of their microcirculation due to IAH, a neonatal or infant patient is at a significantly greater risk than an almost grown adolescent with age-adapted MAP standard limit of 70 or 80 mmHg.

Table 15.2 Age-appropriate standard value areas of mean arterial pressure [mmHg]; modified according to [80]

Age group limits [in months]		Mean arterial pressure (MAP) [mmHg]		
Lower limit	Upper limit	−2 SD	Average	+2 SD
1	3	40	50	60
>3	6	45	60	75
>6	12	50	70	90
>12	47	50	75	90
>47	83	55	75	95
>83	131	60	75	95
>131	167	65	80	95
>167	216	70	83	95

Abbreviation: *SD* standard deviation

For risk stratification of quantified IAPs, it is necessary to be aware of age-appropriate blood pressure values (with ±2 standard deviations [SD], see Table 15.2).

The recommendations published by the WCACS regarding medical, interventional, and emergency surgical therapy options in cases of a relevant IAH or ACS are also valid for children [9]. If using a feeding tube and purgative measures in addition to creating a negative balance with the help of diuretic therapeutics and emergency dialysis procedures is not enough, sufficiently deep analgosedation and even relaxation following previous intubation and mechanical ventilation are necessary [9]. Ascites that can be punctured or other effusions should be relieved generously [81]. Should a decompression be unavoidable due to the IAP dynamic, the clinical and above all intraoperative development of IAP should be used to consider the necessity of a laparostomy (synonym: open abdomen management, abdomen apertum, surgical enlargement of the abdominal wall, etc.). In the framework of the surveillance study mentioned above [23], a decompressive laparotomy was performed on 2/3 of the children studied; the abdomen was left open a median of 7.5 days (mean 9.9 ± 5.5) in 44%. A median of 4.0 (on average 3.2 ± 2.2) operations were necessary to reclose the abdomen. In 76% of the cases, open abdomen management was not associated with any complications. In the remaining cases, infectious septic events were more dominant than wound-healing disorders, adhesions, and failure of foreign materials that had been introduced. Enterocutaneous fistulas, the most common complication following open abdomen management in adults, were not observed in this pediatric study [23].

While a total of 83% of the patients on whom operative decompression was performed survived, 58% of children in whom there was an indication for temporary abdominal wall surgery survived. Thus, when a temporary surgical enlargement of the abdominal wall was implemented, the probability of pediatric patient survival was on average higher than when conservative therapy was administered (survival 40–60%) [23].

The median length of stay for pediatric intensive care patients with ACS who survived was 25.5 days in the ICU (mean 42.9 ± 42.2), and a total of 42.5 days in the hospital (mean 59.6 ± 49.5). Patients who did not survive ACS died medianly after 12 days (average 25.2 ± 35.5). In 74% of these cases, multi-organ failure that

could not be controlled via organ replacement procedures was the cause [23]. In the remaining cases, it was incontrollable pulmonal arterial hypertonia, cardiovascular failure, bleeding complications due to impaired coagulation, and therapy-resistant tumor growth [23].

15.2 Conclusion

Thorough training appears to make it possible to create a sensitization for this topic and accelerate the application of adequate, and courageous, therapy options. Standard operating procedures with flowcharts on age-appropriate and problem-oriented diagnosis as well as therapy should increase the willingness to also act invasively and choose heretofore unpopular methods and options that can massively influence and ensure survival in pediatric patients. Initial outcome data are motivating and suggest that invasive therapy possibilities can be beneficial to survival in cases of abdominal compartment syndrome.

Only this seems to be the way to reduce morbidity and mortality in the mid to long term among the smallest patients.

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