Evolution of Resuscitation: A Historical Perspective

Sir,

In recent years, most of the hospitals conduct resuscitation courses, including advanced cardiac life support (ACLS), pediatric advanced life support (PALS) and neonatal resuscitation courses. The benefits of these courses have been shown earlier. The courses have shown to improve the level of comfort and readiness for the emergencies among the health care provider.[1-4] This brief article is about the evolution of resuscitation.

Basic life support (mouth to mouth breathing). In 1732, James Blair was rescued from a fire in a coal mine and William Tossach, a Scottish surgeon described the effect of mouth to mouth breathing on the same patient. In 1745, the paper of John Fothegill, a London practitioner, recognized the technique of mouth to mouth resuscitation. After that the Society for the recovery of Drowned Persons was formed in Amsterdam in 1767 followed by a Royal Humane Society founded in London in 1774.

Neonatal resuscitation. In 1834, Dr. James Blunndell, a Professor of Obstetrics at Guy's Hospital gave a detailed lecture on newborn resuscitation.

Artificial ventilation. In 1949, Archer Gordon re-evaluated the different methods of artificial ventilation. In 1954, James Elam proved it scientifically. In 1957, Dr. Peter Safar showed that tilting of the head could open the airway.

First intubation. The first report of experimental intubation of the trachea was reported by the great muslim philosopher and physician Avicenna (Abu Ali Al-Hussein Ibn Abdullah Ibn Sinna) in approximately the year 1000. The English physician, Charles Kite, recognized the importance of endotracheal intubation. In 1754, Benjamin Pugh developed the first endotracheal tube for resuscitation of neonates. James Curry in 1815, illustrated the technique of orotracheal intubation.

Heart and lung as a unit in resuscitation. In the 16th century the great Flemish anatomist Andreas Vesalius related ventilation to heart function in an animal.

Chest compression. Although much attention was given to the ventilation part of resuscitation, the role of chest compression was ignored until 1878, when Boehm and subsequently Koenig in 1883 described the role of external chest compression in cats and humans.

Medications. Crile in 1914 recognized the importance of simultaneous chest and abdominal compression and adrenaline was used for the first time.

First resuscitation. The first description of successful resuscitation was recounted in the Bible, in the Book of Kings.

The first recognition. In 1963, America Heart Association (AHA) endorsed the method of resuscitation for medical personnel, which was extended to the public in 1973. The first advanced life support course was offered in 1975, since then resuscitation courses have become an important part of the training of health care providers.

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Congenital chloride diarrhea from the west coast of the Kingdom of Saudi Arabia

Sir,

Congenital chloride diarrhea (CCD) is an autosomal recessive disease. The first case was reported in 1945 by Gamble and Darrow.[1,2] Since then, cases have been reported from different countries all over the world. In the Kingdom of Saudi Arabia (KSA), the first cases were reported in 1988 by Dr. Al-Mershedi from the Riyadh Armed Forces Hospital[3] and after that all cases of CCD in Saudi Arabia have been reported from the central region from different centers including King Faisal Specialist Hospital, and King Fahad National Guard Hospital with an estimated incidence of 1:5500.[4-6] The estimated incidence of CCD in a multi center study from Kuwait was 1:14000.[7] From our study, the estimated incidence in the Jeddah region is 1:7000. With a high incidence of consanguineous marriage in KSA, it would not be surprising if we have a higher incidence of CCD. Further studies are needed to prove this. No study has been carried out to localize the gene locus for CCD in KSA patients. Recently, the CCD gene has been mapped by linkage disequilibrium mapping to chromosomes region 7q31, from Finland where they have the majority of reported cases of CCD.[8-10]

The cases were studied retrospectively from admissions from November 1989 until May 1997. We studied the cases diagnosed according to the features noted below. We report 8 cases; 5 male and 3 female, all of whom are Saudi and 5 out of the 8 are children of 1st cousin marriages. Three children were from one family making antenatal and early neonatal diagnosis easy. The other 2 were cases of chronic diarrhea with ileostomy. We concentrated in this study on clinical presentation, biochemical changes and diagnosis. Consanguinity was present in 7 out of 8 patients, the present national average, and 7 patients developed watery diarrhea in very early neonatal life. Only one patient presented later at 9 months of age with diarrhea and this child's mother was unbooked for antenatal care. Five patients had maternal polyhydramnios. Two patients were referred with a diagnosis of Bartter's Syndrome. Three patients who had mainly abdominal distension in the newborn period with a diagnosis of intestinal obstruction had ileostomy carried out and one also had colostomy performed. Three patients from one family were diagnosed and treated from early life. All patients had neonatal unconjugated hyperbilirubinemia (Table 1).

Congenital chloride diarrhea has the characteristic biochemical changes of hypochloremic, hypokalemic, hyponatremic, metabolic alkalosis. High stool chloride exceeding serum chloride and very low or undetectable urine chloride with high aldosterone and renin are found. Mean biochemical features at presentation are shown in Table 2. The lowest serum chloride of our patients was 52 mmol/L and highest stool chloride 149. All 8 patients had hyponatremia and 6 patients had hypokalemia at the time of presentation. All of them have metabolic alkalosis and low urine chloride. Only one patient has aldosterone and renin levels measured, and these were high. Only one of our patients was shown to have abnormal kidneys on ultrasound, and 5 of them have had reasonable catch up growth. One girl grows below the 3rd centile for her weight and length but her parents are of short stature. Two

Table 1 - Clinical presentation.

Patient No.	Consanguinity	Age of presentation	Age of diagnosis	Gestational age	Family history	Jaundice	Complication	Reason for referral
1 boy	1st cousin	9 months	9 months	35 weeks	-	+	FTT	? Bartter Syndrome
2 boy	No consanguinity	Day 15	4 months	34 weeks	-	+	?internal obstruction -ileostomy +colostomy	Persistant electrolyte distrubances after closure of colostomy
3 girl	1st cousin	Day 10	8 months	Full term	-	+	FTT slow development	? Bartter Syndrome
4 boy	1st cousin	Day 2	Newborn	37 weeks	+	+	-	+ve family history
5 boy	1st cousin	Day 1	Newborn	Full term	+	+		+ve family history
6 girl	1st cousin	Day 1	Antenatal	Full term	+	+		+ve family history
7 boy	2nd degree relative	Day 2	1 month	33 weeks	-	+	Ileostomy volvulus Gut resection Developmental delay	Chronic diarrhea FTT
8 girl	2nd degree relative	Day 5	6 months	31 weeks	-	+	Ileostomy Developmental delay	Chronic diarrhea

Table 2 - Biochemical changes and antenatal ultrasound findings.

	PH	HCO_3 mmol/L	Na mmol/L	K mmol/L	Cl mmol/L	Stool Cl mmol/L	Urine Cl mmol/L	Aldosterone	Renin	Antenatal ultrasound
Normal values	**7.3-7.4**	**22-26**	**135-150**	**3-5**	**96-111**	**16**	**2-16**			
Patient No.										
1 boy	7.65	49	126	2.2	60	79	<15	Not done	Not done	Unbooked
2 boy	7.47	25	128	2.7	70	129	<15	Not done	Not done	Polyhydraminos
3 girl	7.50	21	127	1.2	52	140	<15	High	High	Dilated intestinal loops
4 boy	7.58	31.13	128	2.6	76	149	16	Not done	Not done	Polyhydraminos
5 boy	7.43	20	128	5.1	88	113	<15	Not done	Not done	Cystic bowel
6 girl	7.43	24	130	5.6	97	105	<15	Not done	Not done	Polyhydraminos
7 boy	7.48	26	123	2.6	93	148	<15	Not done	Not done	Polyhydraminos
8 girl	7.50	38	130	3.3	78	133	<15	Not done	Not done	Polyhydraminos

patients have recently been diagnosed and we cannot comment on their growth velocity but they presented with FTT and developmental delay and were born with low birth weight (number 7 and 8).

Congenital chloride diarrhoea is a rare autosomal recessive condition but may be common in KSA with an estimated incidence of 1:7000 and this may be related to a high frequency of the CCD gene and to a high percentage of consanguineous marriages. Proper studies to find the gene locus of CCD in KSA should be performed. All pediatricians, neonatologists, pediatric surgeons and obstetricians should be aware of CCD, as early diagnosis secures the babies' life and enables normal life and development and may reduce unnecessary surgery as the vast majority of cases manifest in very early neonatal life.[11,12] All mothers who were booked for antenatal care developed polyhydraminos,[13-16] and all our patients had neonatal jaundice because of hypovolemia and prematurity. Only one patient had an echogenic kidney.[17] Congenital chloride diarrhea is a relatively common condition seen in KSA, in both the western region and the center, and gene mapping should be encouraged for Saudi children with CCD.

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