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AN ANALYSIS OF THE MEDIAL THICKNESS OF SMALL PULMONARY ARTERIES IN VICTIMS OF THE SUDDEN INFANT DEATH SYNDROME

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ABSTRACT

The medial thickness of small pulmonary arteries in 40 victims of the Sudden Infant Death Syndrome (SIDS) and 12 controls has been determined. For the purposes of analysis infants were allocated according to age into one of 3 groups: 30-90, 91-180 and 181-365 days. The arterial media in the 30-90 day control group was marginally thicker than that in the SIDS victims; no other SIDS related effects were noticed.

INTRODUCTION

Steinschneider (1972) proposed that sleep apnea might form part of the final pathway in SIDS. Naeye (1973) followed up this hypothesis and examined SIDS victims for evidence of chronic alveolar hypoxia. Amongst other supporting evidence he observed a thickening of the media in the small (30-100 μm diameter) pulmonary arteries of the SIDS victims. Subsequent studies produced conflicting data (Mason et al, 1975; Kendeel and Ferris, 1977; Williams et al, 1979). This study was designed to resolve the question of whether medial thickening is a feature of SIDS.

MATERIALS AND METHODS

Lungs from 40 consecutive SIDS victims dying in the Melbourne area were collected over a period of 6 months from June to December. Lungs from 12 controls dying as a result of trauma (10) or acute infection (2) were collected over a period of 3 years (all the controls died before treatment could be initiated). For the purpose of analysis SIDS and control infants were grouped by age (Table 1), following Deacon et al (1979).

Table 1. Breakdown of SIDS and control infants by age (days) and sex

Age (Days)	SIDS Male	SIDS Female	CONTROL Male	CONTROL Female
	Av. Age		Av. Age	
30-90	7	9	2	4
	64 ± 17		63 ± 18	
90-180	9	7	2	1
	126 ± 18		144 ± 44	
181-365	4	4	1	2
	242 ± 73		250 ± 58	

At post-mortem the right lung of each infant was removed; the lower lobe bronchus was infused with Bouin's solution at a pressure of 40 Pa, and the lobe was immersed in fixative. After approximately 3 h of fixation a 5 mm slab of tissue was taken perpendicular to the line of the bronchus approximately midway through the lung; this slab was divided into blocks and fixed for a further 20 h, after which they were processed and embedded in wax. Sections were cut at 3 um, stained for elastin with aldehyde fuchsin and counterstained with Lendrum's MSB.

One section from each block was systematically scanned using a Leitz Orthoplan microscope in conjunction with a drawing tube and a Leitz A.S.M. All pulmonary arteries with well-defined elastic laminae were measured using either a 63x or 40x objective lens.

The perimeter and areas bounded by the internal and external elastic laminae were determined. The diameter of an equivalent distended circular vessel was then determined from the length of the internal elastic lamina (Cook and Yates, 1972). Using this diameter together with the area of the media, the external diameter of the media and the average medial thickness of the vessel were calculated. The data thus derived was expressed as % medial thickness (MT) defined as: %MT = 2x average MT x 100/external diameter (Williams et al, 1979). The data was grouped according to external diameter at 10 μm intervals and the mean of each interval from each infant was used for subsequent calculations.

RESULTS

In all lungs studied %MT showed an inverse exponential relationship with external diameter of the form: $y = a+be^{-k(\text{ext diam})}$ (Fig. 1). The values for 'a' and 'b' for each group were then determined using non-linear estimation techniques.

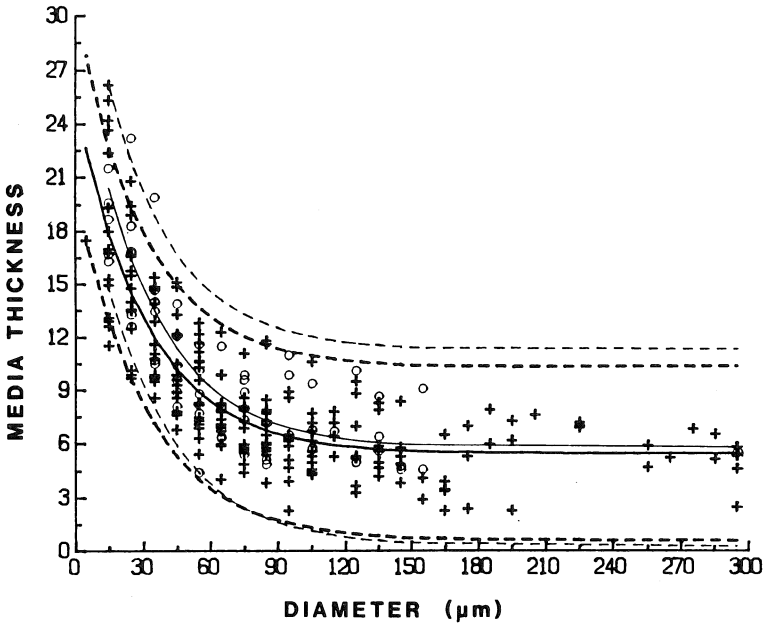

Fig. 1. Data plot for 30-90 day SIDS (+ , thick lines) controls (∘ , thin lines) ± 95% confidence intervals.

The derived data is shown in Table 2.

Table 2. Regression coefficients for Control and SIDS infants by age group

Age		Control	SIDS
30-90	a	5.8 ± 0.7	5.5 ± 0.4
	b	26.1 ± 3.4	19.6 ± 1.3
91-180	a	5.4 ± 0.4	5.1 ± 0.2
	b	17.5 ± 3.9	15.7 ± 1.5
181-365	a	6.9 ± 0.5	6.1 ± 0.2
	b	15.0 ± 3.5	18.2 ± 2.6

Groups were then compared using a parallel curve analysis (one of the non-linear analogues of the test of parallelism) to determine differences. Significance testing revealed that the displacement of the curve for the 30-90 day controls was significantly greater ($P<0.01$) than that for the SIDS victims; no other significant differences were observed.

DISCUSSION

The value of 'a' reflects the medial thickness of large arteries whilst the value of 'a+b' reflects the rate of increase of medial thickness with diminishing vessel size.

Differences in the results of this and previous studies probably relate to methodological factors. Some comment on the method used by Naeye (1973) is offered by Cook and Yates (1972). The technique used by Williams et al (1979) depends on perfusion of post mortem tissue with a hot (60°C) gelatin solution and it is difficult to see how this can be done reproducibly. It can be shown by simple geometry that the MT figure derived in this report is independent of section plane; determination of the true external diameter is dependent on plane of section, and in general this will have been overestimated in this work.

These studies reveal no evidence of medial thickening in SIDS and thus offer no support for the proposition that chronic alveolar hypoxia plays a role in its aetiology.

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