The treatment of bronchitis in children / by Joseph E. winters.

THE TREATMENT OF BRONCHITIS IN CHILDREN.

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THE treatment of bronchitis in children necessitates intimate acquaintance with the structure of the bronchial wall, and with every alteration in this structure at each step and every hour of the morbid process.

These walls unite unique anatomical constituents, each and every one of which influence the symptoms and the treatment of bronchial inflammation, and should, therefore, be present in the mind of every practical physician.

From *without* inward, a bronchus is composed of an *external* fibrous coat of dense fibrilated connective tissue with embedded cartilages. The cartilages are for keeping the tubes open under varying conditions of respiratory pressure. Without this framework the tubes would collapse with inspiration. A middle coat of transverse muscular fibres, which at their ends are inserted, by a ligamentous attachment, into the inner surfaces of the cartilages, a short dis-

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tance from their tips, and into the external fibrous coat. These transverse muscles dilate the bronchus with inspiration. An internal mucous coat, known as the mucosa.

The mucosa is composed of three coats: From *within* outward, an epithelial layer; a homogeneous basement membrane, giving attachment to the epithelium; an *inner* fibrous coat, of longitudinal elastic fibres, containing a very copious and an exceedingly fine network of blood-vessels, likewise longitudinal. This is also known as the vascular coat. The interposition of a perfectly homogeneous membrane between the epithelium and the loose longitudinal network of vessels is of great practical value in bronchitis. This clear homogeneous layer is most aptly designated a limiting or boundary membrane, owing to its intervention between those coats in which the primary, all-determining changes of bronchitis take place, *i.e.*, congestion, desquamation of epithelium.

Superimposed in all these structures are numerous mucous glands.

In no organ of the body is such a complex variety of structural elements involved in an inflammation as in bronchitis.

STRUCTURE OF SMALL TUBES.

Bronchi one-twenty-fourth of an inch or less in diameter are so different in composition from those above this size that when they are the seat of inflammation the type of symptoms announce their implication. They are destitute of cartilaginous framework

and of ciliated epithelium. They contain no mucous glands. The basement or limiting membrane diminishes *pari passu* with that of the bronchi, and in their smallest ramifications is imperceptible—of gravest import in congestion of the tubes. These tubes receive instead of giving support. They have a peculiar zigzag course—most unfortunate with increased secretions.

FINAL ENDING OF DIFFERENT COATS.

The *external* fibrous coat becomes the peri-lobular capsule, the inter-lobular tissue, and is continuous with the sub-pleural tissue. The unity of the outer wall of the bronchus, and the peri-lobular, inter-lobular, and sub-pleural structures, in conjunction with their common source of arterial supply, have pathological bearings of the very highest importance. The *inner* fibrous elastic coat becomes the alveolar wall.

VASCULARIZATION OF BRONCHI.

The bronchial arteries form two plexuses in the bronchial wall—an outer *transverse* plexus in the muscular layer, for supplying the muscular coat, the external fibrous coat, the peri-lobular capsule, the inter-lobular tissue, the pleura, the bronchial glands, and the nervous ganglia. An inner, richer *longitudinal* plexus, in the *inner* fibrous coat, *for supplying the mucous membrane exclusively.* The capillaries of this inner plexus, *to the bronchi of the fourth division*, empty into the pulmonary veins, and are, therefore, in direct communication with the left heart. The pulmonary

veins have no valves. *Injection of them produces injection of the bronchial capillaries* before the pulmonary arteries—of serious concern in bronchitis with mitral lesion.

AERATION OF BLOOD IN BRONCHIAL CAPILLARIES.

As the bronchial capillaries are exceedingly small, and as a stream of air is constantly passing over them, *when the tubes are clear*, the blood is probably aerated before mingling with the arterialized blood of the pulmonary veins.

There are still further anatomical factors equally essential to a basis for competent physiological treatment.

The pulmonary vessels hang in the bronchial tree, and exceed in size the tubes which support them—notably so amongst those tubes which are destitute of protective framework. To the large tube the accompanying pulmonary *artery* is loosely attached, while it clings tightly to the defenceless small tube.

The area of the pulmonary capillaries is less than the area of the systemic capillaries. The amount of blood passing through these vessels at each cardiac contraction is exactly equal to the amount propelled into the systemic circulation. As these vessels lean upon the small bronchi for lateral support, increased pressure in the pulmonary vessels in a young child endangers the integrity of the lumen of the bronchi—every pulmonary congestion in an infant presages evil.

In children up to the fourth or fifth year there is a tendency to rapid and dangerous extension of every bronchial catarrh to the smaller bronchi.

This is explained by the anatomical peculiarities at this period. In young children the longitudinal elastic tissue of the fibro-vascular coat is abundant and loose, and offers slight restraint when pressure in the loosely held *longitudinal* vessels is increased by inflammation.

After the fifth year these structures rapidly assume the adult type, the connective tissue becoming dense and binding, restraining the vessels as in the adult.

What could be more intricate, what more fraught with perplexity and dismay, than bronchitis of the small tubes.

Inadvertent, inattentive treatment invites inevitable disaster, while expert physiological management affords results both felicitous and gratifying.

ALTERATIONS IN THE BRONCHIAL WALL IN ACUTE BRONCHITIS.

Prognosis, and enlightened recourse to therapeutic measures, are based upon and determined by the alterations in the *inner* vascular plexus, at the inception and during the progress of bronchitis.

The inception of bronchitis is *distention of the loose longitudinal plexus in the inner fibrous coat,* immediately beneath the basement membrane. The distended vessels push the basement membrane before them and appear as villous-like prominences on the surface. The basement membrane becomes œdematous and is

thrown into folds. Sometimes the vessels burst and small hemorrhages occur into the mucous membrane. Other vessels are so engorged as to appear like small cavities filled with blood corpuscles.

The rapidity with which other changes succeed this primary alteration is variable. In ordinary cases the epithelium desquamates in twenty-four hours; in severe inflammations, as from influenza and the exanthemata, in four to six hours. The cilia are first thrown off. At this stage there is little or no secretion of mucus, merely redness and tumefaction.

Uncontrolled distention of the vessels, however, is quickly followed by exudation and by increased secretion from the mucous glands. Large quantities of mucus are generated from the epithelial cells alone, while at the same time it wells out from the ducts of the mucous glands—the tubes are rapidly inundated.

Prompt physiological intervention must be resorted to at inception to intercept those further secondary changes which incite complications and fatal terminations.

If not restrained, the inflammation soon extends into the minute bronchi. This is heralded by recession of the lower ribs with inspiration. Blocked tubes, receding chest walls, and weak inspiratory muscles superinduce alveolar collapse as an epiphenomenon.

To obviate fatal issue, extension to the small tubes must be forestalled.

In children under two years bronchitis is one of the most fatal diseases of childhood. Out of 1,000 deaths from this disease, 419 were in children under two, and

490 were under five years of age; nearly all of the remainder (457) were in adults over fifty-five years of age.[1]

An especially fatal form is that which comes soon after birth, as a result of imprudence in bathing, from low temperature of the room, or from exposing the child to the open air.

There are two types: One is sudden in accession, rapid in course, involving the *inner* plexus. Active physiological counteraction must be instituted at once, or the issue is fatal in a few days.

The other is characterized by insidiousness of invasion, indistinctness of symptoms and physical signs, obstinacy of course, yielding little if at all to treatment.

If after the lapse of several weeks this latter form terminates fatally, post mortem discloses involvement of all the ramifications of the *outer* plexus of the bronchial arteries, *i.e.*, the external fibrous coat of the bronchi, the peri-lobular capsule, the inter-lobular connective tissue, the sub-pleural tissue, the bronchial glands and even the nervous ganglia. The capillaries in the connective tissue of the nervous ganglia are engorged, and by pressure on the ganglia cause spasmodic dyspnœa which supervenes in paroxysms most terrible to witness.

The only type of bronchitis of which a clinical account is requisite here, is that of the small tubes—suffocative bronchitis, suffocative catarrh. Always secondary, due to rapid extension from the larger tubes,

[1] One-half the cases in children under five years are fatal (Samuel West).

its accession is so abrupt, so marked by profound collapse, small feeble pulse and anxious countenance, as to give the appearance of a primary affection.

Sudden distention of the inner plexus, by hyperæmic swelling of the mucous membrane, may prove fatal before the stage of mucopurulent secretion. The rapidity of the respiration and pulse are out of all proportion to the other symptoms, and the respirations relatively accelerated far beyond the pulse rate. I have seen four cases in young infants where the respirations were 104 in two, and 108 and 110 in the others. Two pulse beats to one respiration is not uncommon, instead of three or four beats to one respiration, as in the normal condition.

The respiratory act is characteristic: A short labored inspiration, in which all the accessory inspiratory muscles participate, marked recession of the lower part of the thorax, and retraction of all the inferior intercostal spaces, followed by prolonged difficult expiration, in which the abdominal muscles participate. Whenever the expiration becomes prolonged in bronchitis, extension into the fine tubes is to be apprehended.

AUSCULTATION.

At both bases posteriorly, and especially over those narrow strips of lung between the angles of the ribs and the spine, and the tongue-shaped process of lung which overlaps the heart, fine crepitations are heard; or, the tubes may be so blocked by tough mucus, or so narrowed by tumefaction of the mucous membrane,

that neither crepitations nor respiratory murmur are audible—the breath-sound entirely obliterated over both bases posteriorly.

TEMPERATURE.

The temperature has no diagnostic value. A patient may have a temperature of 104° F. at one hour, and of 100° F. at another time of the same day. A paroxysm of extreme dyspnœa, great restlessness, constant tossing from side to side, may be attended by a temperature of 105° F., or even more. After a fit of coughing ending with vomiting, followed by sleep from exhaustion, the temperature drops to 100° F.

TREATMENT.

Clinical experience and pathological investigation yield ample proof that bronchitis in a young child is never slight.

The coryza of to-day may before the morrow spread to the small bronchi, for, like its congener, catarrhal croup, its onslaught is commonly at night. To forestall and thwart extension is the pith of treatment. If this is accomplished fatal issue is averted. This is well-nigh attainable in every instance.

Distention of the branches of the bronchial arteries is the first deviation from the normal. The design must be to restrain and counteract this at its inception.

By the cutaneous capillaries, by the intestinal mucous membrane, and by cardiac inhibition, the initial lesion may be subdued and abridged; irritation of

the mucous membrane may be abated by the air the patient breathes—mild diaphoresis, gentle catharsis, cardiac sedatives, warm soothing air for the irritated respiratory surface.

The temperature of the respired air should be uniformly 72° F.—day and night—sun exposure and open fire if practicable. Ventilation must secure at all hours pure, fresh air.

The child should be in a crib—never on a bed. The crib should be flannel-lined and be in the centre of the room—never near a wall, window, or door. Sometimes it is necessary to place screens about the crib to avoid draught.

Light flannel should envelop the child's body, arms, and legs. The flannel shirt should be secured to the diaper by safety-pins, back, sides and front; long worsted stockings should be worn and likewise fastened to the diaper. The wrap and shirt must be loose and open in front, secured by tapes or safety-pins, assuring ready access for physical examination. A duplicate suit is to be kept in readiness. A quick hot bath precedes flannel envelopment.

In a case of moderate severity, nine-tenths of the treatment has been compassed.

DRUGS.

In severe cases the drug of unfailing, universal efficacy is aconite. The bronchial arteries, branches of the aorta, or intercostal arteries, subject to direct, immediate, forcible cardiac pressure, surge with blood. To restrain and limit this is the aim. Through this

drug arterial pressure is promptly, safely circumscribed. Maximum, frequent doses during the first hours; diminished, less frequent doses after four or six hours; early abandonment. It should be given in water only—tasteless, non-nauseating, it does not affect appetite. For a child of one year—

℞ Tr. aconite (Fleming).....gtt. iv.
Aq. destillat.....oz. iii.

M. Sig. One teaspoonful every fifteen minutes for one hour; every half hour for four or six hours; then every hour for twenty-four more hours.

Turgescence ceasing, the remedy is discontinued.

Arterial pressure is lessened by diaphoresis. Sweet spirits of niter is the pre-eminent diaphoretic. Niter, citrate of potassium, and spirits Mindererus may alone be used where aconite is not urgently indicated, and follows discontinuance of it. The combination often nauseates, and even more frequently affects the sense of taste so disagreeably that nourishment is persistently refused.

Intestinal elimination, diaphoresis, aconite, and niter make up the febrifuge measures. Other antifebriles (baths, sponging, coal-tar derivatives) are, individually and collectively, contraindicated.

Cold to the cutaneous capillaries is unphysiological, pernicious; conduces to extension.

STAGE OF SECRETION.

Excessive secretion may inundate the bronchi and must be anticipated and intercepted.

The agents which diminish secretion are camphor,

carbonate of ammonia, nux vomica, oxygen inhalations, and counter-irritation. Spirits of camphor is the most valuable drug agent.

For a child of one year—

℞ Spirits camph..... ʒ i.
Saccharin..... gr. i.
Spts. etheris nit. (for preventing precipitation of camphor)..... ʒ ii.
Syrp. tolut. (for diminishing pungency)..... ℥ ss.
Aq. gaulth. q.s. *ad*..... ʒ iii.
M. Sig. One teaspoonful every half hour.

Carbonate of ammonia in one-grain doses is a valuable adjuvant to this mixture, but often provokes nausea. Nux vomica has the same objection.

When the tubes are loaded with tenacious secretion, mustard is of priceless service. One part mustard, six parts flour mixed with *cold* water and white of egg, and applied as a paste between two layers of thin linen, covering the entire region where moist sounds are heard, left on twenty to thirty minutes and renewed every two or four hours, according to the condition of the skin.

Where these resources have not been initiated at the opportune moment, or a child has not been seen until the bronchi are flooded with secretions, superinducing laborious and yet shallow breathing; on auscultation very few râles or none at all are heard, breath-sound absent over both bases posteriorly; pulse exceedingly rapid and small and disappears under the finger; suppressed cough from CO_2, medical art is put to its surest test, for even this extremity is surmountable by skil-

ful adaptation of that knowledge and power born of pathology and experience.[1]

Impetuous stimulation is the common, almost universal blunder. The deluged tubes must be unloaded, emptied; respiration and oxygenation re-established. Blocked bronchi soft; yielding thoracic walls; weak, exhausted inspiratory muscles, make lobular collapse irresistible. The only redeemable, relievable factor is the obstruction to the passage of oxygen.

Witness the breathing become more and more shallow, until there is only slight movement of a few ribs and faint jerking contractions of the diaphragm; every untoward phase is accentuating swiftly. The swallowed sputa produces vomiting; the diaphragm, the abdominal muscles, the muscles of the lower part of the chest compress the thorax as in a vise. The tubes are squeezed, the contents forced out, mucopus wells out of the mouth. A sudden deep inspiration, the whole chest expands, the breath-sound is heard over the entire pulmonary area. Air has penetrated the most remote air-sacs; oxygenation is renewed, blood-cells revived, re-animated, cyanosis is replaced by vivid pink!

The position the child was in while vomiting—face downward—is maintained, the secretions gravitating to larger, sensitive bronchi induce cough, and are ejected in place of being swallowed. The cue is obvious—emetics, postural treatment.

When the air tubes are blocked by tenacious mucopus, they can only be freed by active emesis. This should not be repeated more than once or twice in the twenty-

[1] The cases mentioned on page 8 all recovered.

four hours. Two or three clearings are adequate. The child must be kept in the prone position to secure gravitation of secretions to a sensitive mucous surface to avoid refilling. Oxygen inhalation must be *continuous* in this critical state—even during sleep.

These forceful resources are supplemented by mustard packs and maximum doses of spirits camphor and carbonate of ammonia.

PROGNOSIS.

Hitherto harassing attacks of coughing become weaker or cease altogether, owing to obtunded sensibility from CO_2; suppression of râles without corresponding amendment; laborious and yet shallow breathing with recession of lower part of the chest walls and sinking in of the intercostal spaces; tympanitic distention of abdomen interfering with descent of diaphragm; sudden alteration of pulse-respiration ratio; small, rapid, irregular pulse which disappears under finger; apathy, drowsiness, ashy-gray face; fulness of superficial veins, cyanosis, cold extremities, are all most unpropitious tokens.

Diminution in frequency, and increase in depth of respirations, are the first indications of amendment. Râles and breath-sounds return, arteries become fuller, veins less full. In every chest affection in a child the abdomen must be solicitously watched to anticipate apprehensive distention. Dietetic care, and freeing from swallowed sputa, anticipatory of fermentation and tympanitic distention, is uniformly obligatory.

INFLUENCE OF PREVIOUS FEEDING ON PROGNOSIS.

A young child previously fed on *any* patent or proprietary food, on pasteurized or sterilized milk, or for a prolonged period on condensed milk, almost invariably succumbs to bronchitis of the small tubes. The erroneously fed, flabby, soft-of-flesh, rickety child with hypersecretion, must be stimulated after emptying the air channels by vomiting. He who individualizes is the highest type of the medical practitioner.

Laryngo-tracheitis, tracheo-bronchitis, and bronchitis of the large- and medium-sized tubes are usually broken and ended by the management narrated on pages 9, 10, and 11.

An efficient remedy for harassing cough in inflammation of the upper respiratory passage is oleum ricini.

For a child of two years—

℞ Olei ricini..... oz. ½.
Saccharin..... gr. ii.
Acacia pulv..... q. s.
M. Ft. Emulsio et adde.
℞ Spts. etheris nitrosi..... oz. i.
Aq. calcis q.s. *ad*..... oz. iii.
M. Sig. One teaspoonful every hour.

When cough is distressing, counter-irritation over larynx and sternum may be superadded in the form of flaxseed poultice with mustard sprinkled over its surface.

In older children where the irritation of coughing superinduces nervous irritability, it may become nec-

essary to supplement the prescription by small doses of codeine.

In inflammation of larynx, trachea, or bronchi with much temperature, aconite should always be to the fore.

STEAM INHALATION.

Steam inhalation does not meet one physiological, pathological, or symptomalogical indication that is not better, more completely and far more wisely fulfilled by the breathing of warm air, by dilatation of the cutaneous capillaries, by aconite, and by counter-irritation.

Steam inhalation increases susceptibility to cold, to catarrh of the larynx and bronchi—to catarrhal croup.

A croup kettle, therefore, must never be used except for croup.

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