

The Upper Paleolithic Triple Burial of Dolní Věstonice: Pathology and Funerary Behavior

Vincenzo Formicola,^{1*} Antonella Pontrandolfi,¹ and Jiří Svoboda²

¹*Department of Ethology, Ecology, and Evolution, University of Pisa, 56126 Pisa, Italy*

²*Archaeological Institute, Czech Academy of Sciences, 66203 Brno, Czech Republic*

KEY WORDS paleopathology; Upper Paleolithic; funerary behavior; Dolní Věstonice

ABSTRACT This work focuses on paleopathological analysis of one of the skeletons from the Gravettian triple burial of Dolní Věstonice (Moravia) and addresses issues of Upper Paleolithic funerary behavior. The burial includes the well-preserved skeletons of three young individuals. The skeleton in the middle (DV 15) is pathological and very problematic to sex; the other two (DV 13 and DV 14) are males and lie in an unusual position. The young age, the possibility of a simultaneous interment, and the position of the three specimens have given rise to speculations about the symbolic significance of this spectacular and intriguing funerary pattern. The pathological condition of the skeleton in the middle further emphasizes its peculiarity.

Main pathological changes of the DV 15 skeleton include: asymmetric shortening of the right femur and of left forearm bones, bowing of the right femur, right humerus, and left radius, elongation of fibulae, dysplasias of the vertebral col-

umn, and very marked enamel hypoplasias. Scrutiny of the medical literature suggests that the most likely etiology is chondrodysplasia calcificans punctata (CCP) complicated by trauma and early fractures of the upper limbs. CCP is a rare inherited disorder characterized by stippled ossification of the epiphyses. The cartilaginous stippling is a transient phenomenon that disappears during infancy, leaving permanent deformities on affected bones. Among the different forms of CCP, the X-linked dominant form is that resulting in asymmetric shortening and is lethal during early infancy in males. Thus, survival of DV 15 until young adult age would require the specimen to be a female. Clinical findings often associated with the disease (erythemas, ichthyosis, alopecia, cataracts, and joint contractures, among others) would emphasize the singular aspect of this individual, pointing to a condition that should be carefully taken into account when speculating on the significance of that peculiar burial. *Am J Phys Anthropol* 115:372–379, 2001. © 2001 Wiley-Liss, Inc.

Excavations carried out in 1986 by Klíma at the Gravettian site of Dolní Věstonice (Moravia) brought to light a multiple burial including three well-preserved skeletons (Fig. 1). The uncalibrated C14 date obtained from charcoal directly associated with the burial points to an age of $26,640 \pm 110$ B.P., falling within the time span of several dates from the same site (Svoboda, 1995). This date places the finding at an early stage of the Gravettian, a phase of the Upper Paleolithic rich in artistic and symbolic expressions which developed in Europe between 30,000–20,000 years ago (Roebroeks et al., 2000).

The three skeletons belong to young individuals lying in an extended position and covered by burnt spruce logs and branches, possibly part of a wooden funeral structure. The individual in the middle (DV 15), placed in first and partly covered by the other two, shows severe pathological changes and cannot be confidently sexed morphologically due to pelvic deformations. The other two skeletons (DV 13 and DV 14) belong to males and lie in an unusual position: one face down, the other on its side with hands reaching the pubic region of the skeleton in the middle. The heads of all three individuals are covered with red ochre, and DV 15 also exhibits powdered ochre around the pubis. Pierced carnivore ca-

nines and ivory beads form part of the ornamentation of the skulls. However, the association of a singular slate plate with parallel incisions (Emmerling et al., 1993) and of other objects with the burial is more difficult to demonstrate. Finally, analysis of dental traits may indicate genetic relationships among the members of the common grave (Alt et al., 1997).

Stratigraphic evidence and the absence of perturbation of anatomical connections and associated ornaments suggest a simultaneous burial (Klíma, 1987a; Vlček, 1991) or at least interment of the three specimens in a short lapse of time, i.e., before the decay of soft tissues of the individual first buried. In both cases, the death of three young individuals is an exceptional event, and a triple burial in itself represents a very rare funerary pattern during this

Grant sponsor: Murst; Grant number: Cofin.99; Grant sponsor: CNR; Grant number: 97.00579.PF36.

*Correspondence to: Dr. Vincenzo Formicola, Department of Ethology, Ecology, and Evolution, University of Pisa, via A. Volta 6, I-56126 Pisa, Italy. E-mail: vformi@discau.unipi.it

Received 2 December 1999; accepted 10 April 2001.



Fig. 1. Gravettian triple burial found at open-air site of Dolní Věstonice (Moravia). The skeleton in the middle is pathological. From left to right: DV 13, DV 15, and DV 14.

time period, with an analog only at Barma Grande (Grimaldi caves) (Verneau, 1906; Formicola, 1990). Moreover, the position of the hands of DV 13, and more importantly the prone position of DV 14, are very unusual.

The young age and position of the specimens and their possible simultaneous interment have given rise to speculations concerning the symbolic significance of this burial (Klíma, 1987a). The skeletal deformations of the specimen in the middle further emphasize the peculiarity of this burial pattern. Funerary behavior reflects both aspects of social life and beliefs of past populations. Inferring these aspects requires a holistic approach that brings together paleoethnological, paleopathological, and anthropological data. Thus, the paleopathological study of DV 15 combines the traditional focus on history of the diseases and on reconstruction of life conditions of past populations, with the additional goal of addressing issues of Upper Paleolithic funerary behavior.

In this paper we seek a diagnosis of the pathology manifest in DV 15.

THE SKELETON DOLNI VESTONICE 15

The skeleton DV 15 is nearly complete and in an overall good state of preservation. Major damage (mainly due to crushing) affects the vertebral bodies, sacrum, and scapulae. In addition, parts of the skull base and of the palate, nasal bones, left mandibular incisors, sternum, and most of the hand and foot bones are missing.

Epiphysal fusion and dental development indicate that these remains belong to a young adult individual. In agreement with previous analyses (Vlček, 1991, 1997; Jelínek, 1992), we suggest an age of about 20 years. The same parameters yield a slightly younger age (17–19 years) for the other two specimens, DV 14 being the youngest.

While based on morphology of well-preserved hip bones a general agreement exists that DV 13 and 14 are males, the sex of DV 15 is problematic and different determinations have been proposed by Vlček (1991), Jelínek (1992), and Novotný (1992). As already pointed out by these authors, the main difficulty lies in an admixture of male and female characteristics exhibited by the pelvis, possibly linked to the pathological state of the skeleton. Thus, a reliable sex diagnosis cannot be obtained on purely anatomical grounds.

The main pathological changes are mostly restricted to the postcranium and include:

Asymmetric shortening of the femur, with the right side 16 mm shorter than the left (bicondylar length, right = 367 mm; left = 383 mm) (Fig. 2a,b);

Anterior bowing of the upper third of right femoral shaft (Fig. 2c);

Mild coxa vara (neck-shaft angle on right side = 11°) (Fig. 2a), and retroversion of the head (-5°);

Elongation of both fibulae relative to tibial length, i.e., fibular length is equal to or slightly exceeds that of the tibia (Martin n. 1 of right and left fibula = 339 and 337 mm, respectively; Martin n. 1 of right and left tibia = 337 and 337 mm, respectively) (Fig. 2d);

Medial incurving of the distal one-third right humerus. At the point of curvature there is an anomalous increase of the diaphyseal circumference (Fig. 3a);

Slight dorsal bowing of the proximal diaphysis of the left radius and bony callus formation on the distal diaphysis of the ipsilateral ulna. Both left forearm bones are short relative to their counterparts (8–9 mm, respectively) (Fig. 3b,c); and

Diffuse enamel hypoplasias, particularly marked on upper and lower first molars (Fig. 4). Their position on the tooth crowns suggests that a severe disruption took place around 18 months of age.

Additionally, the vertebral column exhibits scoliosis, misshapen spinous processes of the fourth and fifth lumbar vertebrae, and hypoplastic development of lateral portions of the sacrum. Superior pubic rami are short.

Radiographic examination of the bones reveals no signs of fracture or abnormalities of the bony structure, except thickening of the humeral compacta at the point of shaft curvature and of the distal diaphysis of the left ulna (Fig. 5). No Harris lines are detectable, possibly due to a long remodeling period.

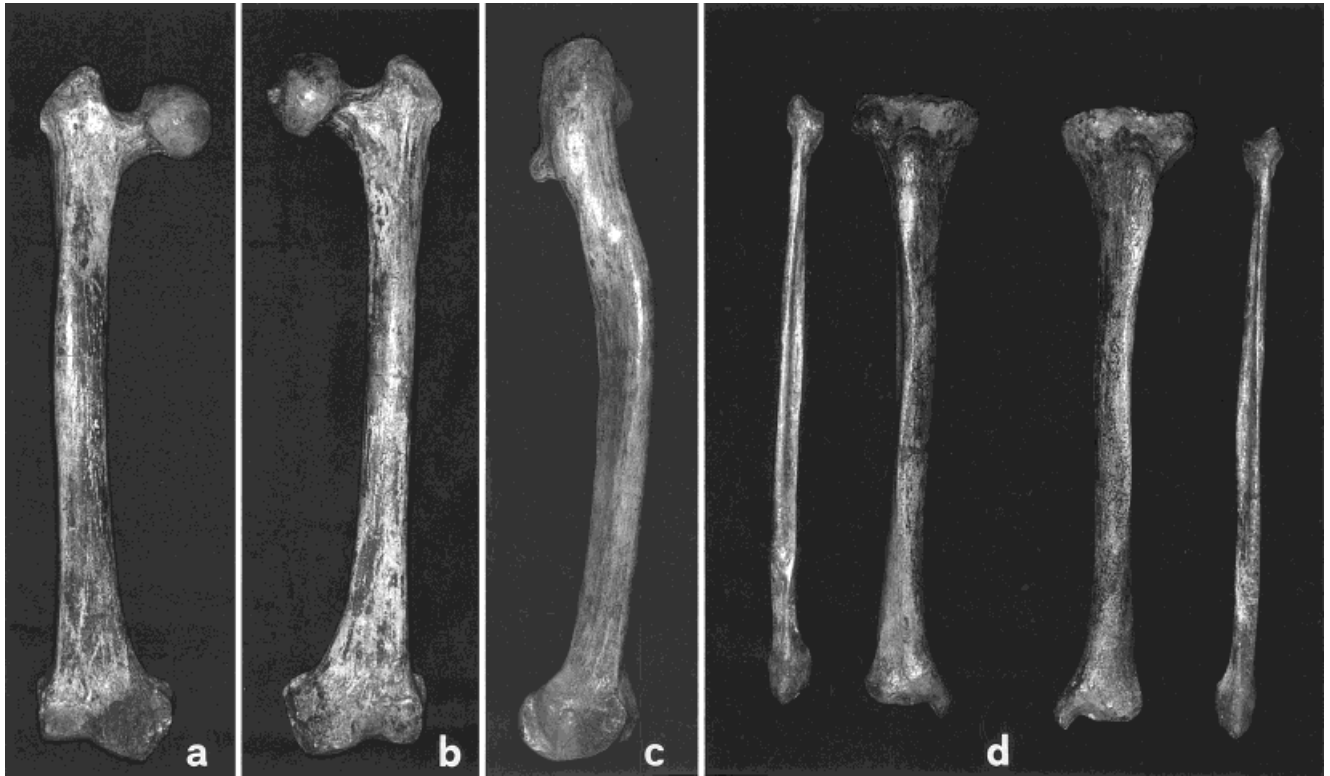


Fig. 2. Lower limb bones of DV 15: right (a) and left (b) femur in anterior view, right femur in lateral view (c), and right and left fibulae and tibiae (d). Note shortening and bowing of right femur, varus deformation of its neck, and relative elongation of fibulae.

DIFFERENTIAL DIAGNOSIS

The early hypotheses

The skeleton of DV 15 has not yet been thoroughly investigated from a paleopathological point of view. However, two hypotheses have been proposed to explain the observed bone deformations:

- 1) hemiparalysis of the right side, possibly resulting from encephalitis suffered in early life (Klíma, 1987b); or
- 2) a rachitic condition (Jelínek, 1992).

Both diagnoses are unlikely. In particular, the well-developed muscular insertions, the normal degree of lateralization, and the thickness of cortical tissue argue against the first possibility, while the absence of bowing of tibiae and fibulae, i.e., the bones generally showing the most obvious manifestations of rickets, is inconsistent with the latter diagnosis.

Another hypothesis has been suggested by Kuklík (1992), who attributes the deformities of the right femur to a congenital disease of poorly known etiology: proximal femoral focal deficiency (PFFD). PFFD is a disease characterized by unilateral shortening of the femur due to failure of normal development of a portion of the proximal femur (Resnick, 1995a). Four classes of defects, based on femoral and acetabular abnormalities, have been proposed (Aitken, 1969). In the milder forms (class A), the femur

is short, incurved, and affected by subtrochanteric varus deformation. The femoral head is regularly connected to the shaft, and the acetabulum is almost normal. In the remaining forms (classes B, C, and D), connections between the femoral head and shaft are lacking and, in increasing order of severity, the shaft, head, and acetabulum are dysplastic or absent (see Fig. 90-23 in Resnick, 1995a).

Regarding Aitken's class A, it is important to note that the shaft is very short (Fixsen and Lloyd-Roberts, 1974; Epps, 1983; Gillespie and Torode, 1983). Thus, the moderate bilateral asymmetry exhibited by DV 15 (about 4%, calculated as $rt - lt / (rt + lt) / 2$) speaks against such an attribution. However, Amstutz (1969) and Hamanishi (1980) further subdivided class A into subtypes including cases previously indicated as femoral hypoplasia with coxa vara (Ring, 1960). In these cases, the shortening of the affected femur is less than 10%. According to Hamanishi (1980) and Resnick (1995a), the lack of discontinuity between two syndromes (femoral hypoplasia and PFFD) suggests a single clinical entity with different degrees of expression that is referred to as "congenital short femur" (CSF). Independently from the severity of shortening, however, the femur is laterally bowed and varus deformity develops in the subtrochanteric shaft (Levinson et al., 1977; Pavlov et al., 1980; Boden et al., 1989; Resnick, 1995a). Additionally, associated anomalies with PFFD/CSF include hypoplastic development of the

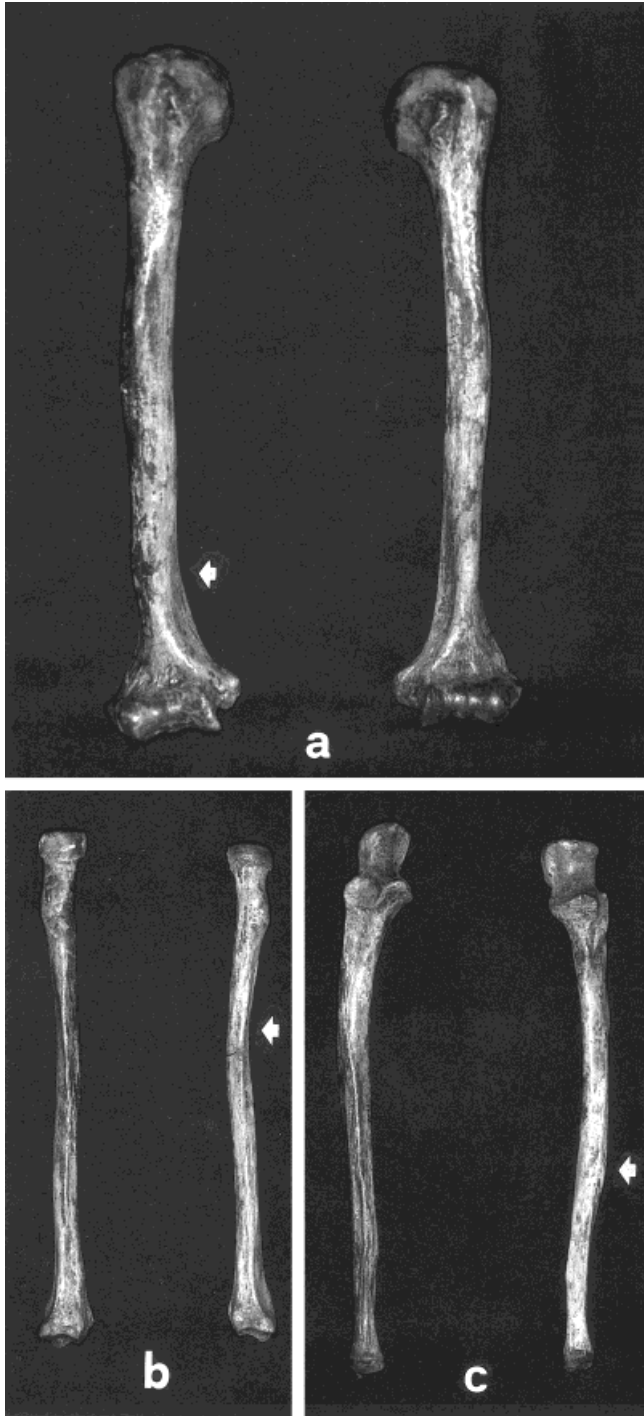


Fig. 3. Upper limb bones of DV 15: anterior views of right and left humeri (a), and medial views of right and left radii (b) and ulnae (c). Note bowing of right humerus and left radius, callus formation on left distal ulna (arrows), and shortening of left lower arm bones.

lesser trochanter, and absence or hypoplasia of the ipsilateral fibula and hemimelia of upper limb bones, particularly of the ulna (Panting and Williams, 1978; Schatz and Kopits, 1978; Hillmann et al., 1987; Boden et al., 1989).

None of the diagnostic features listed above is exhibited by DV 15. Specifically, sagittal bowing,

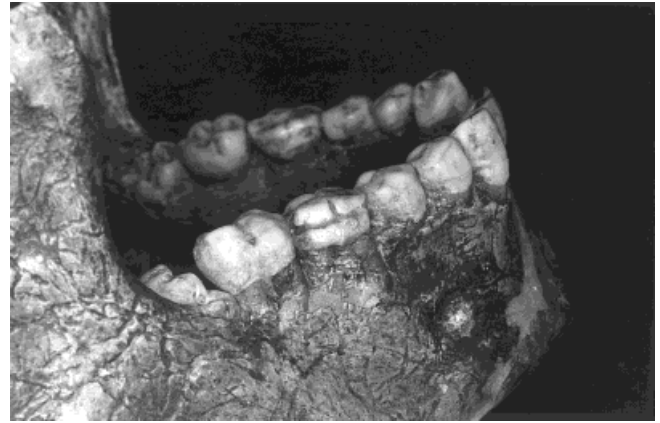


Fig. 4. Marked enamel hypoplasias on DV 15's first lower molar.



Fig. 5. Radiographs of femoral and humeral diaphyses of DV 15 at point of curvature and of left ulna and radius.

varus deformity at the level of the femoral neck, and more generally the absence of other stigmata associated with the disease do not support a diagnosis of PFFD/CSF.

Towards a diagnosis

Using asymmetric shortening of the long bones as a "key trait" in the search for the etiology of the disease, we have identified a few additional conditions including caudal regression syndrome, femoral hypoplasia with unusual facies syndrome, and chondrodysplasia calcificans punctata. The first two syndromes share characteristics and both show a strong relationship with maternal diabetes. In particular, it has been found that high levels of insulinemia in the fetus of a diabetic mother can result in a wide variety of congenital anomalies, including abnormal growth, and soft-tissue and skeletal abnormalities (Resnick, 1995b).

Two patterns of malformations have been identified and referred to as caudal regression syndrome (CRS) and femoral hypoplasia with unusual facies syndrome (FH-UFS). Both result in postural deformities of the lower extremities, sometimes involving asymmetric development of the femur (Johnson et al., 1983; Guidera et al., 1991). However, the severity of femoral hypoplasia and its frequent association with radio-ulnar or radio-humeral synostosis (Daentl et al., 1975) make a diagnosis of FH-UFS very unlikely in DV 15. CRS is equally unlikely, considering that this syndrome also involves sacral agenesis of variable but generally severe intensity, hip dislocations, and foot deformities (Resnick, 1995a).

The third syndrome, chondrodysplasia calcificans punctata (CCP), is an inherited form of multiple epiphyseal dysplasia characterized by stippled calcifications in some areas of enchondral bone formation. Tubular bones, particularly the femur and humerus, and the vertebrae are among the most frequently affected elements. The disease ranges from a severe rhizomelic form resulting in stillbirth or death within the first months of life to a milder disorder, sometimes showing asymmetrical limb shortening (Goldman, 1995). In cases of survival, calcifications disappear by age 1–3 years (Hyndman et al., 1976; Goldman, 1995), but there may be residual deformity. Correlation between severity of stippling and residual deformity has been pointed out (Comings et al., 1968; Spranger et al., 1971; Silengo et al., 1980).

Different types of CCP have been recognized on the basis of phenotype, mode of inheritance, and gene defect localization (Goldman, 1995):

- Rhizomelic, autosomal-recessive: lethal.
- X-linked dominant (Conradi-Hunermann disease): lethal for males, but associated with a good prognosis for females.
- X-linked recessive (Curry type): normal survival.
- X-linked recessive (Sheffield type): normal survival.
- Tibia-metacarpal type: normal survival.

All the different types of CCP exhibit symmetric shortening of the limbs, with the exception of the X-linked dominant form, characterized by asymmetric involvement. Focal disruption of the growth plate, varying from bone to bone, is the likely cause of the asymmetrical nature of the changes (Rimoin et al., 1976). The diagnosis is usually made during the first year of life, based on the peculiar appearance of the newborn and radiographic examination. However, despite the early disappearance of stippling, recognition of skeletal and soft-tissue anomalies allows diagnosis later in life (Comings et al., 1968; Hyndman et al., 1976). The disease has a highly variable clinical expression, as stressed by many authors (Silengo et al., 1980; Manzke et al., 1980; Mueller et al., 1985), who cite the existence

within a single family of individuals mildly and severely affected.

Skeletal changes found associated with femur asymmetry include varus deformity of the femoral neck (Hyndman et al., 1976; Silengo et al., 1980), asymmetric development of both forearm bones (Manzke et al., 1980; Mueller et al., 1985), scoliosis and vertebral anomalies (Goldman, 1995; Happle, 1979), and elongation of the fibulae (Josephson and Oriatti, 1961; Jerre, 1962; Wynne Davies et al., 1985). Thus, a diagnosis of X-linked dominant CCP is supported by the asymmetric shortening of the femur, radius, and ulna, coxa vara, scoliosis, and relative elongation of fibulae.

"Saddle nose" (flat nasal bridge) and contractures at the level of the hip and knee represent additional skeletal anomalies observed clinically (Happle, 1979; Manzke et al., 1980; Goldman, 1995). In DV 15, the presence of a flat nasal bridge cannot be confirmed, given the absence of nasal bones, but this feature tends to become less evident with age (Sheffield et al., 1976). As far as contractures are concerned, there are no traces left on the bones.

Stature, depending on the severity of the disease, can vary from normal to very short (Spranger et al., 1971; Silengo et al., 1980). In DV 15, the stature of about 150 cm obtained from the femur and tibia is short both when compared with the few Gravettian female remains from Moravia and with the whole European Early Upper Paleolithic female sample (Formicola and Giannecchini, 1999).

Finally, the very marked enamel hypoplasias shown by DV 15's first molars point to dramatic perturbations in health status during early life (around age 18 months, based on the position of the defects on tooth crowns), a very critical period for children affected by CCP (Lischi and Menichini, 1967; Sheffield et al., 1976; Silengo et al., 1980).

The main obstacle in accepting CCP as the only possible etiology lies with the presence of bowing deformities. These changes are part of the lethal rhizomelic form, but are not typically associated with the X-linked dominant form of CCP. Considering that bowing of long bones is only occasionally found in subjects affected by that form of the disease (Kampf, 1939; Brogdon and Crow, 1958; Weber, 1958; Andersen and Justensen, 1987; Mason and Kozlowsky, 1973; Wynne-Davies et al., 1985), this condition can hardly be responsible for the simultaneous incurving of humerus, radius, and femur. The involvement of other factors is required to justify the observed pathological pattern. Traumatic injuries provide a likely explanation.

The old orthopedic literature reports that a few cases of congenital abnormalities of long bones were initially regarded as healed birth or intrauterine fractures (Ring, 1959). Such injuries are related to different factors, including breech presentation and labor difficulties (Behrman and Mangurten, 1977; Resnick et al., 1995), and result initially in severe shortening and bowing deformities. In these cases,

as well as in cases of fractures suffered by very young children, follow-up examinations show marked corrections and improvements of both changes in the absence of signs of repair, due to a long period of remodeling (Ridén, 1935; Madsen, 1955; Bakalim and Wilppula, 1972; Vahvanen and Aalto, 1978; Hagglund et al., 1988; Tachdijan, 1990). It has also been shown that the tendency toward spontaneous correction of angular deformities and of length discrepancies is great, and that even severe displacements left to heal in an anatomically bad position have a good prognosis.

As far as the humerus is concerned, a supracondylar fracture is a rather common injury in children and is produced by a fall on the hand with the elbow in hyperextension. Interestingly, the residual deformation often results in varus deviation, as in DV 15. Varus deviation is a consequence both of the direction of the fracturing force (French, 1959; Graham, 1967) and of the pull of the strong pronator teres muscle in absence of the opposed action of the biceps brachii muscle due to the break in the humerus (Tachdijan, 1990).

Acute plastic bowing deformities (APBD) are an additional entity recently recognized by orthopedists (Borden, 1974; Cail et al., 1978; Price, 1996), and consist of broad bowing of the shaft with a shape representing an exaggeration of the usual curvature. The mechanism responsible for these bowing deformities lies in strong compressive longitudinal forces applied to both ends of naturally curved long bones. Stress ranging between elastic and fracture limits causes bending that remains after stress is removed. Lower arm bones are the bones most frequently involved, typically a result from a fall onto an outstretched hand (Naga and Broadrick, 1977; Stuart-MacAdam et al., 1998). In many instances, the fracture of one of the lower arm bones is associated with APBD of the other bone (Borden, 1975; Crowe and Swischuck, 1977; Resnick et al., 1995; Price, 1996).

Bowing of the left radius and callus formation shown by the ipsilateral ulna of DV 15 are consistent with a scenario involving, respectively, APBD and a healed early fracture. As already pointed out, varus deviation affecting the right humerus might also result from a very early injury. This hypothesis is also supported by the anomalous shape and thickening of the compacta of the humerus at the point of curvature. More problematic is referring the incurving of the femur to the same etiology in the absence of the changes shown by humerus and ulna. Moreover, referring whole bowing deformities to a sequel of trauma would not explain the additional anomalies exhibited by DV 15 (asymmetric development of femur and of lower arm bones, elongation of fibulae). Finally, as already pointed out, bowing of long bones has been occasionally found in the X-linked dominant form of CCP; regarding the femur specifically, the radiograph of a bowed femur in an affected girl

is reported in a textbook on skeletal dysplasias (Fig. 4.23 in Wynne-Davies et al., 1985).

In conclusion, the combination of CCP and trauma provides the most likely explanation for the pathological pattern exhibited by DV 15. In particular, while the X-linked dominant form of CCP is probably responsible for most of the changes, bowing deformations of the upper limb bones likely result from traumatic injuries suffered during early life.

FINAL CONSIDERATIONS

While examples of traumatic injuries to limb bones are not uncommon in the Upper Paleolithic record, DV 15 provides very early evidence of CCP in the paleontological material and increases our knowledge on the history of inherited disorders. The diagnosis of CCP, however, has important implications going beyond mere paleopathological value.

Specifically, the diagnosis implies that the DV 15 skeleton belongs to a female, considering that the asymmetric shortening is typical of the X-linked dominant form, which is lethal during early infancy in males. Moreover, the diagnosis provides clues about therapeutic knowledge of Upper Paleolithic populations, since the survival of similarly affected children is very problematic in the absence of adequate treatment and care. Recurrent infections of the respiratory and gastrointestinal tracts, respiratory and feeding difficulties, and failure to thrive are among the most frequent causes of health disturbances (Mosekilde, 1952; Mason and Kozlowsky, 1973; Sheffield et al., 1976; Goldman, 1995). The extremely severe enamel hypoplasias dated to a very early stage of DV 15's life provide a likely record of at least one of those disruptions.

The diagnosis also suggests the possibility that DV 15 experienced some kind of soft-tissue anomalies, since cataracts, epicanthus, erythemas, ichthyosis, and alopecia are frequently associated clinical findings (Happle, 1979; Manzke et al., 1980; Kozlowsky et al., 1988; Goldman, 1995). Thus, skeletal deformations, as well as the other stigmata of the disease, emphasize the singular aspect and the "diversity" of this individual, pointing to a condition that should be taken into account when speculating on the significance of this burial.

The Upper Paleolithic fossil record includes other cases of diseases resulting in physical deformations significantly associated with peculiar funerary contexts. Undoubtedly the most emblematic case is the Romito (Calabria, Italy) chondrodystrophic dwarf buried together with an old woman in a cave important for its expressions of mobiliary and parietal art (Fruyer et al., 1988). Also in Italy, in the Arene Candide necropolis, rich in ornamental and symbolic objects (Cardini, 1980), a few skeletons show changes that are probably due to an inherited form of rickets (Formicola, 1995). A further example comes from Gravettian Moravia, where spectacular grave goods, including an articulated ivory male figure (Jelínek et al., 1959), were found associated

with the poorly preserved skeleton Brno 2. Only a few fragmentary parts of the postcranial bones are preserved, but what remains is affected by very severe periostitis (Oliva, 2000). Finally, one of the two children from the Gravettian site of Sungir, found buried head to head with extremely rich grave goods (Bader, 1970), exhibits bowing of the femora (Bukhman, 1984; Buzhilova, 2000). The etiology of this deformity is not clear, but the burial emphasizes issues of social perception of diversity and of the role of these individuals in their society. Rich ornamentation, elaborate funerary behavior, and site of inhumation shed light on ideological aspects, strengthening the idea that a few Upper Paleolithic burials included selected individuals and that physical diversity may have played a role in selective burial patterns from that period.

ACKNOWLEDGMENTS

Radiographs were kindly provided by E. Trinkaus. We also acknowledge V. Alekshin for information and reprints on Sungir material. Thanks are also due to an anonymous referee for careful comments and suggestions.

LITERATURE CITED

- Aitken GT. 1969. Proximal femoral focal deficiency: definition, classification, and management. In: Aitken GT, editor. Proximal femoral focal deficiency: a congenital anomaly. Symposium held in Washington, June 1968. Washington, DC: National Academy of Sciences. p 1–22.
- Alt KW, Pichler S, Vach V, Klíma B, Vlček E, Sedlmeier J. 1997. Twenty-five thousand-year-old triple burial from Dolní Věstonice: an Ice Age family? *Am J Phys Anthropol* 102:123–131.
- Amstutz HC. 1969. The morphology, natural history and treatment of proximal femoral deficiency. In: Aitken GT, editor. Proximal femoral focal deficiency: a congenital anomaly. Symposium held in Washington, June 1968. Washington, DC: National Academy of Sciences. p 50–76.
- Andersen PE, Justensen P. 1987. Chondrodysplasia punctata. Report of two cases. *Skeletal Radiol* 16:223–226.
- Bader ON. 1970. Das zweite Grab in der paläolithischen Siedlung Sungir im mittleren Russland. *Quartar* 21:103–104.
- Bakalim G, Wilppula E. 1972. Supracondylar humeral fractures in children. *Acta Orthop Scand* 43:366–374.
- Behrmann RE, Mangurten HH. 1977. Birth injuries. In: Behrmann RE, editor. Neonatal perinatal medicine: diseases of the foetus and infant. St. Louis: C.V. Mosby. p 146–170.
- Boden DS, Fallon MD, Davidson R, Mennuti MT, Kaplan FS. 1989. Proximal femoral focal deficiency. *J Bone Joint Surg [Am]* 71:1119–1129.
- Borden S. 1974. Traumatic bowing of the forearm in children. *J Bone Joint Surg [Am]* 56:611–616.
- Borden S. 1975. Roentgen recognition of acute plastic bowing of the forearm in children. *AJR Radium Ther Nucl Med* 125:524–530.
- Brogdon BG, Crow NE. 1958. Chondrodystrophia calcificans congenita. *AJR* 80:443–448.
- Bukhman AI. 1984. Roentgenological studies of the children's skeletons from the Upper Paleolithic site Sungir [in Russian]. In: Zubov AA, Kharitonov VM, editors. Sungir anthropological investigations. Moscow: Nauka. p 203–204.
- Buzhilova AP. 2000. The analysis of anomalies and indicators of physiological stress in non-mature Sungir individuals. In: Alexeeva TI, Bader NO, editors. *Homo sungirensis*. Upper Palaeolithic man: ecological and evolutionary aspects of the investigation. Moscow: Nauchny: Mir. p 302–314.
- Cail WS, Keats TE, Sussman MD. 1978. Plastic bowing fracture of the femur in a child. *AJR* 130:780–782.
- Cardini L. 1980. La necropoli mesolitica delle Arene Candide (Liguria). *Mem Ist It Paleontol Um* 3:9–32.
- Comings DE, Papazian C, Schoene HR. 1968. Conradi's disease. *J Pediatr* 72:63–69.
- Crowe JE, Swischuck LE. 1977. Acute bowing fractures of the forearm in children. *AJR* 128:981–984.
- Daentl DL, Smith DW, Scott CI, Bryan DH, Gooding CA. 1975. Femoral hypoplasia—unusual facies syndrome. *J Pediatr* 86:107–111.
- Emmerling EH, Geer B, Klíma B. 1993. Ein Mondkalenderstab aus Dolní Věstonice. *Quartar* 43/44:151–162.
- Epps CH. 1983. Current concepts review: proximal femoral focal deficiency. *J Bone Joint Surg [Am]* 65:867–870.
- Fixsen JA, Lloyd-Roberts GC. 1974. The natural history and early treatment of proximal femoral dysplasia. *J Bone Joint Surg [Br]* 56:86–95.
- Formicola V. 1990. The triplex burial of Barma Grande (Grimaldi, Italy). *Homo* 39:130–143.
- Formicola V. 1995. X-linked hypophosphatemic rickets: a probable Upper Paleolithic case. *Am J Phys Anthropol* 98:403–409.
- Formicola V, Giannecchini M. 1999. Evolutionary trends of stature in Upper Paleolithic and Mesolithic Europe. *J Hum Evol* 36:319–333.
- Frayer DW, Macchiarelli R, Mussi M. 1988. A case of chondrodystrophic dwarfism in the Italian Late Upper Paleolithic. *Am J Phys Anthropol* 75:549–565.
- French PR. 1959. Varus deformity of the elbow following supracondylar fractures of the humerus in children. *Lancet* 7100:439–441.
- Gillespie R, Torode IP. 1983. Classification and management of congenital abnormalities of the femur. *J Bone Joint Surg [Br]* 65:557–568.
- Goldman AM. 1995. Heritable diseases of connective tissue, epiphyseal dysplasias, and related conditions. In: Resnick D, editor. *Diagnosis of bone and joint disorders*. Philadelphia: W.B. Saunders. p 4095–4162.
- Graham HA. 1967. Supracondylar fractures of the elbow in children. *Clin Orthop* 54:85–101.
- Guidera KJ, Raney E, Ogden JA, Highhouse M, Habal M. 1991. Caudal regression: a review of seven cases, including the mermaid syndrome. *J Pediatr Orthop* 11:743–747.
- Hagglund G, Hansson LI, Wiberg G. 1988. Correction of deformity after femoral birth fracture: 16-year follow-up. *Acta Orthop Scand* 59:333–335.
- Hamanishi C. 1980. Congenital short femur. *J Bone Joint Surg [Br]* 62:307–320.
- Happle R. 1979. X-linked dominant chondrodysplasia punctata. *Hum Genet* 53:65–73.
- Hillmann JS, Mesgarzadeh M, Revesz G, Bonakdarpour A, Clancy M, Betz RR. 1987. Proximal femoral focal deficiency: radiologic analysis of 49 cases. *Radiology* 165:769–773.
- Hyndman WB, Alexander DS, Mackie KW. 1976. Chondrodystrophia calcificans congenita (the Conradi-Hunermann syndrome). *Clin Pediatr* 15:317–321.
- Jelinek J. 1992. New Upper Paleolithic burials from Dolní Věstonice. *ERAUL* 56:207–227.
- Jelinek J, Pelíšek J, Valoch K. 1959. Der fossile Mensch Brno II. *Anthropos (Brno)* 9:5–30.
- Jerre T. 1962. Dysplasia epiphysialis punctata. *Acta Orthop Scand* 32:315–323.
- Johnson JP, Gooch WM, Petersen J, Beattie JF. 1983. Femoral hypoplasia-unusual facies syndrome in infants of diabetic mothers. *J Pediatr* 102:866–872.
- Josephson BM, Oriatti MD. 1961. Chondrodystrophia calcificans congenita. Report of a case and review of the literature. *Pediatrics* 28:425–435.
- Kampf E. 1939. Chondrodystrophia calcificans congenita. *Z Kinderheilkd* 61:124–126.
- Klíma B. 1987a. Une triple sépulture du Pavlovien à Dolní Věstonice, Tchécoslovaquie. *Anthropologie* 91:329–334.
- Klíma B. 1987b. Das jungpaläolithische Massengrab von Dolní Věstonice. *Quartar* 37/38:53–62.

- Kozlowsky K, Bates EH, Young LW, Wood BP. 1988. Radiological case of the month: dominant X-linked chondrodysplasia punctata. *Am J Dis Child* 142:1233.
- Kuklík M. 1992. Die Reflexion über den Befunden aus dem jungpaläolithischen Dreigrab in Dolní Věstonice nach der genetischen Ansicht. *Acta Mus Natl Pragae* 48:148–151.
- Levinson ED, Ozonoff MB, Royen PM. 1977. Proximal femoral focal deficiency (PFFD). *Radiology* 125:197–203.
- Lischi G, Menichini G. 1967. L'évolution clinique et radiologique de la chondropathie calcifiante congénitale. *Helv Paediatr Acta* 22:289–301.
- Madsen ET. 1955 Fractures of the extremities in the newborn. *Acta Orthop Scand* 34:41–74.
- Manzke H, Christophers E, Wiedemann HR. 1980. Dominant sex-linked inherited chondrodysplasia punctata: a distinct type of chondrodysplasia punctata. *Clin Genet* 17:97–107.
- Mason RC, Kozlowsky K. 1973. Chondrodysplasia punctata. A report of 10 cases. *Radiology* 109:145–150.
- Mosekilde E. 1952. Stippled epiphyses in the newborn and in infants. *Acta Radiol* 37:291–297.
- Mueller RF, Crowle PM, Jones RAK, Davison BCC. 1985. X-linked dominant chondrodysplasia punctata. A case report and family studies. *Am J Med Genet* 20:137–144.
- Naga AH, Broadrick GL. 1977. Traumatic bowing of the radius and ulna in children. *NC Med J* 38:452–456.
- Novotny V. 1992. Pelves and sexual dimorphism in hunters of Dolní Věstonice [in Czech]. *Acta Mus Natl Pragae* 48:152–163.
- Oliva M. 2000. The Brno II Upper Paleolithic burial. In: Roebroeks W, Mussi M, Svoboda J, Fennema K, editors. *Hunters of the golden age. The Mid Upper Paleolithic of Eurasia (30,000–20,000 BP)*. Leiden: University Press. p 143–153.
- Panting AL, Williams PF. 1978. Proximal femoral focal deficiency. *J Bone Joint Surg [Br]* 60:46–52.
- Pavlov H, Goldman AB, Freiburger RH. 1980. Infantile coxa vara. *Radiology* 135:631–640.
- Price CT. 1996. Injuries to the shafts of the radius and ulna. In: Rockwood CA, Wilkins KE, Beaty JH, editors. *Fractures in children*. Philadelphia: Lippincot-Raven. p 515–547.
- Resnick D. 1995a. Additional congenital or heritable anomalies and symptoms. In: Resnick D, editor. *Diagnosis of bone and joint disorders*. Philadelphia: W.B. Saunders. p 4269–4330.
- Resnick D. 1995b. Disorders of other endocrine glands and of pregnancy. In: Resnick D, editor. *Diagnosis of bone and joint disorders*. Philadelphia: W.B. Saunders. p 2076–2104.
- Resnick D, Goergen TG, Niwayama G. 1995. Physical injury: concepts and terminology. In: Resnick D, editor. *Diagnosis of bone and joint disorders*. Philadelphia: W.B. Saunders. p 2561–2692.
- Ridén A. 1935. Birth fractures of the femur. *Surg Gynecol Obstet* 60:1098–1105.
- Rimoin DL, Silberberg R, Hollister DW. 1976. Chondro-osseous pathology in the chondrodystrophies. *Clin Orthop* 114:137–152.
- Ring PA. 1959. Congenital short femur. *J Bone Joint Surg [Br]* 41:73–79.
- Ring PA. 1960. Congenital abnormalities of the femur. *Arch Dis Child* 36:410–417.
- Roebroeks W, Mussi M, Svoboda J, Fennema K, editors. 2000. *Hunters of the golden age. The Mid Upper Paleolithic of Eurasia (30,000–20,000 BP)*. Leiden: University Press.
- Schatz SL, Kopits SE. 1978. Proximal femoral focal deficiency. *AJR* 131:289–295.
- Sheffield LJ, Danks DM, Mayne V, Hutchinson LA. 1976. Chondrodysplasia punctata—23 cases of a mild and relatively common variety. *J Pediatr* 89:916–923.
- Silengo MC, Luzzatti L, Silverman FN. 1980. Clinical and genetic aspects of Conradi-Hunermann disease. *J Pediatr* 97:911–917.
- Spranger JW, Opitz JM, Bidder U. 1971. Heterogeneity of chondrodysplasia punctata. *Humangenetik* 11:190–212.
- Stuart-MacAdam P, Glencross B, Kricum M. 1998. Traumatic bowing deformities in tubular bones. *Int J Osteoarchaeol* 8:252–262.
- Svoboda J. 1995. L'art gravettien en Moravie: contexte, dates et styles. *Anthropologie* 99:258–272.
- Tachdjian MO. 1990. *Pediatric orthopedics*. Philadelphia: W.B. Saunders.
- Vahvanen V, Aalto K. 1978. Supracondylar fracture of the humerus in children. *Acta Orthop Scand* 49:225–233.
- Verneau R. 1906. *Les grottes de Grimaldi*. Anthropologie. Monaco: Imprimerie de Monaco.
- Vlček E. 1991. Die Mammuthjäger von Dolní Věstonice. *Archaeol Mus* 22:1–136.
- Vlček E. 1997. Human remains from Pavlov and the biological anthropology of the Gravettian human population of South Moravia. In: Svoboda J, Skrdla P, editors. *Pavlov I Northwest. The Upper Paleolithic burial and its settlement context*. Dolní Věstonice Stud 4:53–153.
- Weber A. 1958. Zur Frage der Chondrodystrophia calcificans congenita. *Helv Paediatr Acta* 13:228–238.
- Wynne-Davies R, Hall CM, Apley AG. 1985. *Atlas of skeletal dysplasias*. Edinburgh: Churchill Livingstone.