

PROBLEMS OF DIFFERENTIAL DIAGNOSIS IN  
PLEISTOCENE MAMMAL PATHOLOGY. SHORT COMMUNICATION

Don Brothwell

*Department of Archaeology, University of York, England, YO1 7EP*

VĖLYVUOJU PLEISTOCENU DATUOJAMŲ ŽINDUOLIŲ PATOLOGIJŲ  
DIFERENCINĖS DIAGNOZĖS PROBLEMOS. TRUMPAS PRANEŠIMAS

Don Brothwell

*Archeologijos katedra, Jorko Universitetas, Jungtinė Karalystė YO1 7EP*

**Santrauka.** Straipsnyje analizuojami atskiros vėlyvuoju pleistocenu datuojamos mamutų kaulų patologijos iš Lynford vietovės Rytų Anglijoje. Atlikta dešimties rūšių 32 gyvūnų kaulų kolekcijos analizė. Dažnai gauti mamutų griaučių tyrimo rezultatai interpretuojami neteisingai, ypač analizuojant kaklo ir krūtinės slankstelių keterinių ataugų patologijas.

**Raktažodžiai:** mamutai, *Mammuthus primigenius*, kaulai, patologijos.

Over the years, the one thing which has become clear to me in palaeo-pathological diagnosis, is that often it is far less easy than we think, and that we tend to consider too few alternatives. Unlike medical and veterinary work on the living, we usually have calcified tissue and little else. In the future, we can perhaps expect increasing support from immunological and DNA evidence related to specific disease, but for the present we are left with little more than bones and teeth. My contribution to this meet-

ing is to expand a little on this problem of diagnosis, by reference to two Upper Pleistocene cases from the mammoth site at Lynford in East Anglia. Only 32 animals are represented, for 10 species, but parts of eleven mammoths are present and a surprising amount of pathology was noted in this eleven, raising the question as to whether some pathology is linked to hunting trauma (Schreve, 2006). But that is another story.

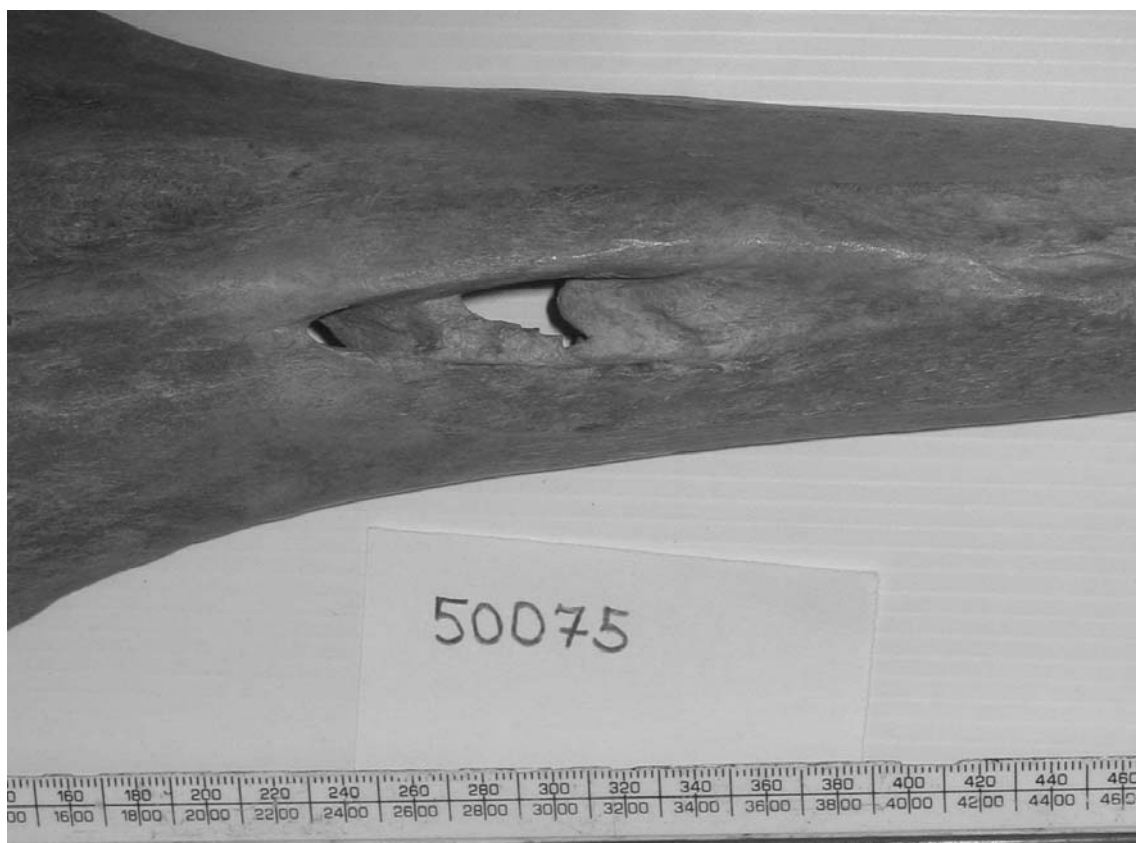


Figure 1. Anterior view of the perforation in the spinous process of Lynford 50075

The first case I want to discuss (Lynford 50075) is a nearly complete spinous process of a mammoth (Figure 1). This is one of three specimens at the site displaying the anomaly, not necessarily in the same animal. It shows as a deep posterior concavity, low on the spine, which perforates through to the anterior margin to a more limited degree. The bone is generally smooth and without evidence of periostitis or lytic activity on the surface. The condition has also been noted in seven specimens of mammoth at Krakow in Poland (Wojtal, 2001) and two cases at Sevsk in Russia, where the animals were between 1-2 years and 10-15 years old (Maschenko et al., 2006). The east European cases were more pronounced anomalies, with larger antero-posterior holes, but again with symmetrical smooth surfaces. After a considerable literature search, I have found no reference to this kind of anomaly in any other mammal. Gruneberg (1952, 1963) describes various congenital and gene controlled vertebral abnormalities in mice, including spina bifida occulta, but perforations of the spinous process are not featured. Johnson (1986) also notes abnormal neural arches, but no perforations of the spinous process. Spinous process clefting is known in mammals, but not perforations. In discussing human ver-

tebral anomalies, Barnes (1994) indicates that neural arch clefts, fused arches and curves spinous processes occur. She also illustrates a total bifid spinous process (Fig 3.40, p121), which indicates that the separate foetal situation can remain into adult life (though rare). In the mammoth, do we see this latter anomaly, but with the partial union at the upper and lower ends of the spinous process? The sex of the mammoth cases is not known, but there is clearly age variation. So how do we classify this condition or conditions of smooth holes in the spinous process? Trauma and old healed infection seem highly unlikely, as also does a neoplastic process. The similarity in position and form strongly argue for a congenital or environmentally related condition, but what? An inborn massive enlargement of a nutrient foramen seems highly unlikely, but could it have a genetic and adaptive value nevertheless? The hear of a mammoth must have produced considerable stress on the cervical and thoracic spinous processes and associated ligaments, so any adaptive strengthening of the supraspinous and interspinous ligaments would have been advantageous. Could any perforations then, be an attachment modification, even a kind of enthesopathy?



Figure 2. The Lynford spinous process (51392) displaying considerable expansion, probably caused by a neoplasm

The second case of pathology is also, to me at least, problematic. Again it involves the spinous process of a Lynford Mammoth (51392; Figure 2). Marked but rounded expansion of the lower part of the spinous process is seen to be associated with numerous perforations

from the inside onto the outer surface. There are also restricted areas of external 'erosion', which I initially considered to be post-mortem, but on more careful examination feel now that they are part of the ante-mortem pathology. There are no surface areas of sub-periosteal new

bone or periostitis. There are no areas of pitting or striations suggestive of infection. But associated with some holes are limited areas of lytic destruction. The CT scans reveal much remodelling inside the bone, with connections to the exterior surface. So what are we dealing with here? A differential diagnosis must clearly include congenital abnormality, microbial and mycotic infection, and neoplasms. The absence of clear inflammatory changes is against a diagnosis of tuberculosis or actinomycosis for instance. Wound infection would also produce characteristic surface changes, which are not present. The relatively smooth bone swelling and perforations is also not typical of a mycotic condition. So a neoplasm of some form appears to be more likely, but how far can we reduce the diagnostic alternatives when we are dealing with tumours? This is surely one of the most difficult aspects of zoopalaeopathology, and I therefore raise this as an interesting diagnostic problem for the meeting. The possible neoplastic pathology is therefore a swollen spinous process with internal remodelling and some perforations to a smooth exterior surface, except for restricted 'erosions' of a possible lytic nature. It is not an irregular or spicular mass, which argues against some tumours such as osteosarcoma. On the other hand, conditions which can cause bone expansion and cavitation would surely include chondrosarcoma, benign bone cysts, fibro-osseous tumour and multiple cartilaginous exostoses. One major problem is that tumour prevalence varies considerably between species, as also does the preferential position within the skeleton. But we have only limited information on the bone pathology of the elephant family. Nevertheless, it is interesting to speculate that the spinous process of dogs at least is a favoured site for the development of benign multiple cartilaginous exostoses, which can result in an expanded bone mass with some similarities to this mammoth case. Cystic or benign chondromatous bone lesions and even a chondrosarcoma should perhaps come in second and third place as other diagnostic possibilities. There is clearly no easy answer, but I would argue that there is some value in attempting a tentative diagnosis even with tumours.

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